MINISTRY OF HEALTH PROTECTION OF UKRAINE
ODESSA NATIONAL MEDICAL UNIVERSITY

Faculty Medical 2
(faculty name)

Department of Normal and Pathological Clinical Anatomy
(name of department)

CONFIRMED by
Acting vice-rector for scientific and pedagogical work
Svitlana KOTYUZHYNSKA

"____" _____ 20___

METHODOLOGICAL DEVELOPMENT TO PRACTICAL CLASSES ON PATHOMORPHOLOGY

Faculty, course Medical, III (STN)
Educational discipline Pathomorphology
(name of academic discipline)

Approved:
Meeting of the department of normal and pathological clinical anatomy
Odessa National Medical University

Minute No. 1 of "30" серпня 2022

The Head of the department _____ Nataliia NESKOROMNA
(signature) *(First Name Last Name)*

Developers:

Sytnikova Varvara Oleksandrivna, MD in medicine, professor

Lytvynenko Marianna Valeriivna, PhD in medicine , associate professor

Buryachkivskyi Eduard Stanislavovych, PhD in medicine, associate professor

Narbutova Tamara Evgenivna, PhD in medicine, assistant

Vasyliev Vladyslav Vadymovych, assistant

Syvyi Sylvester Mykhailovych, assistant

Practical lesson No. 1

Topic: Introduction to pathomorphology. The subject and tasks of pathomorphology. Methods and techniques of patho-anatomical diagnostics. The main stages of the development of pathomorphology. Rising level of knowledge. Morphological changes of cells as a response to stressful and toxic damage (parenchymatous/cellular dystrophies). Cellular dystrophies: hyaline-droplet, hydropic, fatty.

Goal: To study the goals, tasks, objects of pathomorphology and methods of pathomorphological studies, the main stages of the development of pathomorphology, the morphology of reversible and irreversible damage to cells and tissues, intracellular accumulation of proteins, carbohydrates and lipids and to interpret these morphological changes.

Basic concepts:pathomorphology, pathological process, dystrophy, consequences and complications of intracellular and extracellular accumulation of proteins, carbohydrates and lipids and interpret these morphological changes

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:

emphasize the definition or provide an explanation. Pathomorphology, microscopy, biopsy, autopsy, electron microscopy; staining according to Van Gieson, Sudan 3, hematoxylin-eosin, toluidine blue. Dystrophies: intracellular, stromal-vascular, mixed, hereditary, acquired, protein, carbohydrate, fat, pigment, mineral; goose liver, tiger heart, etc.

2.2. block diagram on the topic as a list of didactic units of the topic

Pathological anatomy consists of two parts: general - studies general pathological processes (dystrophy, necrosis, inflammation, etc.) and special - studies the morphology of processes that make up the structural basis of diseases. In combination with each other, these processes acquire a new quality, making up the material maintenance of various syndromes and diseases.

Tasks of pathological anatomy:

Pathological anatomy is essentially a clinical discipline that solves the following tasks: - study of etiology - the causes of pathological processes and diseases;

- study of pathogenesis - mechanisms of development of pathological processes and diseases;

- study of morphogenesis - the structural basis of the pathogenesis of diseases and pathological processes;

- study of sanogenesis - the structural foundations of the body's recovery processes;
- study of thanatogenesis - mechanisms of death;
- study of pathomorphosis - clinical and morphological signs of the disease that arise during treatment (therapeutic pathomorphosis);
- development of iatrogenic problems - study of diseases and their complications, which arose as a result of medical manipulations and medical errors;
• development of diagnostic problems - refers to various aspects of teaching about diagnosis.

Levels of research on the structural basis of diseases.

Pathological processes and diseases are studied at different levels of the structural and functional organization of living matter:
• organismal level - makes it possible to observe the disease of a whole organism
• organ level - involves the study of pathological processes in organs
• system level - involves the study of pathological processes within a separate system (blood system, cardiovascular system, connective tissue system, etc.)
• tissue level - the study of pathological processes in various tissues
• cellular level - study of cells and intercellular substance using a light microscope
• subcellular level - study of the ultrastructural pathology of the cell and the intercellular environment
• molecular level - study of changes in the fine structure of protein macromolecules, nucleic acids, etc.

Material (object) and research methods.

The material for the study of pathological anatomy is obtained during the autopsy of dead patients, surgical interventions, biopsies, and experiments.

Autopsy (necropsy, autopsy, dissection) is one of the classic research methods in pathological anatomy. Thanks to this method, the clinical diagnosis is verified, possible errors in its formulation are revealed (various degrees of discrepancy between clinical and patho-anatomical diagnoses), the cause of the patient's death, features of the course of diseases, the effectiveness of their treatment are established, statistics of mortality, lethality, etc. are developed.

Biopsy (from the Greek. Bios - life, orsis - vision) is a method of intravital examination of a piece of organ or tissue. Thanks to the biopsy, the clinical diagnosis is verified during the patient's life, it becomes possible to study the dynamics of the pathological process at different levels of the structural organization of living matter, the expediency and adequacy of clinical methods of examination and treatment of the patient, the prognosis of the disease and the possible consequences of adverse effects of drugs. Under these conditions, a pathologist becomes a clinical pathologist, a full participant in the diagnostic and treatment process.

The experimental method is necessary for studying the mechanism of disease development (pathogenesis), the dynamics of structural changes underlying it (morphogenesis), the action of certain drugs, and testing various methods of surgical interventions. A certain limitation in the use of this method is explained

by the fact that it is impossible to obtain models of some human diseases in an experiment (ulcer disease of the stomach and duodenum, rheumatism, typhoid) or the changes observed in the experiment are not adequate to those found in pathology in humans (atherosclerosis, hypertension, etc.).

Immunohistochemical study. In some pathological conditions, especially tumors, it is difficult and even impossible to determine the type of tissue or its origin (histogenesis) with the help of histological or cytological staining. Similar difficulties arise when determining the type of causative agent of infection. Meanwhile, verification is important here for diagnosis and forecasting. Therefore, various additional methodological approaches are used. One of them is the immunohistochemical method. In this case, cytological preparations additionally apply solutions with antibodies to the desired antigens - tumor, viral, microbial, autoantigens, etc. Antigens are invisible with conventional histological staining. Antibodies in sera carry a label: either a fluorochrome, that is, a dye that glows in the dark (in other words, one that gives fluorescence), or a staining enzyme.

Methods of molecular biology. In well-equipped pathology departments and scientific research institutes, methods of molecular biology are used for end-of-life diagnostics: flow cytometry and the in situ hybridization technique, that is, on the spot, on a histological section.

Study of chromosomes. Chromosome analysis is carried out in numerous modern pathology departments and scientific research institutes, which allows to determine deviations in the genetic apparatus (genome) of cells, which have an innate or acquired character.

Electron microscopy. In the course of diagnostic studies on the material taken during the patient's life, electron microscopy is often used - transmission (in a passing beam, similar to light-optical microscopy) and scanning (removing the surface relief). The first is used more often, especially for studying the details of cell structure in ultra-thin tissue sections, identifying microbes, viruses, deposits of immune and other complexes, etc.

Dystrophy as one type of cell damage

Dystrophy is a general pathological process, which is characterized by a violation of metabolism in cells or tissues, which is accompanied by their accumulation or redistribution. Morphologically, these processes are characterized by the appearance of structural changes in the cell or intermediate tissue, that is, their damage or alteration occurs.

The main forms of cell damage are:

ischemic (hypotoxic) damage;

free radical damage;

toxic damage.

The main causes of dystrophy are violations of cellular and extracellular trophic mechanisms, which include:

violation of cell autoregulation processes;

violation of the function of transport systems of food (blood, lymph, microcirculatory channel, intermediate tissue);

violation of the coordination of integration systems of trophic.

Mechanisms and classification of dystrophies

infiltration - penetration and accumulation in cells and interstitial tissue of products of metabolic disorders from blood and lymph;

transformation - the transition of substances (proteins) from a labile state to a stable one or the formation of substances of one type of metabolism from common derivatives of those products from which proteins, fats, carbohydrates are obtained, for example, the polymerization of glucose into glycogen, the formation of hyaline in the walls of vessels, the transformation of components of fats and carbohydrates into proteins.

pathological synthesis - the synthesis of cellular substances that are not normally characteristic of it (alcohol hyaline in hepatocytes, the appearance of glycogen in the cells of the thin section of the loop of Henle in diabetes, abnormal protein synthesis in plasmacytoma);

decomposition (phanerosis) - disintegration of membrane structures (cytoskeleton) of the cell by destruction of lipoprotein complexes (fibrinous necrosis in rheumatism, obesity of hepatocytes in diphtheria).

Dystrophies are classified according to different principles:

I. Depending on the type of disturbed exchange:

a) protein (dysproteinoses);

b) fat (lipidosis);

c) hydrocarbons;

) mineral.

II. Depending on the localization of the process:

1) parenchymatous (cellular);

2) stromal-vascular (mesenchymal, extracellular);

3) mixed.

III. Depending on the origin:

1) acquired nature;

2) hereditary.

IV. Depending on the prevalence of the process:

a) local;

b) general.

Parenchymatous (intracellular) dystrophies.

Dystrophies are called parenchymatous, in which the products of impaired metabolism accumulate in the parenchyma of internal organs, that is, in highly specialized cells of the heart, kidneys, and liver.

There are the following types of parenchymal dysproteinoses: "granular" dystrophy, hyaline-droplet dystrophy, vacuolar (hydropic) dystrophy, pathological keratosis. Macroscopically, with parenchymal dysproteinosis, the organs increase in size, become lethargic to the touch, on cross section, their parenchyma (kidneys) breaks above the capsule, loses its luster, and becomes dull.

Microscopicallycharacteristic is the appearance of granularity in the cytoplasm of cells, their swelling, hydration, intercellular borders become blurred, specialized structures disappear (brush border of nephrocytes, striated somatic muscles). The isolated appearance of granularity in the cytoplasm without signs of damage to various cell structures cannot be considered as an independent form of dysproteinosis, as it can be a reflection of intracellular regulatory processes, hyperplasia of ultrastructures or observed in conditions of cell hyperfunction.

Parenchymatous lipidosis.

Parenchymatous lipidosis is characterized by a violation of the metabolism of cytoplasmic fat, which is manifested in its excessive accumulation in the cell, the appearance of fat where it is not normally found, or the formation of fat of an unusual chemical composition. Most often, neutral fat accumulates in the cells. Fats are detected using special reactions: the color of Sudan III and scarlet fever - red color, Sudan IV and osmic acid - black color, Nile blue sulfate - red color (neutral fat). With conventional methods of histological processing of the material, fat in the cage is not detected.

Among the morphogenetic mechanisms of dystrophy, the following are distinguished: infiltration, transformation, pathological synthesis and decomposition. In the pathology of cells, it is difficult to give preference to any of them, since the influence of one mechanism on another is observed or their close contact occurs. Most often, fatty dystrophy occurs in the myocardium, liver, and kidneys.

The causes of fatty dystrophy of the myocardium are hypoxia and intoxication, which are observed in anemia, chronic cardiovascular failure, arsenic, phosphorus poisoning, diphtheria, alcohol consumption, etc. In these cases, the morphogenetic mechanisms of the process are determined depending on the type of cell damage: hypotoxic or toxic.

During hypoxia, there is a decrease in oxidative phosphorylation in cardiomyocytes, the mechanism of anaerobic glycolysis is "turned on", which leads to a decrease in ATP synthesis, damage to mitochondrial structures and disruption of fatty acid b-oxidation processes, as a result of which small drops of lipids accumulate in the cytoplasm.

In case of intoxication, not so much lipophanerosis occurs due to the breakdown of lipoproteins of membrane complexes, but rather the process of destruction of mitochondria. This is accompanied by a decrease in □-Oxidation of lipids, which accumulate in the sarcoplasm.

Macroscopicallythe size of the heart increases, its chambers expand, the myocardium becomes sluggish, yellow striations appear on the endocardium in the area of the papillary muscles ("tiger heart").

Microscopicallyfatty dystrophy is focal in nature: fat accumulates in groups of cardiomyocytes located along the venous bed of capillaries and small veins, which corresponds to the macroscopic appearance of characteristic striations from the endocardium.

Fatty liver dystrophy is observed in alcoholism, diabetes, intoxication, obesity, as well as in nutritional disorders due to insufficient protein in the daily diet, etc. It is based on the accumulation of neutral fat in hepatocytes, which occurs as a result of impaired enzymatic cell systems, that is, enzymopathy.

Its development is due to the following mechanisms:

- excessive penetration of triglycerides (neutral fat) and fatty acids into the hepatocyte;
- violation of the processes of utilization (oxidation) of fatty acids in the mitochondria;
- disruption of lipid elimination processes outside the cell;

hereditary defect of enzymes involved in fat metabolism.

Parenchymatous carbohydrate dystrophies.

Parenchymatous carbohydrate dystrophies occur when glycogen or glycoprotein metabolism is disturbed. Glycogen is formed as a result of the polymerization of glucose. The main representatives of glycoproteins are mucins and mucoids. Mucoid (mucous substance) is included in the composition of various tissues (main substance), mucins are the basis of mucus, which is produced by the epithelium of mucous membranes or glands.

In diabetes, there is an inefficient use of glucose by tissues, as a result of which its level in the blood (hyperglycemia) and urine (glucosuria) increases. Glycogen synthesis increases in the liver, fatty dystrophy of hepatocytes occurs, glycogen granules accumulate in the epithelium of the narrow knee of Henle's loop and distal convoluted tubules: as a result of resorption of glucose from provisional urine, its polymerization occurs in cells with the formation of glycogen.

Glycogenoses (storage diseases, or thesaurismoses) occur when there is a hereditary absence of the enzyme involved in the breakdown of glycogen. Today, 6 types of them are known, caused by the insufficiency of various enzymes: Gierke's disease, Pompe's disease, Hers' disease, etc.

Their macro-micromorphology and ultrastructure are different depending on the type of parenchymal dystrophy.

Yes, Macroscopically, the kidneys with dull swelling are enlarged in size, have a sluggish consistency, lack blood, with a dull shade. On the section, the parenchyma with signs of swelling, along the edges of the section, extends beyond the boundaries of the connective tissue capsule. The surface of the cut loses its shine, becomes dull, grayish in color, reminiscent of meat dipped in boiling water. A similar picture is sometimes observed as a result of postmortem

changes. The main signs of the end-of-life process macroscopically are more significant swelling of the organ, and microscopically - an increase in the size of cells.

Microscopicallythe epitheliocytes of the convoluted proximal tubules are increased in size, protrude into the lumen of the tubules, as a result of which they narrow and become slit-like. The cytoplasm of the cells is opaque, contains pink protein grains of different sizes. The nuclei are weakly contoured and have a basal localization. Homogeneous or fine-grained masses of protein (protein cylinders) are in the lumen of the tubules.

Colloidal dystrophy of the thyroid gland is microscopically characterized by enlargement of follicle lumens, which acquire an irregular shape, their epithelium becomes cubic, located on the basement membrane. In the follicles there is an accumulation of thick masses of homogeneous pink colloid.

In the case of mucous dystrophy (gastric cancer), hypersecretion of mucus in the form of pale pink clusters, in which there are mucus-forming cancer cells with a hyperchromic nucleus located on the periphery ("ring-shaped" cells), is observed microscopically in the tumor.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. General organization of the pathomorphology course
2. Definition of pathological anatomy (pathomorphology) as a subject
3. Development of pathomorphology as an independent science
4. Goals and objectives of our subject
5. Methods of pathological anatomy: autopsy and biopsy (with macroscopy, microscopy: light, phase-contrast, luminescence microscopy, histochemistry, immunohistochemistry, electron microscopy, genetic studies, etc.).
6. General information about pathogenic factors (endogenous and exogenous)
7. Types of cellular reactions (cellular response) to damage and development pathologies.
8. Definition of dystrophies (degenerations), causes of their development.
9. Mechanisms of development of dystrophies.
10. Types of dystrophies according to various signs, their morphology, characteristics.
11. Intracellular (parenchymal) dystrophies, classification.
12. Intracellular protein dystrophies: granular, hyaline-droplet, hydropic, keratoid. Morphological characteristics, complications, consequences
13. Intracellular fatty dystrophy: dust-like, small droplet, large droplet; fatty degeneration of the heart, kidneys, liver. Morphological characteristics, complications, consequences.
14. Intracellular carbohydrate degeneration. Accumulation diseases.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 Test tasks

1. A 25-year-old patient with a clinical picture of nephrotic syndrome underwent a puncture biopsy of the kidney. During microscopic examination, the cells of the epithelium of the proximal tubules of the nephron are enlarged in volume, in the cytoplasm there are vacuoles with a transparent liquid, the nucleus is shifted to the periphery. What kind of dystrophy was found in the epithelium of the tubules?

And hydropic

B Grainy

CZhirova

Rohova

EHyaline droplet

2. During a histological examination of the myocardium of a 47-year-old patient with rheumatic heart disease (sectional material), large optically empty vacuoles were found in cardiomyocytes. When stained with osmic acid, they are black, when stained with Sudan III - yellow-red. Name the type of pathological process:

Liver dystrophy

BHyaline droplet dystrophy

Hydropic dystrophy

DCarbohydrate dystrophy

Dysproteinosis

3. A 35-year-old woman was diagnosed with pharyngeal diphtheria. She died of acute heart failure. On autopsy: the heart cavity is widened in cross section, the heart muscle is dull, flaccid, variegated on cross-section, with yellowish areas under the endocardium. What type of dystrophy is detected in cardiomyocytes?

AND Zhirova

B Hydrocarbon

CBalloon

DHyaline droplet

Hydropic

4. At the autopsy of a 45-year-old woman who died of chronic alcohol intoxication, the liver was sharply enlarged, had a dough-like consistency, and was yellowish in color. Microscopically, optically empty vacuoles of various sizes are found in the cytoplasm of hepatocytes when stained with hematoxylin and eosin. What kind of dystrophy does the place have?

Aparenchymatous fatty

BCarbohydrate parenchymatous

CHyaline droplet

DMesenchymal fat

Hydropic

5. At the autopsy of a 45-year-old woman who died of chronic alcohol intoxication, the liver was sharply enlarged, had a dough-like consistency, and was yellowish in color. Microscopically, optically empty vacuoles of various sizes are found in the cytoplasm

of hepatocytes when stained with hematoxylin and eosin. What kind of dystrophy does the place have?
Aparenchymatous fatty
BCarbohydrate parenchymatous
CHyaline droplet
DMesenchymal fat
Hydropic
6. At the autopsy of a 49-year-old man who was hospitalized with hepatotropic intoxication and died suddenly, the liver was enlarged, flabby, yellow-brown in color; drops of fat are visible on the surface of the liver cut and the knife blade. Microscopically: the hepatocytes of the periphery of classical liver lobes contain a mass of small droplets that fill the cytoplasm and push the nucleus to the periphery. Which process most likely takes place in the liver?:
Liver dystrophy
BSphingomyelin lipidosis (Niemann-Pick disease)
Generalized gangliosidosis (Norman-Landing disease)
Ganglioside lipidosis (Tay-Sachs disease)
Cerebroside lipidosis (Gauche disease)
7. The child was diagnosed with pharyngeal diphtheria in the clinic. She died of acute heart failure. At the autopsy, it was found that the cavities of the heart are expanded in diameter, the heart muscle has a dull, lethargic, variegated appearance, with yellowish areas. In the cytoplasm of some cardiomyocytes with preserved cytoplasm, small vacuoles are detected, on frozen sections, vacuoles are stained with Sudan III in a yellow-hot color. What type of dystrophy is detected in cardiomyocytes?:
Azhirova
BHyaline - dropsy
Hydropic
D Hydrocarbon
EBalloon
8. At the autopsy of the corpse of a woman who died with symptoms of heart failure, the heart is enlarged in volume, flabby; myocardium - clay-yellow, dull; from the side of the endocardium, a yellow-white streak is visible (tiger heart). Microscopically: the groups of cardiomyocytes do not have transverse striations, the cytoplasm of cardiomyocytes contains small drops that are stained black with Sudan IV. Your diagnosis?:
Liver dystrophy of the myocardium
BCardiosclerosis
Rheumatic myocarditis
Heart obesity
EMyomalacia
9. In a 62-year-old man who died due to increasing symptoms of heart failure, an enlarged heart was found at autopsy. The heart has a flaccid consistency, the chambers are stretched, the myocardium is dull, clay-yellow on cross-section. From the

endocardium, a yellow-white striation is visible, which is especially pronounced in the papillary muscles. What is the most likely pathological process?:
A Fatty dystrophy of the myocardium
IN Myomalacia
WITH Cardiosclerosis
D Heart obesity
IS Dilated cardiomyopathy
10. In a woman with severe intoxication due to sepsis, which served as the direct cause of death, an autopsy revealed a "tiger heart". Microscopically, lipids were found in the cytoplasm of cardiomyocytes. What morphogenetic mechanism of development mainly underlies this dystrophy?:
Decomposition
BSedimentation
CTransformation
D Infiltration
Distorted synthesis

3.2. Algorithm of description of macropreparation and micropreparation

Micropreparations.1. Fatty dystrophy of the liver (color Sudan III, m.zb.) Pay attention to the fact that in fatty liver dystrophy, the formation of fat droplets begins in the protoplasm of hepatocytes on the periphery of the lobes, therefore, here the drops of fat (red color) are the largest, and in the centers of the particles are smaller. Label: 1-small-drop fatty dystrophy, 2-large-drop fatty dystrophy

2. Hydropic dystrophy of the kidney tubule epithelium (G+E, m.zb). Vacuoles are visible in the cytoplasm of nephrocytes (1), the lumen of the tubules is not determined; in the cavity of the glomerular capsule - a protein liquid (2).

Macro drug.Represented by a liver measuring 25X13X11 cm. Yellow-green color. The surface of the organ is smooth, the edges are rounded, the consistency is testy. On the section of the liver, it is dark with drops of fat. A greasy mark remains on the knife. Conclusion: parenchymatous (intracellular) fatty dystrophy of the liver — "goose liver"

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the

statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Morphological changes of the extracellular matrix (stroma) as a response to damage (stromal-vascular dystrophies). Pathomorphology of extracellular accumulation of complex proteins (hyalinosis), fats and lipids. Exhaustion of the body. Violations of metabolism and their metabolism. Morphology of pathological accumulation of endogenous and exogenous pigments. Morphology of mineral metabolism disorder".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition.

Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 2

Topic: Morphological changes of the extracellular matrix (stroma) as a response to damage (stromal-vascular dystrophies). Pathomorphologyextracellular accumulation of complex proteins (hyalinosis), fats and lipids. Exhaustion of the body. Violations of metabolism and their metabolism. Morphology of pathological accumulation of endogenous and exogenous pigments. Morphology of mineral metabolism disorder.

Goal: To study the irreversible damage of cells and tissues, extracellular accumulation of proteins, carbohydrates and lipids, stages of disintegration of connective tissue and to interpret these morphological changes.

Basic concepts: Hyalinosis, amyloidosis, obesity, glazed spleen, sebaceous spleen, sago spleen, simple obesity heart, large white kidney, melanosis, calcification, stone formation, hemoglobinogenic pigments, jaundice, hemosiderosis.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:

*emphasize the definition or provide an explanation.*Hyaline and its types, hyalinosis and its types, amyloid, amyloidosis and its types, obesity and its types, glazed spleen, sebaceous spleen, sago spleen, simple obesity heart, large white kidney, sebaceous kidney, lipomatosis, melanosis, calcification and its types , stone formation (stone disease and its types: nephrolithiasis, urolithiasis, sialolithiasis, gallstone disease) hemoglobinogenic pigments, jaundice and its types, hemosiderosis and its types;

2.2. Flow chart on the topic as a list of didactic units of the topic

Stromal-vascularare called dystrophies in which productsviolatedmetabolismaccumulatein intermediate (connective) tissue and vessel walls.

INdepending onspecies violatedmetabolism is distinguished:stromal-vascular proteinsdystrophy (dysproteinoses),stromal-vascular fattydystrophy (lipidosis) andstromal-vascular hydrocarbonsdystrophy

amongstromal-vascular dysproteinosesdistinguish:mucoid swelling,fibrinoid swelling(Disorganization, necrosis),hyalinosisandamyloidosis.mucoid swelling-fibrinoid swelling-hyalinosisisstagesone and the samesame process(For example, in rheumatic diseases).amyloidosisis an independent formdysproteinosis.

mucoid swelling- viewdysproteinosis, which is characterized by an uneven redistribution of acidicglycosaminoglycans(mainlyhyaluronicacids) in the main substance andswelling collagenfibers

It is observed most often in rheumatic diseases, characterized by an increase in vascular and tissue permeability. As a result, it happensswellingof the main substance andcollagenfibers in the composition of connective tissue and vessel walls (basophiliamain substance,decrease fuchsinophiliafibers). The process is reversible, but if its cause is preserved, the effect of toxic immune complexes in rheumatism passesfibrinoid swelling.

fibrinoid swelling- viewdysproteinosis, in which it is observedmoredeep damagecollagenfibers and main substance,which onecharacterized by a significant increase in vascular tissue permeability and outputwithblood fibrinogen.

At the heart of the dystrophic process is the appearance of a qualitatively new substance,which oneis calledfibrinoidand has hematogenous (exitwithblood fibrinogen and otherscoarsely dispersedproteins) andhistiogenic(productsdecay collagenfibers and main substance)origin. The main mechanisms of its development are infiltration and decomposition.

when paintingpicrofuchsin collagenfibers losefuchsinophilia, andare strugglingwith silver (reactionGomorrah), become pironinophilic(reactionBrush),metachromasiaof the main substance disappears (as a result of depolarization of acidicglycosaminoglycans).

Process, as opposed tomucoid swelling, Has an irreversible character, occurs in the wallsarteriole, Heart valves,wall-mountedisndocardiwith rheumatic diseases, endsfibrinoidnecrosis (disorganization) andhyalinosis.

hyalinosis(fromGreek"Hyalos" - transparent, vitreous) - typedysproteinosis, in which the connective tissue or vessel wallsaccumulatecompacted, translucent masses of protein that resemblehyalinecartilage.

the main componenthyalineisfibrillarwhite. In addition, in hisstorageinclude blood plasma proteins, including fibrin, lipids, components of immune complexes (complement, immunoglobulins).

At the heart of the mechanismhyalinosisthere are processes of destructioncollagenfiberswithfurther impregnationtheirvarious blood plasma components (fibrinogen, globulins, immune complexes,lipoproteins) Against the background of increased vascular and tissue permeability (plasmarrhagia).

distinguishhyalinosisvessels andhyalinosisconnective tissue. In the first case (vascularhyaline)insmall arteries are involved in the processcaliberandarterioles. At the same time, a significant increase in permeability is observedtheir, Plasmarrhagia,hyalineappearsin subendothelialspaces, muscle atrophy gradually occurslayer,narrowingvessel lumen, homogenization of their walls.

changesin the walls of blood vessels have a hemodynamic,metabolicor immune nature. Therefore vascularhyalinecan be different: a) simplehyaline(Hypertensive disease, atherosclerosis,changesage-related); b) hereditaryhyaline- consisting ofhim are foundimmune complexes, fibrin, decay productscollagenfibers (rheumatic diseases); in)lipohyaline- consisting ofhim are foundlipids and b-lipoproteins(diabetes:diabetic microangiopathy).

hyalinosisconnective tissue itself occurs with hypoxia, chronic inflammation, scleroticchanges, withfibrinoiddisorganization, etc. At the same time, bundlescollagenfibers are lostfibrillation, become homogeneous, transforminginstructureless masscartilaginousconsistency

atvascularhyalinosis, atrophy is observed,changeforms andconsolidationinternal organs (for example, a primary compacted kidney in hypertensive disease); athyalinosis, in fact, of the connective tissue, it becomes significantly compacted, translucent, grayish in color (for example,hyalinosisvalve leaflets atmitralstenosis).INDepending on the localization (hyalinosis arteriole, valve leaflets) and the prevalence of the process may cause functional failure of organs (heart - in hypertensive disease, kidneys - inchronic glomerulonephritis). In other caseshyalinosisNOTcausesnoticeablechanges(hyalinosisscar, andnonvolutive changesin separate bodies andothers).

Stromal-vascular lipidosis.

Stromal-vascular lipidosisoccur when the metabolism of neutral fats is disturbed orcholesteroland its ethers. This form of dystrophy can be conditionally divided intotwogroups

AND.magnification fatin adipose tissue - at the same time fataccumulates infat depots, which is calledobesity. Classificationhimis built on different principles.

1) by external signs:
a) symmetrical type;
b) upper type;
c) average type;
d) lower type.
2) by the mechanism of development:
a) alimony form;
b) cerebral form;
c) endocrine form;
d) hereditary.
3) by percentage of predominance to body weight:
and)ANDdegree 20-29%;
b) II degree 30-49%;
c) III degree 50-59%;
d) IV degree more than 100%.
4)inDepending on the quantityadipocytesand their sizes:
a) hypertrophic type;
b) hyperplastic type.
Morphologically, heart obesity ("simple obesity") acquires the most important importance. The heart increases in size, is surrounded by fat, like a case. Fat sprouts betweencardiomyocytes, Causing their atrophy. This process is most pronounced in the stroma of the right ventricle, where myocardial rupture can occur most often.
II.violationthe exchange of cholesterol and its esters is most often observed in the walls of the aorta and large-caliber vessels and is the subject of study next semester.

Stromal-vascularcarbohydrate dystrophies.

Stromal-vascularcarbohydrate dystrophies arise as a result of the appearance in the tissues of an imbalance betweenglycoproteinsandglycosaminoglycans.violationexchangeglycoproteins causesslimming of tissues. Withchromotropicsubstances are released from compoundswithprotein andaccumulatein the intermediate tissue, occurssubstitution collagenfibersmucousmass, stellate cells appearwithprocesses of irregular shape.slimetissues is observed with myxedema, cachexia of any kindgenesis.violationexchangeglycosaminoglycansis most often hereditary and is observed in diseases of accumulation, ormucopolysaccharidoses(Example,gargoyle: Characteristic is "tower» Skull,splenomegaly,hepatomegaly, heart disease,umbilicalor inguinalhernias).
So,stromal-vasculardystrophies occur as a resultviolation exchangeproteins,fatsand carbohydrates and are characterizedaccumulationproductsmetabolism instroma of organs and vessel walls. In some cases, the dystrophic process is staged (for example, withdysproteinosis:mucoid swelling–fibrinoid swelling-hyalinosis).
progressiondystrophicchangesclinically characterizedviolationorgan functionswiththe development of their deficiency,which onedetermines the peculiarities of the clinical

symptoms of the process (hyalinosisheart valves atmitralstenosis, developmentnephrotic syndromeatamyloidosiskidneys andothers)
Macro-micromorphologyand ultrastructuralchangesare different in internal organs, which underlie the different nature of their functional insufficiency.
Thus, with obesity of the heart, macroscopically, the organ is enlarged in sizein1.0-1.5 times, sluggish, under epicardsignificant growth of adipose tissue, heart chambers are enlarged, cardiacmusclebrown-brown in section, valves are thin, translucent, chords andtrabecularmuscles without changes, wall thicknessbothventricles is reduced.
microscopically observedexcrescenceadipose tissueininterstitialmyocardium with atrophic changes cardiomyocytes.
Athyalinosisvessels of the spleen draw attention to themselves microscopicallychangessmall vesselscaliber- central arterieslymphoidfollicles andarteriolered pulp. Their walls are thickened,enlightennarrowed, the number of cellular elements is reduced.hyalinein the form of homogeneous homogeneous masses appears under the endothelium and is uniformly deposited along the perimeter of the openings of vessels. In separate places of accumulationhimhave an uneven character. Endothelium in vessels is preserved. The masseshyaline, which are underby him, Delimited outside m. elastica interna, whichit turns outwhen dyeing on elastic fabric. Sometimes it is split, and its thin fibers are chaotically surrounded by hyaline masses. Muscle atrophy gradually occurs along the periphery of the latterlayer, And atsignificanttheir accumulation, the division into layers in the vessel walls disappears altogether, and they acquire a homogeneous, uniform character.
Athyalinosis arteriolemicroscopically observedthickeningwalls that are acquiredhomogenouspink color, the number of cellular elements decreases, the lumen narrows.
atmucoidswellingvalvehearts (histochemicalreaction according to Hale) the valve leaf is thickened, there is an uneven accumulation of acidic substances in the main substanceglycosaminoglycans(hyaluronicacid) in the form of blue-green areas, there is no cellular reaction.

2.3. List of questions to check basic knowledge on the subject of the lesson.
1. Concept of dystrophy (degeneration), classification, mechanisms of development.
2. Classification of stromal-vascular (mesenchymal) dystrophy, morphological characteristics.
3. Stromal-vascular protein dystrophy, types, stages of connective tissue disorganization.
4. Mucoid swelling. morphological characteristic, consequences.
5. Fibrinoid swelling, morphological characteristics, consequences.
6. Hyalinosis: types of hyalinosis, morphological characteristics, consequences. Types of hyaline.
7. Amyloidosis: classification, causes of development. Morphological characteristics, consequences, complications, causes of death.
8. Stromal-vascular fatty dystrophy, causes of development, classification.

9. Obesity, atherosclerosis, lipomatosis as variants of stromal-vascular dystrophy: morphological characteristics, consequences.
10. Stromal-vascular carbohydrate dystrophy: types, causes of development, morphological characteristics, consequences.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 Test tasks

1. A 42-year-old man, who had suffered from chronic granulomatous periodontitis, chronic purulent osteomyelitis of the lower jaw for 8 years, died of chronic kidney failure. What complication of purulent osteomyelitis developed in the kidneys?
AND Amyloidosis
B Hyalinosis
Fatty dystrophy
Atrophy
ENecrosis of the epithelium of convoluted tubules
2. Macroscopically, the liver is enlarged, compacted, the tissue is grayish-yellow in color, with a greasy sheen. What pathological process is the basis of the described changes?
AND Amyloidosis
B Hyalinosis
Fatty dystrophy
DMucoid swelling
EHemachromatosis
3. The operatively excised connective tissue of the deformed mitral valve gives a basophilic reaction when stained with hematoxylin and eosin, and under the influence of toluidine blue it turns lilac (metachromasia). What changes in the connective tissue are revealed by these types of reactions?
AND Mucoid edema
BFibrinoid necrosis of connective tissue
Swelling of connective tissue
Petrification
Hyalinosis
4. A 58-year-old patient suffered from chronic osteomyelitis of the lower jaw for many years. Recently, in the blood analysis - hypoproteinemia, dysproteinemia; in urine - proteinuria, protein cylinders. He died of chronic kidney failure. On autopsy, the kidneys are enlarged, dense, wax-like. What pathological process in the kidneys was revealed at the autopsy?
AND Amyloidosis
B Chronic glomerulonephritis
Hydronephrosis

Chronic pyelonephritis
Interstitial nephritis
5. Macroscopically, the liver is enlarged, compacted, the tissue is grayish-yellow, with a greasy sheen. What pathological process is the basis of the described changes?
AND Amyloidosis
B Hyalinosis
Fatty dystrophy
DMucoid swelling
EHemachromatosis
6. A 19-year-old man suffered from bronchoectopic disease since early childhood and died of kidney failure. At autopsy, in addition to multiple bronchoectopic cavities filled with purulent exudate, enlarged kidneys of a dense consistency were found, the cortical layer was thickened, white, dense, the pyramids were clear, and the kidneys were anemic. Name the process that developed in the kidneys?:
AND Secondary amyloidosis
BSecondary nephrosclerosis
C Glomerulonephritis
Chronic pyelonephritis
Congenital polycystic kidney disease
7. At autopsy, the 58-year-old deceased had a deformed, thickened mitral valve that did not close completely. Microscopically: foci of collagen fibers are eosinophilic, give a positive reaction to fibrin. Most likely it is:
AND Fibrinoid swelling
Amyloidosis
CFibrinous inflammation
D Mucoid swelling
EHyalinosis
8. A 66-year-old patient suffered peritonitis 10 years before his death. At autopsy, the capsule of the liver and spleen is sharply thickened, compacted, translucent in some places. Most likely it is:
AND Hyalinosis
BMucoid swelling
Amyloidosis
DFibrinoid swelling
ENecrosis
9. A 32-year-old woman was ill with infectious-allergic vasculitis for several months. She died of a brain hemorrhage. The wall of the vessels of the microcirculatory channel is significantly thickened, homogeneous, eosinophilic, sharply Schick-Positive, the lumen of the vessels is narrowed. There is no metachromasia when stained with toluidine blue. The reaction to fibrin is positive. Such a microscopic picture indicates the exacerbation of the process and the presence of:
AND Fibrinoid swelling
B Mucoid swelling

Sclerosis (fibrosis)
Dhyalinosis
Amyloidosis
10. The man suffered from diabetes for a long time. He died of a brain hemorrhage. Macroscopically, the small vessels of the brain resemble thickened vitreous tubes of cartilaginous consistency. Microscopically, the wall of arterioles looks homogeneous, eosinophilic, sharply Schick-Positive. These vessels are stained purple and green with methyl violet and iodine-Grun, respectively. Such a microscopic picture indicates the presence of:
AND Lipohyaline
BSimple hyaline
Amyloid
DD. Complex hyaline
EHyaline-droplet dystrophy

3.2. Algorithm of description of macropreparation and micropreparation

Micropreparations: 1. Mucoid swelling of the heart valve (note by toluidine blue, m.zb.) The heart valve (1) and the parietal endocardium (2) are sharply metachromatic due to the accumulation of acidic glycosaminoglycans in them. Moderate lymphohistiocytic infiltration of valve tissue.

2. Hyalinosis of vessels and the white body of the ovary (G+E, m.zb.) The walls of vessels located in the medullary layer of the ovary are sharply thickened, have a homogeneous appearance, are almost devoid of nuclei and are hyalinized; lumens of vessels are sharply narrowed, vessels appear in the form of homogeneous, rounded formations of pink color. The preparation contains large pale pink, homogeneous or with a weakly expressed fibrous structure, scalloped edges of the formation with a small number of nuclei of connective tissue cells - the so-called white bodies. Mark: 1-hyalinosis of the vessel walls of the ovary, 2-hyalinosis of the protein body of the ovary.

Macro drug. It is represented by a kidney measuring 10X6X4 cm, gray-brown in color. The kidney is very dense, has a greasy shine, and its surface is smooth. On the cross-section, the cortical and medulla are poorly differentiated, the parenchyma is thickened in places up to 4.5 cm. Conclusion. Kidney amyloidosis. (Great white kidney, waxy kidney, sebaceous kidney)

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

1. **Evaluation of theoretical knowledge on the subject of the lesson:**
   - methods: survey, solving a situational clinical problem, tests
   - the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.
2. **Assessment of practical skills on the subject of the lesson:**

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Necrosis - definition, terms and phases of development, consequences. Clinical and morphological forms of necrosis. Pathological anatomy of multiple organ failure. Fundamentals of Thanatology. Death, mechanisms, signs. Biological, medical, social aspects due to a chronic incurable disease. The concept of thanatogenesis. Structural mechanisms of cessation of activity of vital organs during the natural course of the disease. Complications of stopping the work of the heart, lungs, brain, kidneys, liver."

Suggested topics for essays.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 3

Topic: Violations of metabolism and their metabolism. Morphology of pathological accumulation of endogenous and exogenous pigments. Morphology of mineral metabolism disorder.

Goal: To study pathomorphological changes of tissues and organs withpathological accumulation of endogenous and exogenous pigments; learn the morphology of mineral metabolism disordersinterpret these morphological changes.

Basic concepts: Melanosis, calcification, stone formation, hemoglobinogenic pigments, jaundice, hemosiderosis.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:

*emphasize the definition or provide an explanation.*Melanosis, calcification and its types, stone formation (stone disease and its types: nephrolithiasis, urolithiasis, sialolithiasis, gallstone disease), hemoglobinogenic pigments, jaundice and its types, hemosiderosis and its types.

2.2. Flow chart on the topic as a list of didactic units of the topic

Mixed dystrophies.

Mixed dystrophies are characterized by metabolic disorders in the parenchyma, stroma, walls of blood vessels, organs and tissues. Mixed dystrophies occur in disorders of the metabolism of complex proteins - chromoproteins, nucleoproteins, lipoproteins, minerals.

Violation of chromoprotein metabolism. Endogenous pigments - chromoproteins - are divided into hemoglobinogenic, proteinogenic or tyrosinogenic and lipidogenic.

Hemoglobinogenic pigments: ferritin, hemosiderin, bile pigments, hematoidin, hematins, porphyrin.

Ferritin is an iron protein containing up to 23% iron. Depending on the origin, anabolic and catabolic ferritin are distinguished. Anabolic ferritin is formed from iron absorbed in the intestine, catabolic from iron in hemolyzed erythrocytes. Normally, ferrin is found in the liver, spleen, bone marrow, and lymph nodes, taking part in the synthesis of hemoglobin, hemosiderin, and cytochromes. In conditions of pathology, the amount of ferritin can increase. The irreversibility of shock accompanied by vascular collapse is explained by ferritinemia, since the active form - SH-ferritin, which has vasoparalytic and hypotensive properties, acts as an adrenaline antagonist.

Hemosiderin consists of a protein - globin and a prosthetic pigment part - heme, it is formed intracellularly in the form of brown grains. It contains iron and is determined by the Perls reaction (under the action of ferric blue potassium and hydrochloric acid, a blue color is formed - "Berlin blue"), turns black from ammonium sulphide. In pathological conditions, excessive accumulation of hemosiderin is observed - hemosiderosis. General hemosiderosis develops with intravascular destruction of erythrocytes and

occurs in diseases of the hematopoietic system, intoxication with hemolytic poisons, some infectious diseases, transfusions of other blood groups, etc. The spleen, liver, bone marrow and lymph nodes become rusty brown.

A disease close to general hemosiderosis is hemochromatosis, which can be primary (impaired absorption of iron in the small intestine) and secondary. The disease is associated with an overload of the body with iron. In the form of ferritin and hemosiderin, it is deposited mainly in parenchymal elements of various organs, as a result of which they acquire a brown color. The result is sclerosis and atrophy of internal organs.

Data on the prevalence of hemochromatosis force one to treat with caution the fortification of food products with iron, which is widely practiced, the consequences of which, in relation to risk groups for the development of microelement overload, are practically not studied.

The classification of microelement diseases associated with iron metabolism disorders is reflected in the Online Mendelian Inheritance in Man (OMIM) register.

Bile pigments - bilirubin, biliverdin, urobilin.

Bilirubin is formed in the reticular cells of the spleen, liver and bone marrow. From these cells, bilirubin enters hepatocytes, where bile is synthesized.

An increase in the content of bile pigments in the blood and staining of tissues in yellow color (jaundice) is observed in various pathological conditions.

Suprahepatic (hemolytic) jaundice occurs with hemolysis of erythrocytes. It occurs with blood diseases (anemia, leukemia), some infectious diseases (malaria, sepsis, typhoid) and intoxications.

Hepatic (parenchymal) jaundice occurs with infections and intoxications (viral hepatitis, sepsis, acute toxic dystrophy of the liver, phosphorus, arsenic, mushroom poisoning). Liver cells lose the ability to synthesize bilirubin and secrete it into the bile ducts.

Subhepatic (mechanical) jaundice develops when the outflow of bile from the liver is obstructed. Occurs with gallstone disease, biliary tract cancer, etc. Bile stagnation leads to stretching of the bile ducts and rupture of bile capillaries. Bile enters the blood, causes jaundice and general intoxication of the body.

Hematoidin is a bright orange crystalline pigment that does not contain iron and is formed outside the cells in the centers of hemorrhages and infarcts under anaerobic conditions.

Hematins are an oxidized form of heme and are formed during hemolysis of oxyhemoglobin. They have the appearance of dark brown or black diamond-shaped crystals or grains, give birefringence in polarized light (anisotropic), contain iron in a bound state, dissolve in alkalis, sparingly soluble in acids, discolored by hydrogen peroxide.

Hematins found in tissues include: malarial pigment (hemomelanin), hydrochloric acid hematin (hemin) and formalin pigment. Histochemical properties of these pigments are identical.

Hemomelanin is a malarial pigment. It is formed in the body of a malarial plasmodium that parasitizes erythrocytes. It has the appearance of black-brown grains. When erythrocytes are destroyed, it enters the blood and undergoes phagocytosis by cells of the reticuloendothelial system. The spleen, liver, lymph nodes, bone marrow, and brain acquire a gray-aspid color.

Hydrochloric acid hematin (hemin) is formed in erosions and ulcers of the stomach under the action of enzymes of gastric juice and hydrochloric acid on hemoglobin. The site of the defect of the gastric mucosa acquires a brown-black color. Hydrochloric hematin crystals in polarized light reveal the properties of anisotropy and dichroism.

Formalin pigment in the form of dark brown needles or granules is found in tissues when they are fixed in acidic formalin (this pigment is not formed if formalin has a pH greater than 6.0). It is considered a hematin derivative.

Porphyrins are precursors of the prosthetic part of hemoglobin, having the same tetrapyrrole ring as heme, but without iron. Porphyrins are chemically similar to bilirubin: they dissolve in chloroform, ether, and pyridine. The method of detecting porphyrins is based on the ability of solutions of these pigments to give red or orange fluorescence in ultraviolet light (fluorescent pigments).

Normally, a small amount of porphyrins is found in blood, urine, and tissues. They have the property of increasing the sensitivity of the body, primarily the skin, to light and therefore play the role of melanin antagonist.

With disorders of porphyrin metabolism, porphyrias occur, which are characterized by an increase in the content of pigments in the blood (porphyrinemia) and urine (porphyrinuria), a sharp increase in sensitivity to ultraviolet rays (photophobia, erythema, dermatitis). Acquired and congenital porphyria are distinguished.

Acquired porphyria is observed with intoxication (lead, sulfazole, barbiturates), vitamin deficiency (pellagra), pernicious anemia, and some

liver diseases. There is a violation of the function of the nervous system, increased sensitivity to light, jaundice often develops, skin pigmentation, a large amount of porphyrins is found in the urine.

Congenital porphyria is a rare hereditary disease. When porphyrin synthesis is disrupted in erythroblasts, the erythropoietic form develops, and when porphyrin synthesis is disrupted in liver cells, the hepatic form of porphyria develops.

Violation of the metabolism of proteinogenic (tyrosinogenic) pigments

Melanin is a black-brown pigment contained in the cells of the epidermis, hair, iris and retina of the eyes. Its composition includes carbon, nitrogen, and sulfur. In the melanoblasts of the basal layer of the epidermis, dioxyphenylalanine is formed from tyrosine under the influence of tyrosinase in the presence of vitamin C, which in turn is transformed into melanin under the influence of tyrosinase. The pigment can be captured by macrophages - melanophages and transported deep into the tissues. Regulation of melanin exchange is carried out by endocrine glands: adrenal glands, gonads, pituitary gland, thyroid gland. There are racial and individual differences in melanin content. A physiological increase in melanin in the skin is observed under the action of ultraviolet rays. Violation of melanin metabolism can be manifested in an increase (hyperpigmentation) and decrease (hypopigmentation) of its content. And that

Hyperpigmentation develops with cachexia, vitamin diseases (pellagra, scurvy), Addison's disease (a sharp decrease in adrenal function with tuberculosis, amyloidosis). Local hyperpigmentation: pigment spots, colon melanosis, chloasma during pregnancy, some tumors (melanoma). General hypopigmentation - albinism (congenital disease). Local hypopigmentation - leukoderma, vitiligo.

Adrenochrome, a product of adrenaline oxidation, is found in the form of granules in the cells of the medulla of the adrenal glands.

The pigment of enterochromaffin cell granules scattered in various parts of the gastrointestinal tract is a derivative of tryptophan. In tumors from these cells, called carcinoids, there are usually many granules containing pigment.

Violation of the metabolism of lipidogenic pigments

Lipofuscin is a glycoprotein in which fats predominate, and phospholipids are among them. Lipofuscin is a normal component of the cell. In pathological conditions, the amount of lipofuscin increases sharply (lipofuscinosis). It can be primary (congenital) and secondary, it is observed most often in the elderly, as well as in debilitating diseases that cause

cachexia (brown atrophy of the myocardium, liver), with increased functional load (lipofuscinosis of the myocardium in heart disease), with phagocytosis (lipofuscinosis of the macrophage) .

Lipochromes are represented by lipids in which carotenoids are dissolved, which are the source of the formation of vitamin A. Lipochromes give a yellow color to adipose tissue, adrenal cortex, and blood serum. In conditions of pathology, there is an excessive accumulation of lipochromes (diabetes). With cachexia, lipochromes condense in adipose tissue, which becomes ocher-yellow.

Ceroid is a lipopigment of mesenchymal cells, mainly macrophages. Ceroid formation is most often observed with tissue necrosis, especially if lipid oxidation is exacerbated by hemorrhage.

Violation of nucleoprotein metabolism

Nucleoproteins are formed from protein and nucleic acids (DNA and RNA). The final product of nucleic acid metabolism is uric acid and its salts. Therefore, the presence of uric acid and its salts in tissues, which is observed in uric acid infarction and gout, indicates a violation of nucleoprotein metabolism.

Uric acid infarction occurs in newborns who have lived for at least two days, and is manifested by the precipitation of amorphous masses of uric acid sodium and ammonium in the tubules and collecting tubules of the kidneys. These cells on the section of the kidney have a triangular shape, reminiscent of a heart attack.

Gout is a disease that is characterized by periodic drops of sodium uric acid in the synovium and cartilage of small joints, ankle and knee joints, in tendons and joint bags, in the cartilage of the auricles. At the place of deposition of salts, necrosis develops, surrounded by an inflammatory reaction with the accumulation of giant cells of the type of foreign bodies - a gouty lump is formed, which may later become covered with ulcers. Often, gout is a congenital metabolic disorder (primary gout), sometimes it is a complication of other diseases (secondary gout), such as nephrocirrhosis, blood diseases, etc.

Urinary stone disease, like gout, can be primarily associated with purine metabolism disorders and be a manifestation of uric acid diathesis. In the kidneys, the accumulation of uric acid and uric acid sodium salts in the tubules with obturation of their lumen, the development of secondary inflammatory and atrophic changes is noted.

Violations of mineral metabolism (mineral dystrophies)

More than 20 elements are involved in mineral exchange. Disturbances in the metabolism of calcium, potassium, copper and iron are of the greatest practical importance.

Calcium is associated with the processes of permeability of cell membranes, excitability of the neuromuscular apparatus, blood coagulation, regulation of the acid-base state, formation of the skeleton, etc. Calcium exchange is carried out by the neurohumoral pathway. Disorders of calcium metabolism in body tissues are called calcification (calcific dystrophy). Its morphological manifestation is the precipitation of calcium salts from the dissolved state and their accumulation in cells or intercellular substance. By prevalence, the process can be general or local.

Calcific dystrophy can be cellular, extracellular and mixed. The process can be systemic (widespread) and local. There are three forms of calcification: 1) metastatic, 2) dystrophic, 3) metabolic.

Metastatic calcification (calcareous metastases) is a general process of calcium release from the depot and delayed removal from the body, which causes calcium to precipitate in tissues and organs with an alkaline environment (artery wall, myocardium, lungs, gastric mucosa, kidney tubules).

Dystrophic calcification (petrification) - has a local character, the precipitation of lime is usually found in dead tissues and tissues with deep dystrophic changes or necrosis (caseous foci in tuberculosis, gums in syphilis, heart attacks, parasites, dead fetus, scars, cartilage).

Metabolic calcification (calcific gout) is a local or systemic disease in which lime accumulates in the skin, tendons, muscles, nerves, and vessel walls. The reason is not established.

Violation of calcium metabolism can be accompanied by a decrease in the amount of calcium in the depot (bone system), occurs in rickets, osteomalacia, parathyroid osteodystrophy.

Rickets is a chronic disease characterized by a change in phosphorus-calcium metabolism with a violation of bone mineralization and the process of bone formation with the development of bone deformations.

Copper is a mandatory component of the cytoplasm, where it participates in enzymatic reactions.

Acquired copper deficiency is rare, mainly in children and adults who are on parenteral nutrition for a long time. Such patients develop anemia and leukopenia.

Congenital disorder of copper metabolism develops in Wilson-Konovalov disease (hepatocerebral dystrophy). An autosomal recessive disease characterized by a decrease in serum ceruloplasmin (a copper-binding protein). The disease is manifested by significant deposition of copper in the cells of the liver, kidneys, brain and cornea. Different types of changes are detected in the liver - chronic active hepatitis, large- or small-nodular cirrhosis. Angiotoxic changes (paralysis of small vessels, stasis, hemorrhages, edema, foci of necrosis, cysts) and cytotoxic changes (dystrophy and necrosis of nerve cells and astroglia; characteristic appearance ugly nuclei, naked nuclei, chromatolysis). A greenish Kaiser-Fleischer ring appears in the peripheral parts of the cornea,

Formation of stones

Stones (concrements) are dense formations lying freely in cavity organs or excretory ducts of glands. Stones are formed as a result of precipitation of salts from liquids located in these cavities or ducts.

The appearance of stones (shape, size, color, structure) is different, depending on localization in one or another cavity, chemical composition, mechanism of formation. There are huge stones and microliths. The shape of the stone often repeats the cavity it fills: round or oval stones - in the urinary and gall bladders, appendages - in the bowls and cups of the kidneys, cylindrical - in the ducts of the glands. Stones can be single or numerous. In the latter case, the stones often have a faceted, polished surface (faceted stones). The surface of stones is not only smooth, but also rough (oxalates, for example, resemble mulberry berries), which injures the mucous membrane and causes its inflammation. The color of the stones is determined by their chemical composition: white (phosphates), yellow (urates), dark brown or dark green (pigment) stones. In some cases, stones have a radial structure (crystalloid), in others - layered (colloidal), in others - layered-radial (colloidal-crystalloid). The chemical composition of stones is also different. Gallstones can be cholesterol, pigment, calcareous or cholesterol-pigment-calcareous (complex or combined stones). Urinary stones can consist of uric acid and its salts (urates), calcium phosphate (phosphates), calcium oxalate (oxalates), cystine and xanthine. Bronchial stones usually consist of mucus encrusted with lime. Urinary stones can consist of uric acid and its salts (urates), calcium phosphate (phosphates), calcium oxalate (oxalates), cystine and xanthine. Bronchial stones usually consist of mucus encrusted with lime. Urinary stones can consist of uric acid and its salts (urates), calcium phosphate (phosphates), calcium oxalate (oxalates), cystine and xanthine. Bronchial stones usually consist of mucus encrusted with lime.

Most often, stones are formed in the biliary and urinary tracts and are the cause of the development of gallstones and urolithiasis. They are also

found in other cavities and ducts: in the excretory ducts of the pancreas and salivary glands, in the bronchi and bronchiectasis (bronchial stones), in the crypts of the tonsils. A special type of stones is the so-called venous stones (phlebolites), which are petrified blood clots that have separated from the wall, and intestinal stones (coprolites), which occur when the contents of the intestine are encrusted.

The pathogenesis of stone formation is very complex and is determined by general and local factors. General factors include all kinds of metabolic disorders (fat, nucleoprotein, carbohydrates, minerals). For local ones - secretion disorders, inflammatory processes. The presence of stones can lead to diseases. Their complications are unfavorable (obturation of the ducts, inflammation, necrosis and perforation of the wall, formation of adhesions and cysts).

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Definition of mixed dystrophies, classification.
2. Definition of pigment, classification of pigments.
3. Classification of endogenous pigments, characteristics.
4. Hemoglobinogenic pigments: physiological and pathological, their pathology, metabolism.
5. Jaundice: definition, classification, causes of development, morphological characteristics.
6. Hemosiderosis: classification, definition, causes of development, morphological characteristics.
7. Porphyria: definition, causes of development, morphological characteristics.
8. Lipidogenic pigments, pathology of their metabolism, causes of development, morphological characteristics.
9. Proteinogenic pigments, pathology of their metabolism, causes of occurrence, development, morphological characteristics.
10. Calcification: classification, causes of development, morphological characteristics, complications and consequences.
11. Violation of copper metabolism. Causes of development, morphological characteristics, complications and consequences.

12. Definition of stone (concrete). Stone formation: classification, causes of development, morphological characteristics, complications and consequences.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 Test tasks

1. During the autopsy of the body, it was found: the lungs are dense, brown in color due to the deposition of endogenous pigment. It is known that the patient had chronic

venous stasis in the small circle of blood circulation during his life. What pathological process caused such a picture?
AND Hemosiderosis
BMelanosis
CZhovtyanitsa
Porphyria
ECalcinosis
2. At the autopsy of the body of a deceased 70-year-old man, the heart is reduced in size, fatty tissue under the epicardium is absent, the myocardium is dense, brown in color. Under microscopy, cardiomyocytes are reduced in size, there are many granules of the brown lipofuscin pigment in the sarcoplasm. Determine the nature of the pathological process in the myocardium:
AND Brown atrophy
Amyloidosis
Fatty dystrophy
D Hemochromatosis
Hypertrophy
3. At the autopsy of a woman who suffered from parathyroid adenoma with hyperproduction of parathyroid hormone and died of chronic kidney failure, lime deposits were found in the stomach, lungs, and kidneys. What is the mechanism of development of calcification?
AND Metastatic
B Metabolic
C Dystrophic
D Mixed
E-
4. A 46-year-old patient with rheumatic stenosis died of chronic pulmonary and heart failure. An autopsy revealed dense brown lungs. What pigment caused the color of the lungs?
AND Hemosiderin
BMelanin
Lipofuscin
Porphyrin
EHemozoin
5. In typhoid fever, necrotized Peyer's patches of the small intestine are yellow-brown in color. What pigment permeates the necrotic tissue?:
AND Bilirubin
BMelanin
Lipofuscin
DHemoglobin
EIndol
6. A patient with a mitral valve defect developed a cough and sputum of a rusty color. What pigment caused this color of sputum?:
AND Hemosiderin
B Hemoglobin
Iron sulfide
D Hemomelanin
EMelanin
7. In a patient with peptic ulcer disease with bleeding during endoscopy, liquid the color of coffee grounds was found in the stomach. What pigment caused this color of the stomach contents?:
And Porphyrin
BFerritin
Hemosiderin

Hydrochloric acid hematin
EBilirubin
8. In a patient suffering from secondary syphilis, foci of skin depigmentation appeared in the upper parts of the back. Name the pathological process in the skin?:
AND Leukoderma
BParakeratosis
Metaplasia
D Leukoplakia
Dysplasia
9. In a patient with an acute stomach ulcer, which was complicated by gastric bleeding, the vomitus is colored dark brown, like "coffee grounds". The presence of which pigment in emetic masses determines their color?:
AND Hydrochloric acid hematin
Bilirubin
Iron sulphide
D Hemomelanin
EHemoglobin
10. A 70-year-old man came to the hospital with complaints of pain in the small joints of his hands and feet. Joints are deformed, painful. An elevated level of uric acid salts in the blood and urine was revealed. What kind of metabolic disorders are we talking about?:
AND Nucleoproteins
B Potassium.
Chromoproteins
Calcium
Lipoproteins
11. An autopsy of a man who died of chronosepsis revealed atrophy of skeletal muscles, brown atrophy of the myocardium, and liver. Violation of the metabolism of which pigment was detected in the deceased?:
AND Lipofuscin
BMelanin
C Hemosiderin
D Lipochrome
Ehemomelanin
12. The man suffered from hemoblastosis for a long time. The autopsy revealed that the bone marrow, spleen, liver, and lymph nodes were brown in color. Perls's histochemical reaction was performed. It was established that reticular, endothelial and histiocytic elements of these organs contain blue granules. What pigment was detected when using the specified reaction?:
AND Hemosiderin
BHematoidin
C Hematoporphyrin
DBilirubin
EHematoidin
13. Cells containing brown pigment were found in the sputum of a patient with mitral heart disease. Perls' reaction is positive. What is this pigment?:
AND Hemosiderin
BMelanin
Bilirubin
Porphyrin
EHematoidin

3.2. Algorithm of description of macropreparation and micropreparation

Micropreparations: 1. Dystrophic calcification of the myocardium (see G.+E, m.zb.) In the tissue of the myocardium, the presence of dystrophic cardiomyocytes is observed, in the cytoplasm of which intensively basophilic (dark purple) accumulations of lime accumulate. Mark: 1. Dystrophic calcification of cardiomyocytes.

2. Skin with Addison's disease (G+E, m.zb.) The drug is presented as a skin fragment. In the deep layers of the epidermis, an increase in the production and accumulation of a large amount of the brown pigment melanin by melanocytes is detected. In the surface layers of the dermis, individual macrophages contain phagocytosed melanin - these are melanophores. Mark: 1. Accumulation of melanin by melanocytes of the epidermis, 2.- melanophores

Macro drug. It is represented by a kidney measuring 10X6X4 cm, gray-brown in color. The kidney at the bottom of the cut has a thickening of the parenchyma up to 1 cm. And the expansion of the pelvis, in which the concretions are located, are smooth yellow-brown stones, 2.0X1.0X1.0 cm in diameter. Conclusion. Nephrolithiasis, hydronephrosis.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

3. **Evaluation of theoretical knowledge on the subject of the lesson:**
   - methods: survey, solving a situational clinical problem, tests
   - the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.
4. **Assessment of practical skills on the subject of the lesson:**
   - methods: assessment of the correctness of the performance of practical skills
   - the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

   The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .

"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Necrosis - definition, terms and phases of development, consequences. Clinical and morphological forms of necrosis. Pathological anatomy of multiple organ failure. Fundamentals of Thanatology. Death, mechanisms, signs. Biological, medical, social aspects due to a chronic incurable disease. The concept of thanatogenesis. Structural mechanisms of cessation of activity of vital organs during the natural course of the disease. Complications of stopping the work of the heart, lungs, brain, kidneys, liver."

Suggested topics for essays.

1. Morphology of gangrene, specific types of gangrene: noma, bedsores, Fournier's gangrene.

5. List of recommended literature (main, additional, electronic information resources):

Main:

4. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
5. The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
6. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

10. http://moz.gov.ua- Ministry of Health of Ukraine
11. www.ama-assn.org– American Medical Association /American Medical Association
12. www.who.int- World Health Organization
13. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
14. http://bma.org.uk– British Medical Association
15. www.gmc-uk.org- General Medical Council (GMC)
16. www.bundesaerztekammer.de– German Medical Association
17. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
18. http://www.webpathology.com/- Web Pathology

Practical lesson No. 4

Topic: Necrosis - definition, terms and phases of development, consequences. Clinical and morphological forms of necrosis. Pathological anatomy of multiple organ failure. Fundamentals of Thanatology. Death, mechanisms, signs. Biological, medical, social aspects due to a chronic incurable disease. The concept of thanatogenesis. Structural mechanisms of cessation of activity of vital organs during the natural course of the disease. Complications of stopping the work of the heart, lungs, brain, kidneys, and liver.

Goal: To study the basic concepts and types of necrosis, its stages and morphological manifestations of each type of necrosis. Learn the consequences and complications of necrosis. Know and be able to recognize the signs of death.

Basic conceptsI: Necrosis, Coagulation (dry) necrosis, Infarct, Caseous necrosis, Zenker's, Fibrinoid, Fat, Enzymatic fatty, Non-enzymatic fatty, Gangrene, Dry gangrene, Wet gangrene, Gas gangrene, Bedsores, Colicative necrosis, Brain infarction, Necrosis, Apoptosis, PON, Death.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop.

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic: emphasize the definition or provide an explanation. Necrosis, Coagulation (dry) necrosis, Infarct, Caseous necrosis, Zenker's, Fibrinoid, Fat, Enzymatic fatty, Non-enzymatic fatty,

Gangrene, Dry gangrene, Wet gangrene, Gas gangrene, Bedsores, Colicative necrosis, Brain infarction, Necrosis, Apoptosis, PON, Death.

2.2. Block diagram on the topic as a list of didactic units of the topic;

Necrosis(From the Greek. Nekros - dead) - death, death of cells and tissues in a living organism under the influence of a pathogenic factor. This type of cell death is not genetically controlled.

Causes of necrosis. Factors that cause necrosis:

physical(Fire injuries, radiation, electricity, low and high temperatures - frostbite and burns)

toxic (acids, alkalis, saltshard metals, enzymes, medicines, ethyl alcohol, etc.);

biological(Bacteria, virus, protozoa),

allergic(Endo - and exoantigens, for example, fibrinoid necrosis in infectious-allergic and autoimmune diseases, the Artus phenomenon)

vascular(Heart attack - vascular necrosis);

trophoneurotic(Decubitus, non-healing ulcers).

Depending on the mechanism of action of the pathogenic factor, the following are distinguished:

direct necrosis, Doom by the direct action of the factor (traumatic, toxic and biological necrosis)

indirect necrosis, which occurs directly due to the vascular and neuroendocrine systems (allergic, vascular and trophoneurotic necrosis).

Clinical and morphological forms of necrosis

Necrosis is manifested by various clinical and morphological changes. The differences depend on the structural and functional features of organs and tissues, the speed and type of necrosis, as well as the causes of its occurrence and conditions of development. Among the clinical and morphological forms of necrosis, coagulation (dry) necrosis and colliquation (wet) necrosis are distinguished.

Coagulation (dry) necrosis

With this type of necrosis, the cells retain their shape for several days. Cells devoid of a nucleus appear as a mass of coagulated, homogeneous, pink cytoplasm.

The mechanism of coagulation necrosis is not clear enough. Coagulation of cytoplasmic proteins makes them resistant to the action of lysosomal enzymes and, in this connection, slows down their dissolution.

Coagulation necrosis usually occurs in organs rich in proteins and poor in fluids, for example, in the kidneys, myocardium, adrenal glands, spleen, mainly as a result of insufficient blood circulation and anoxia, the action of physical, chemical and other factors, for example, coagulation necrosis of liver cells in viral damage or under the action of toxic agents of bacterial and non-bacterial origin. Coagulation necrosis is also called dry, because it is characterized by the fact that

with it, the dead areas that arise are dry, dense, crumbling, white or yellow in color.

Coagulation necrosis includes:

A. Heart attack- a type of vascular (ischemic) necrosis of internal organs (except the brain). This is the most common type of necrosis.

B. Caseous (cheese)necrosis develops in tuberculosis, syphilis, leprosy, as well as in lymphogranulomatosis. It is also called specific, because it is most often found in specific infectious granulomas. In the internal organs, a dry, limited area of whitish-yellowish tissue appears, which crumbles easily. In syphilitic granulomas, very often such areas do not crumble, but are pasty, reminiscent of Arabic glue. This is a mixed (that is, extra- and intracellular) type of necrosis, in which both parenchyma and stroma (both cells and fibers) die at the same time. Microscopically, this area of the tissue is structureless, homogeneous, stained pink with hematoxylin and eosin, chromatin lumps of nuclei (karyorrhexis) are clearly visible.

B. Wax-like, or Zenker's necrosis(Necrosis of muscles, more often of the front abdominal wall and thigh, with severe infections - typhoid and typhus, cholera)

G. Fibrinoid necrosis -type of necrosis of connective tissue, which was previously considered in the lecture "Stromal-vascular dystrophies" as a result of fibrinoid swelling. Fibrinoid necrosis is observed in allergic autoimmune diseases (for example, rheumatism, rheumatoid arthritis and systemic lupus erythematosus). Collagen fibers and smooth muscles are severely damaged Average rating of blood vessel lining. Fibrinoid necrosis of arterioles is observed in malignant hypertension. This necrosis is characterized by the loss of the normal structure of collagen fibers and the accumulation of a homogeneous, bright pink necrotic material that is microscopically similar to fibrin. Please note that the concept of "fibrinoid" differs from the concept of "fibrinous", so the latter means the accumulation of fibrin, for example, during blood coagulation or during inflammation.

D. Fat necrosis:

1. Enzymatic fat necrosis: fat necrosis most often occurs in acute pancreatitis and damage to the pancreas, when pancreatic enzymes leave the ducts in the surrounding tissues. Pancreatic lipase acts on triglycerides in fat cells, splitting them into glycerol and fatty acids, which, interacting with plasma calcium ions, form calcium soaps. At the same time, opaque, white (like chalk) plaques and nodules (steatonecrosis) appear in the adipose tissue surrounded by the pancreas.

With pancreatitis, it is possible for lipase to enter the bloodstream with subsequent wide distribution, which is the cause of fat necrosis in many parts of the body. Subcutaneous adipose tissue and bone marrow are most often damaged.

2. Non-enzymatic fat necrosis:non-enzymatic fat necrosis is observed in the mammary gland, subcutaneous fat tissue and in the abdominal cavity. Most patients have a history of trauma. Non-enzymatic fat necrosis is also called traumatic fat necrosis, even if trauma is not identified as the underlying cause.

Non-enzymatic fat necrosis causes an inflammatory response, which is characterized by the presence of numerous macrophages with foamy cytoplasm, neutrophils and lymphocytes. Then comes fibrosis, and this process can be difficult to distinguish from a tumor.

E. Gangrene(From the Greek. Gangraina - fire): this is necrosis of tissues that are connected to the external environment and change under its influence. The term "gangrene" is widely used to denote a clinical and morphological condition in which tissue necrosis is often complicated by a secondary bacterial infection of various degrees of severity or, being in contact with the external environment, undergoes secondary changes. Dry, wet, gas gangrene and bedsores are distinguished.

1. Dry gangrene- this is necrosis of tissues that communicate with the external environment, which occurs without the participation of microorganisms. Dry gangrene most often occurs on the limbs as a result of ischemic coagulation necrosis of tissues. Necrotized tissues appear black, dry, they are clearly separated from the adjacent functional tissue. Demarcation inflammation occurs at the border with healthy tissues. The color change is due to the transformation of hemoglobinogenic pigments in the presence of hydrogen sulfide into iron sulfide. Examples can be dry gangrene:

extremities with atherosclerosis and thrombosis of its arteries (atherosclerotic gangrene), obliterating endarteritis; with frostbite or burns; fingers with Raynaud's disease or vibration disease; skin with typhus and other infections.

2. Wet gangrene:develops as a result of layering on necrotic tissue changes of a severe bacterial infection. Under the action of enzymes of microorganisms, secondary colicivation occurs. Cell lysis by enzymes that are not produced in the cell itself, but penetrate from the outside, is called heterolysis. The type of microorganisms depends on the localization of gangrene. Moisture gangrene usually develops in tissues rich in moisture. It can be found on the limbs, but more often in the internal organs, for example, in the intestines with obstruction of the mesenteric arteries (thrombosis, embolism), in the lungs as a complication of pneumonia (influenza, measles). Children weakened by an infectious disease (more often measles) may develop wet gangrene of the soft tissues of the cheeks and perineum, which is called a noma (from the Greek nome - water cancer). Acute inflammation and accumulation of bacteria is the reason why that the necrotic area becomes swollen and red-black, with widespread dissolution of dead tissue. With wet gangrene, necrotizing inflammation may occur, which is clearly demarcated from adjacent healthy tissue and, thus, difficult to treat surgically. As a result of the vital activity of bacteria, a specific smell arises. Very high mortality rate.

3. Gas gangrene:gas gangrene occurs when the wound is infected with anaerobic flora, for example, Clostridium perfringens and other microorganisms of this group. It is characterized by widespread tissue necrosis and the formation of gases as a result of the enzymatic activity of the bacterium. The main manifestations are

similar to wet gangrene, but with the additional presence of gas in the tissues. Crepitation (cracking phenomenon during palpation) is a frequent clinical symptom of gas gangrene. The fatality rate is also very high.

4. Bedsores(Decubitus): bedsores are distinguished as a type of gangrene - necrosis of the surface parts of the body (skin, soft tissues) that are subject to compression between the bed and the bone. Therefore, bedsores appear more often in the area of the sacrum, spinous processes of the vertebrae, and the greater trochanter of the femur. According to its genesis, this is trophoneurotic necrosis, because the vessels and nerves are compressed, it increases tissue trophic disorders in seriously ill patients suffering from cardiovascular, oncological, infectious or nervous diseases.

Colicative (moist) necrosis

Colicative (wet) necrosis: characterized by the melting of dead tissue. It develops in tissues relatively poor in proteins and rich in liquid, where there are favorable conditions for hydrolytic processes. Cell lysis occurs as a result of the action of own enzymes (autolysis). A typical example of moist colliquative necrosis is an area of gray softening (ischemic infarction) of the brain.

Brain infarction is often called softening, because the main macroscopic sign is a decrease in the elasticity of the brain tissue in the affected area in all terms. During the first day, it is a vaguely limited area of a bluish shade, soft to the touch. By the end of the first day, the area becomes clearer and paler. In the following days, the brain matter in this area becomes even more sluggish, yellowish in color, sometimes even with a greenish tint. In the first weeks, the volume of the brain increases slightly due to its swelling. After 1-1.5 months. at the site of the infarction, a clearly defined cavity is formed, which contains a cloudy liquid and detritus. Determining the exact timing of a heart attack is too difficult not only by its appearance, but also by histological picture.

Microscopically, the brain tissue is homogeneous, structureless, slightly pink in color when stained with hematoxylin and eosin. Resorption of dead tissues is carried out by macrophages, which have the appearance of fat-granular balls. The result of necrosis. Necrosis is an irreversible process. With a relatively favorable outcome, reactive inflammation occurs around the dead tissue, which separates the dead tissue. Such inflammation is called demarcation, and the zone of separation is called the demarcation zone. In this zone, blood vessels expand, hemoptysis, edema occurs, a large number of leukocytes appear, which release hydrolytic enzymes and melt necrotic masses. Necrotic masses are absorbed by macrophages. Following this, the cells of the connective tissue multiply, which replaces or overgrows the area of necrosis. When dead masses are replaced by connective tissue, their organization is discussed. In such cases, a scar forms at the site of necrosis (a scar at the site of a heart attack). Overgrowth of the area of necrosis with connective tissue leads to its encapsulation. Calcium salts can be deposited in dead masses with dry necrosis and in the area of death that has fallen under the organization. In this case, calcification (petrification) of the necrosis

center develops. In some cases, bone formation - ossification - is noted in the area of death. During the resorption of tissue detritus and the formation of a capsule, which occurs with wet necrosis and most often in the brain, a cavity - a cyst - appears at the site of death. Calcium salts can be deposited in dead masses with dry necrosis and in the area of death that has fallen under the organization. In this case, calcification (petrification) of the necrosis center develops. In some cases, bone formation - ossification - is noted in the area of death. During the resorption of tissue detritus and the formation of a capsule, which occurs with wet necrosis and most often in the brain, a cavity - a cyst - appears at the site of death. Calcium salts can be deposited in dead masses with dry necrosis and in the area of death that has fallen under the organization. In this case, calcification (petrification) of the necrosis center develops. In some cases, bone formation - ossification - is noted in the area of death. During the resorption of tissue detritus and the formation of a capsule, which occurs with wet necrosis and most often in the brain, a cavity - a cyst - appears at the site of death.

Adverse outcome of necrosis- purulent (septic) melting of the site of death. Sequestration is the formation of an area of dead tissue that does not undergo autolysis and is not replaced by connective tissue and is freely located among living tissues. Sequestrations usually occur in bones with inflammation of the bone marrow - osteomyelitis. A sequestral capsule and a cavity filled with pus form around such a sequestration. Sequestration often leaves the cavity through fistulas, which close only after its complete release. A type of sequestration - mutilation - rejection of the ends of the fingers.

Apoptosis,or programmed cell death - a process by which internal or external factors, activating the genetic program, lead to the death of the cell and its effective removal from the tissue. Apoptosis is a mechanism of cell death that has a number of biochemical and morphological differences from necrosis.

Apoptosis is a biochemically specific type of cell death characterized by the activation of non-lysosomal endogenous endonucleases that cleave nuclear DNA into small fragments. Morphologically, apoptosis is manifested by the death of single, randomly located cells, accompanied by the formation of round bodies surrounded by a membrane ("apoptotic bodies"), which are immediately phagocytosed by the surrounding cells.

Signs of general death

Signs of general death are: corpse cooling, cadaveric suffocation, cadaveric desiccation, redistribution of blood, corpse stains, decay of corpse tissues. The cooling of the corpse ("algor mortis") occurs as a result of the cessation of metabolic processes and the gradual equalization of the temperature of the body and the environment. Morbidity ("rigor mortis") is characterized by a sharp tightening of somatic muscles due to the disappearance of ATP acid from them after death and the accumulation of lactic acid in them (after 2 - 5:00 after ascertaining death). Dehydration of the body occurs as a result of evaporation of moisture from the surface: this applies to the skin, eyeballs, mucous membranes.

The redistribution of blood is characterized by its accumulation in the veins, while the formations of the arteries remain empty. Postmortem blood clotting is possible in the veins. Corpse spots arise in connection with the redistribution of blood and are presented in the form of corpse hypostases (appear after 3 - 6:00) (appears much later as a result of hemolysis of erythrocytes). Corpse decay is due to the processes of autolysis and the life of the body in connection with the reproduction of putrefactive microorganisms in the intestines.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Definition of necrosis.
2. Causes of necrosis. Mechanisms of irreversible cell damage.
3. Classification of necrosis according to various criteria.
4. The concept of coagulation necrosis, collicative necrosis, caseous necrosis.
5. Heart attack as a type of necrosis: definitions, types, their morphological characteristics, complications and consequences.
6. Gangrene as a type of necrosis: definition, types, their morphology, characteristics, complications, consequences.
7. Bedsores and noma as separate types of gangrene: causes of development, morphological characteristics, results.
8. Sequestration as a type of necrosis, morphological characteristics, consequences.
9. Fibrinoid, fatty and waxy (Zenker) necrosis, morphological characteristics.
10. Stages of necrosis. Morphological characteristics.
11. Consequences and complications of necrosis.
12. Comparative characteristics of necrosis and apoptosis
13. Death, definition of death, types of death.
14. Postmortem changes in the body.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 Test tasks

1. In a child after measles, examination of the soft tissues of the cheeks and perineum revealed vaguely demarcated, swollen, red-black areas that fluctuate slightly. What complication did the child develop?

AND Wet gangrene
B Dry gangrene
Gas gangrene
D Bedsores
Atrophic ulcer

2. In a 6-year-old child hospitalized for measles pneumonia, the dentist found a dirty-gray area measuring 2x2.5 cm without clear boundaries on the mucous membrane of the cheek. Soft tissues are swollen, dim, with an unpleasant smell. What is the most likely diagnosis established by the dentist?

AND Noma

BGangrenous stomatitis
Pustular stomatitis
Phlegmonous stomatitis
Ulcerative stomatitis
3. A 70-year-old patient was operated on for "acute abdomen". During the operation, about 80 cm of the ileum was found to be black, the peritoneum was dull, the lumen of the superior mesenteric artery was blocked by a thrombus. What process developed in the intestine?:
AND Gangrene
B White heart attack
Coagulation necrosis
DWhite heart attack with a hemorrhagic crown
Bedsores
4. A patient with diabetes developed a sharp pain in the right foot. On examination, the big toe is black, the tissues of the foot are swollen, the epidermis is peeling off, discharge with an unpleasant smell. What clinical and morphological form of necrosis developed in the patient?:
AND Gangrenous moisture
B Dry gangrene
Heart attack
D Bedsores
ESequestration
5. At the autopsy of a 57-year-old man who died of typhus, it was found that the muscles of the front abdominal wall and thighs are dense, whitish-yellow in color, resembling a stearin candle. The described changes in the muscles are a manifestation of which pathological process:
AND Waxy necrosis
B Fibrinoid necrosis
C Caseous necrosis
D Colic necrosis
EApoptosis
6. During the autopsy of the deceased from pulmonary edema, a large yellow-gray focus was found in the myocardium, and a fresh thrombus was found in the coronary artery. Specify the diagnosis:
AND Myocardial infarction
BCardiosclerosis
Myocarditis
Amyloidosis
Cardiomyopathy
7. In an immobile patient with insufficient blood circulation, after a stroke, the skin and soft tissues above the sacrum were reddened and swollen, after the rejection of the epidermis, ulcers opened in the tissues. What process developed in the patient?:
AND Bedsores

Abscess
Dry gangrene
Phlegmon
Heart attack
8. During the microscopic examination of the liver tissues, it was found that some cells broke up into small fragments with separate organelles and remnants of the nucleus, surrounded by a membrane. There is no inflammatory reaction. Select the pathological process for which the described changes are characteristic?:
AND Apoptosis
B Necrosis
CKaryorhexis
DPlasmolysis
EPlasmorexis
9. In a patient who suffered from a long-term demarcated lameness, the tissues of the toes are dry, black in color, reminiscent of a mummy. A two-color line is located at a short distance from the blackened area (the red color is adjacent to practically unchanged tissues, and the white-yellow color is to the changed tissues). What type of necrosis does this patient have?:
AND Gangrene
Maceration
Heart attack
Sequestration
Bedsores
10. A 77-year-old patient has a pinched inguinal hernia. During laparotomy: the wall of the intestine is cyanotic, swollen, swollen, covered with fibrin threads, peristalsis is not detected. Did the pathological process develop in the intestinal wall due to restriction of the hernia?:
AND Wet gangrene
BCollicive necrosis
Bedsores
D Dry gangrene
E Coagulation necrosis

3.2. Algorithm of description of macropreparation and micropreparation

Micropreparations: 1. Caseous necrosis of the lymph node (G+E, m.zb) Lymphoid tissue is visible under the capsule in the preparation of the lymph node, part of which and the entire brain substance is replaced by a necrotic mass stained with pink eosin. Mark: 1-caseous necrosis, 2-preserved lymphoid tissue, 3-node capsule.

2. Necrotic nephrosis (G+E, m.zb.) The drug contains cortical and medullary substance of the kidneys. Pay attention to the changes in the convoluted tubules: the absence of nuclei in the cells of the epithelium due to karyolysis, the swelling of the cells and the disappearance of the borders between them, granular detritus in the lumen

of the tubules. The preserved intermediate connective tissue is swollen. The glomeruli and straight tubules remain constant, preserving the traditional color of the nuclei. Mark: 1-necrosis of the epithelium of convoluted tubules, 2-straight tubules and glomeruli are not changed.

Macro drug. Represented by a foot. 1-5 fingers and 6 cm. metatarsals are black, dry, cracked, with desquamation of the epidermis. Affected areas are separated from healthy parts by an uneven clear demarcation line. Conclusion. Dry gangrene of the foot.

3. 3 Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

1. Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

2. Assessment of practical skills on the topic of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:
At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.
The topic of the next lesson is emphasized: "Final lesson. (Subsection Disorders of blood and lymph circulation. Inflammation). Practical experience".

5. List of recommended literature(main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 5

Topic: Final lesson. (Division of Dystrophies and Necrosis). Practical experience.

Goal: Conduct a summary: determine the ability of students to independently diagnose pathomorphological pathological processes studied in practical classes No. 1-3, the ability to understand and apply the basic concepts of these topics, identify the level of theoretical and practical training of students.

Basic concepts: Provided in the materials of the relevant topics.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Individual survey of knowledge of terminology on topics: emphasize the definition or provide an explanation. Provided in the materials of previous relevant topics.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1. Diagnosis of 1 macropreparation on relevant topics,

3.2. Diagnostics of 1 micropreparation on relevant topics.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

5. **Evaluation of theoretical knowledge on the subject of the lesson:**
   - methods: survey, solving a situational clinical problem, tests
   - the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.
6. **Assessment of practical skills on the subject of the lesson:**
   - methods: assessment of the correctness of the performance of practical skills
   - the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

   The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.

"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Acute systemic circulatory disorders (acute coronary insufficiency, shock) and systemic circulatory disorders in chronic heart failure and their consequences. Regional blood circulation disorders (hyperemia, ischemia, plasmarrhagia, bleeding and hemorrhage). Violation of the formation and circulation of lymph".

Suggested topics for essays.

1. Thromboembolism. Definition, types, pathomorphology, clinical significance

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V.

Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 6

Topic: Acute systemic circulatory disorders (acute coronary insufficiency, shock) and systemic circulatory disorders in chronic heart failure and their consequences. Regional blood circulation disorders (hyperemia, ischemia, plasmarrhagia, bleeding and hemorrhage).

Goal: Familiarize yourself with blood circulation disorders, types of hematuria and hyperemia, causes of ischemia, types and causes of bleeding, familiarize yourself with such concepts as: plasmorrhagia, stasis, shock (shock organs).

Basic concepts: Hyperemia: physiological, pathological, arterial, venous, edema, anasarca, bluish induration of the spleen, cyanotic induration of the kidney, brown induration of the lungs, nutmeg liver; bleeding and hemorrhages: epistaxis, hemoptysis, vomiting blood, cyclic and acyclic uterine bleeding, blood in stool, urine, hematomas, hemorrhagic inclusions, ecchymoses, purpura, pithecia, hemopericardium, hemoperitoneum, hemothorax, hydrothorax, pyothorax, hemarthrosis;

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:

*Highlight the definition or provide an explanation.*Hyperemia: physiological, pathological, arterial, venous; edema, anasarca, bluish induration of the spleen, cyanotic induration of the kidney, brown induration of the lungs, nutmeg liver; bleeding and hemorrhages: epistaxis, hemoptysis, vomiting blood, cyclic and acyclic uterine bleeding, blood in stool, urine, hematomas, hemorrhagic inclusions, ecchymoses, purpura, pithecia, hemopericardium, hemoperitoneum, hemothorax, hydrothorax, pyothorax, hemarthrosis.

2.2. Block diagram on the topic as a list of didactic units of the topic;

Disorders of blood circulation can be divided into 3 groups: 1) disorders of blood filling, determined by complete blood (arterial or venous) and anemia; 2) violation of the permeability of the vessel wall, which should include bleeding (hemorrhage) and plasmarrhagia; 3) violation of blood flow and state (ie, rheology) of blood in the form of stasis, sludge phenomenon, thrombosis, and embolism.

Thoroughbred(hyperemia) can bearterial and venous.

Arterial full blood - increased blood filling of an organ, tissue due to increased arterial blood flow. It can be general,which is observed with an increase in the volume of circulating blood (plethora) or the number of erythrocytes (erythremia). In such cases, there is a red color of the skin and mucous membrane and an increase in blood pressure. More often, arterial hyperemia is local in nature and occurs for various reasons.

Physiological arterial hyperemia that occurs under the influence of adequate doses of physical and chemical factors, feelings of anger, shyness (reflex hyperemia), when the function of organs increases (working hyperemia), andpathological arterial hyperemia.

Based on the features of the etiology and mechanism of development, the following types of pathological arterial hyperemia are distinguished: angioneurotic (neuroparalytic); collateral; hyperemia after anemia (postanemic); vacant; inflammatory; hyperemia on the basis of arteriovenous fistula.

*Angioneurotic (neuropathic) hyperemia*observed as a result of irritation of vasodilator nerves or paralysis of vasoconstrictorsnerves

*Collateral hyperemia*arises in connection with the obstruction of blood flow inthe main arterial trunk closed by a thrombus or embolus. In such cases, blood is directed through collateral vessels.

Hyperemia after anemia(postaemic) develops in those cases when the factor that caused compression of the artery (tumor, accumulation of fluid in the cavities, ligature, etc.) and ischemia of the tissues is quickly eliminated.

Vacuated hyperemia(from Latin Vacuus - empty) develops in connection with a decrease in barometric pressure. It can be common, for example, in divers and caisson workers when quickly rising from a place of increased pressure. The hyperemia that occurs at the same time is associated with gas embolism, thrombosis of blood vessels, and hemorrhages.

Local vacant hyperemia appears on the skin under the influence of, for example, medical cans, which form a rarefied space (vacuum) over certain areas.

*Inflammatory hyperemia*is a constant companion of inflammation.

*Hyperemia on the basis of arteriovenous fistula*occurs in those cases when, for example, with a gunshot wound or other injury, there is a connection between an artery and a vein, then the arterial blood is directed into the vein.

Venous full blood - increased blood filling of an organ or tissue in connection with a violation (reduction) of blood outflow; blood flow at the same time is unchanged or reduced. Congestion of venous blood (congestive hyperemia) leads to dilation of veins and capillaries, slowing of blood flow in them, which is associated with the development of hypoxia and increased permeability of the basal membranes of capillaries.

Venous congestion can be general or local.

*General venous congestion*develops in cardiovascular diseasessystems that cause acute or chronic heart (cardiovascular) failure; can be both acute and chronic.

In acute general venous congestion, which is a manifestation of the syndrome of acute heart failure (insufficiency of myocardial contractility during myocardial infarction, acute myocarditis), as a result of hypoxic damage to histohematal barriers and a sharp increase in capillary permeability in tissues, plasma seepage (plasmorrhagia) and edema are observed , stasis in capillaries and multiple hemorrhages of a diapedic nature; dystrophic and necrotic changes develop in parenchymal organs. The structural and functional features of the organ in which acute venous stasis occurs determine the preference for edematous-plasmorrhagic, hemorrhagic or dystrophic and necrotic changes, their combination is possible.G

*Chronic general venous congestion*is a manifestation of the syndrome of chronic heart (cardiovascular) failure, which complicates many chronic heart diseases (heart defects, ischemic heart disease, chronic myocarditis, myocardiopathy, endocardial fibroelastosis, etc.). It often causes severe, irreversible changes in organs and tissues. By maintaining a state of tissue hypoxia for a long time, it determines the development of not only plasmarrhagia, edema, stasis and hemorrhages, dystrophy and necrosis, but also atrophic and sclerotic changes. Sclerotic changes, that is, the development of connective tissue, are associated with the fact that chronic hypoxia stimulates the synthesis of collagen by fibroblasts and fibroblast-like cells. Connective tissue displaces parenchymal elements, stagnant compaction (induration) of organs and tissues develops. Aqueous cycle in chronic venous congestion

With chronic venous stasis, the liver is enlarged, dense, the edges are rounded,the dissection surface is gray-yellow with a dark red speck, similar to a nutmeg, so such a liver is called "nutmeg".

Two types of changes occur in the lungs with chronic venous congestion - multiple hemorrhages, which cause hemosiderosis of the lungs, and the development of connective tissue, that is, sclerosis. The lungs become large, brown and dense - brown induration (induration) of the lungs.

Kidneys with chronic venous stasis become enlarged, dense andcyanotic - cyanotic induration of the kidneys; especially full-blooded veins of the brain substance and border zone.

Chronic venous congestion in the spleen also leads to its cyanosisindurationIt is enlarged, dense, dark cherry in color; follicle atrophy and pulp sclerosis are determined. With general chronic venous stasis, cyanotic induration is characteristic of other organs as well.

Local venous congestion(hyperemia) is observed when the outflow of venous blood from one or another organ or part of the body is obstructed due to the closure of the lumen of the vein (thrombus or embolus) or its compression from the outside (tumor developed by connective tissue). Thus, acute venous hyperemia of the gastrointestinal tract develops with thrombosis of the portal vein. Nutmeg liver and nutmeg cirrhosis are found not only with general venous congestion, but also with inflammation of the hepatic veins and their thrombosis (obliterating thrombophlebitis of the hepatic veins), which is characteristic of the disease (syndrome) of Bad - Chiari. Thrombosis of the renal veins can be the cause of cyanotic induration of the kidneys. Vein thrombosis also leads to venous stasis and swelling of the limb, if the collateral blood circulation is insufficient.

Anemia

Anemia(ischemia) is a decrease in blood supply to a tissue, organ, or part of the body as a result of reduced blood flow. We are talking about both insufficient blood supply and complete cessation of blood flow.

Tissue changes that occur with anemia are associated with hypoxia or anoxia (oxygen starvation). Depending on the cause that leads to ischemia, the time of its occurrence, the duration of hypoxia, the degree of tissue sensitivity to it, with ischemia, there are either subtle changes at the level of ultrastructures, or gross destructive changes that can even lead to ischemic necrosis - a heart attack .

With acute anemia, dystrophic and necrobiotic changes occur. Their precursors are histochemical and ultrastructural changes: disappearance from the tissueglycogen, reduction of activity of redox enzymes and destruction of mitochondria. Based on the data of electron-histochemical study of tissue changes in acute ischemia and infarction, acute ischemia should be considered as a pre-necrotic (pre-infarction) condition. With long-term anemia, atrophy of parenchymal elements and sclerosis develops as a result of increased collagen-synthesizing activity of fibroblasts.

*Angiospastic anemia*occurs as a result of spasm of the artery in connection with the influence of various irritants. Yes, a painful stimulus causes spasm of arteries and anemia in some parts of the body. The same mechanism of action of vasoconstrictor drugs (adrenaline). Angiospastic ischemia also occurs with negative emotional affects ("angiospasm of unreacted emotions").

*Obstructive anemia*develops as a result of thrombosis or embolism, withgrowth of connective tissue in the lumen of the artery, inflammation of its wall (obliterating endarteritis), narrowing of the lumen of the artery by atherosclerotic plaque. Obstructive ischemia due to thrombosis of an artery often completes angiospasm, and,

conversely, angiospasm complements the obturation of an artery with a thrombus or embolus.

*Compression anemia*appears when an artery is compressed by a tumor, tourniquet, or ligature.

*Ischemia due to redistribution of blood*observed in cases of hyperemia after anemia. Such is, for example, ischemia of the brain during the release of fluid from the abdominal cavity, where a significant amount of blood flows. The meaning and consequences of anemia are different; depend on the specifics of the cause and the duration of its influence. Thus, anemia due to spasm of arteries is short-lived, and it does not cause any special disorders.

Bleeding (hemorrhage)is the exit of blood from the lumen of a blood vessel orheart cavity, into the environment (external bleeding) or into body cavities (internal bleeding). Examples of external bleeding can be hemoptysis (haemoptoa), nosebleeds (epistaxis), vomiting blood (haemotenesis), the appearance of blood in the stool (melaena), bleeding from the uterus (metrorrhagia). With internal bleeding, blood can accumulate in the pericardial cavity (hemopericardium), pleura (hemothorax), abdominal cavity (hemoperitoneum). If blood accumulates in the tissues during bleeding, it is called hemorrhage. It follows that hemorrhage is one of the types of bleeding. Accumulation of coagulated blood in a tissue with a violation of its integrity is called a hematoma, and when tissue elements are stored, it is called a hemorrhagic impregnation (hemorrhagic infiltration).

Flat hemorrhages in the skin and mucous membranes are called bruises, and small point-like hemorrhages are called petechiae or ecchymoses.

The consequences of bleeding (hemorrhages) can be various: resorption of blood, formation of cysts at the site of hemorrhages (brain), encapsulation or germination of the hematoma by connective tissue, attachment of infection and suppuration.

Plasmarrhagia is the exit of plasma outside the blood vessel. The consequence of plasmarrhagia is the impregnation of blood plasma of the vessel wall and the tissues surrounding it - plasma impregnation; this is one of the manifestations of impaired vascular permeability, which normally provides transcapillary exchange.

Stasis–itstoppage of blood flow in vesselsmicrocirculatory channel (mainly in capillaries). Stoppage of blood flow begins slowly, which is defined as a prestatic state, or prestasis.

The main properties of the sludge phenomenon are the sticking together of erythrocytes, leukocytes or platelets; at the same time, plasma viscosity increases, which causes difficulty in blood perfusion through the vessels of the microcirculatory channel. Sludge phenomenon (syndrome) is one of the varieties of stasis.

Shock is an acute pathological process caused by actionan overpowering stimulus characterized by disruption of the activity of the central nervous system, metabolism and autoregulation of the microcirculatory system, which leads to destructive changes in organs and tissues.

The basis of the shock of various origins is a single complex multiphase mechanism of development. The early period of shock is characterized by relatively specific signs, which are due to the peculiarities of etiology and pathogenesis.

Depending on the cause, the following types of shock are distinguished: 1) hypovolemic, which occurs with a sharp decrease in the volume of circulating blood (or liquid); 2) traumatic, the trigger of which is excessive afferent (mainly painful) impulse; 3) cardiogenic, which arises as a result of a rapid decline in the contractile function of the myocardium and an increase in the flow of afferent (mainly "hypoxic") impulses; 4) septic (toxic-infectious), the cause of which is endogenous intoxication by pathogenic microflora.

In the late period of shock, the relative specificity of signs due to the peculiarities of its etiology and pathogenesis disappears, and the clinical and anatomical manifestations become stereotyped.

Morphological changes of shock are characterized by hemocoagulation disorders in the form of DVZ-syndrome, hemorrhagic diathesis, liquid cadaveric blood, which can be the basis of the diagnosis of shock at the autopsy of the deceased. During a microscopic examination of hemodynamic and rheological properties of blood, widespread spasm of blood vessels, microthrombi in the microcirculation system, signs of increased permeability of capillaries, and hemorrhages are found.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Classification of circulatory disorders.
2. Types of blood circulation disorders associated with damage to the vascular wall: bleeding, hemorrhage, plasmarrhagia, edema. General definition and characteristics, causes of development, clinical significance.
3. Bleeding. Definition, classification, morphological characteristics of certain species, complications and consequences.
4. Bleeding. Definition, classification, morphological characteristics of certain species, complications and consequences.
5. Plasmarrhagia. Definition, morphological characteristics of consequences.
6. Swelling. Definition, morphological characteristics of consequences.
7. Types of circulatory disorders associated with circulatory disorders: arterial congestion, venous congestion, ischemia, shock. General definition and characteristics, causes of development, clinical significance.
8. Arterial hyperemia. Definition, classification, morphological characteristics of individual species, complications and consequences.
9. Venous hyperemia. Definition, classification, morphological characteristics certain types, complications and consequences.
10. Ischemia. Definition, classification, morphological characteristics of certain species, complications and consequences.
11. Shock. Definition, classification, stages of development.
12. Concept of shock organ (shock kidney, shock lung), morphological characteristics, complications and consequences.

13. Concept of hemostasis. Types of circulatory disorders associated with blood flow disorders: stasis, thrombosis, embolism, disseminated intravascular coagulation. General definition and characteristics, causes of development.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 Test tasks

1. At the autopsy of the deceased, who was ill, Mrwith a heart scan, an enlarged liver of a variegated appearance, with a pattern of nutmeg on the section, was found. Name the type of blood circulation disorder:
AND General venous complete blood
BHemorrhage
Bleeding
D General arterial complete blood
Anemia
2. A young man suddenly died during an emotionally stressful job. An autopsy revealed uneven blood filling of the myocardium. Histochemically: decrease in glycogen content. Electron microscopic: destruction of mitochondria, contractures of myofibrils. Specify the probable circulatory disorder?:
AND Acute ischemia
BAcute venous hyperemia
Angioneurotic arterial hyperemia
Chronic ischemia
Vacant arterial hyperemia
3. In a sick person, 25 years after the formation of a thrombus in the main arterial trunk, signs of hyperemia of synergist arteries appeared. What is hyperemia?:
AND collateral
BAngioneurotic
Hyperemia after anemia
D Vacant
EInflammatory
4. At the autopsy of a 30-year-old man with aortic valve insufficiency due to rheumatism, a wedge-shaped area of dense consistency, dark red in color, with clear borders was found in the lungs. The base of the wedge is located subpleural, the top is directed to the root of the lungs. What pathological process developed in the lungs?:
AND Hemorrhagic heart attack
BHematoma
CHemothorax
Abscess
Ischemic heart attack

5. In a deceased 30-year-old man who suffered from decompensated heart disease for a long time, the liver is enlarged, dense, the edges are rounded, the surface of the cut is variegated with dark red spots. What is the name of such a liver?:
AND Nutmeg liver
BShock liver
CMore variegated liver
DToxic liver
E Toxic liver
6. At the autopsy of a 30-year-old woman who died suddenly, blood was found in the pericardial cavity. What is this process called?:
AND Hemopericardium
B Hemorrhagic infiltration
CSynets
DHematoma
EHemothorax
7. During the autopsy of the corpse of a person who died some time after surgery on the organs of the chest, the pleural cavities contained about 1 liter of fluid (the thoracic lymphatic duct was damaged during the operation). Biochemical examination of the fluid revealed the presence of albumin, globulins, lipids, electrolytes, mesotheliocytes, blood elements were absent. What is the accumulation of fluid in the pleural cavity called?:
AND Chylothorax
BHydrothorax
CPiothorax
Empyema of the pleura
E Hemothorax
8. At the autopsy of the corpse of a 56-year-old man who died of acute heart failure, an area of necrosis of an irregular shape and a whitish color with a perifocal zone of hyperemia and hemorrhage was found in the wall of the left ventricle. Name the type of pathological process in the heart of the deceased?:
AND Ischemic heart attack with a hemorrhagic coronary artery
BHemorrhagic heart attack
Ischemic heart attack
DMyomalacia
Colicative necrosis
9. A patient with periodontitis has swelling of the gums. They have a dark red color. What local blood circulation disorder prevails in the patient's gums?
AND Venous hyperemia
Arterial hyperemia
Ischemia
Thrombosis
Embolism
10. The patient, who has been suffering from rheumatism for a long time, died of cardiopulmonary insufficiency. During the autopsy, brown induration of the lungs was revealed. What type of blood circulation disorder causes similar changes in the lungs?
AND Chronic left ventricular failure
BChronic right ventricular failure
Acute left ventricular failure
DAcute right ventricular failure
Portal hypertension

11. During the autopsy of the deceased, who suffered from hypertensive disease, a cavity was found in the substance of the brain, the walls of which have a rusty color. What preceded the occurrence of these changes?
AND Hematoma
B Diapedesic hemorrhages
Ischemic heart attack
DPlasmorrhages
Abscess

3.2. Algorithm of description of macropreparation and micropreparation

Micropreparations: 1. Nutmeg liver (G+E, m. zb.) In the drug, the central parts of the liver lobes look pink-red, and the periphery - blue. In the central parts of the lobules, there is a sharp expansion and overflow of blood in the central veins intertrabecular sinusoids; the cells of the liver parenchyma, located between the full-blooded capillaries, are dilated and crowded, and are in a state of atrophy. On the periphery of the lobule, where congestion is much less pronounced, beams of liver cells are preserved. Branches of the bile ducts, the hepatic artery of the portal vein (hepatic triad) are located in the intermediate connective tissue. Mark: 1-central vein dilated and filled with blood 2-dilated sinusoids 3-hepatic trabeculae.
2. Stasis of brain vessels (G+E, m.zb.) Stasis in brain capillaries. Capillaries of brain tissue are sharply expanded, made of closely adjacent to each other, in some places vaguely contoured erythrocytes. Label: 1-dilated capillary, 2-erythrocytes, 3-brain matter.
Macro drug. Represented by an enlarged liver

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.

"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Inflammation: causes, morphogenesis. Pathomorphology of exudative inflammation".

Suggested topics for essays.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine

2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 7

Topic: Violations of hemostasis: hemorrhagic syndrome, thrombosis, DVZ-syndrome. Embolism. Thromboembolism of the pulmonary artery, thanatogenesis.

Goal: Familiarize yourself with blood circulation disorders: embolism, thrombosis (and its types), and DVZ-syndrome.

Basic concepts:hemostasis, thrombosis, thrombus (white, red, mixed, hyaline, obturational, parietal, agonal, spherical, morantic), embolism (thromboembolism, air, gas, tissue, amniotic fluid, foreign bodies, fatty) DVZ-syndrome.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:

*Highlight the definition or provide an explanation.*Thrombosis, thrombus (white, red mixed, occlusive, parietal, moranic, axial, spherical, agonal), Embolism (thromboembolism, gas embolism, air, tissue, foreign body embolism, fat, amniotic fluid embolism)

2.2. Block diagram on the topic as a list of didactic units of the topic;

Disorders of blood circulation can be divided into 3 groups: 1) disorders of blood filling, determined by complete blood (arterial or venous) and anemia; 2) violation of the permeability of the vessel wall, which should include bleeding (hemorrhage) and plasmarrhagia; 3) violation of blood flow and state (ie, rheology) of blood in the form of stasis, sludge phenomenon, thrombosis, and embolism.

Thrombosis

Thrombosis is a lifelong coagulation of blood in the lumen of blood vessels or heart cavities. The clot formed in this case is called a thrombus.

*Stages of thrombosis:*1) formation of prothrombinase; 2) formation of thrombin; 3) formation of fibrin.

In addition to the coagulation system, there is also an anticoagulation system, which ensures a normal liquid state of the blood. Thus, thrombosis is a manifestation of dysregulation of hemostasis systems (coagulation and anticoagulation).

*Features of a thrombus:*1) the thrombus attaches to the vessel wall at the site of its damage (that is, where the process of thrombus formation began); 2) the thrombus has a corrugated surface (due to layering of platelets and fibrin); 3) the consistency of the thrombus is dense, dry, fragile.

Types of blood clots by structure and appearance: 1) White (consists of platelets, fibrin and leukocytes). It is formed more often in arteries, slowly, with fast blood flow. 2) Red (contains platelets, fibrin and erythrocytes). It forms more often in the veins, slowly, with slow blood flow. 3) Mixed (contains elements of both white and red blood clots, has a layered structure). It distinguishes the head (white thrombus structure), body (mixed thrombus) and tail (red thrombus). Such thrombi are more often found in veins, in the cavities of aneurysms of the aorta and heart. 4) Hyaline (does not contain fibrin, consists of destroyed erythrocytes, platelets and precipitated plasma proteins, resembles hyaline from the outside). It occurs more often in the vessels of the microcirculatory channel.

Thrombi can be paramural (most of the lumen of the vessel remains free) and occluding (obstructive). Mural thrombus is more common on heart valves, endocardium, auricles, large arteries with atherosclerosis and large veins with thrombophlebitis, aneurysms of the heart and blood vessels. The obturator is formed more often in veins and small arteries with the growth of a wall thrombus, less often in large arteries and the aorta.

Thrombosis is the leading triggering factor of DVZ-syndrome and thromboembolic syndrome.

Consequences of thrombosis: 1) aseptic autolysis of a thrombus (under the influence of proteolytic enzymes of leukocytes); 2) organization, canalization and vascularization of the thrombus (ingrowth of connective tissue into the thrombus with the subsequent appearance of slits and channels lined with endothelium that contain blood); 3) calcification of the thrombus (sometimes stones are formed - phlebolites); 4) detachment of a thrombus and its transformation into a thromboembolism, which is the source of thromboembolism; 5) purulent melting (when purulent bacteria hit thrombotic masses). Can be observed with sepsis; 6) strengthening of the aneurysm wall of the heart and large vessels (for example, in case of myocardial infarction); 7) obturating blood clots lead to the development of a heart attack or gangrene, portal hypertension syndrome (with blockage of the portal vein), splenomegaly (blockage of the splenic vein), etc.

Embolism- this is the circulation in the blood or lymph of particles that do not occur normally, with their subsequent clogging of blood vessels. These particles are

called emboli. Emboli are more likely to move with the blood flow: 1) from the venous system of the large circulatory circle and the right part of the heart to the vessels of the small circulatory circle (that is, emboli of the veins of the lower extremities can migrate to the pulmonary vessels); 2) from the left half of the heart, aorta and large arteries in the arteries of the heart, brain, kidneys, spleen, limbs, etc. (that is, along the course of the great circle of blood circulation); 3) from the branches of the portal system of the liver to the portal system.

Sometimes the embolus, due to its weight, moves retrogradely: it descends from the vena cava into the renal, splenic veins, etc.

In the presence of defects of the interatrial and interventricular membrane, emboli, bypassing the lungs, fall from the small circle of blood circulation into the large one (paradoxical embolism). Paradoxical embolism can also include microembolism due to arteriovenous anastomoses.

Types of emboli depending on the nature of emboli: thromboembolism occurs when a thrombus or part of it breaks off. If emboli become thrombi of the veins of a large blood circulation, thromboembolism of the pulmonary artery occurs, which leads to death (if large branches are blocked) or hemorrhagic lung infarction (if small branches are blocked). If thrombi of heart valves, aorta or large arteries become emboli, then organ infarctions develop. Fat embolism. The source of embolism is drops of fat (body fat). It develops with traumatic crushing of adipose tissue, bone marrow (fractures of tubular bones), injection of oil solutions. Death occurs when brain vessels are blocked by emboli. Air embolism. Occurs when air is injected into the bloodstream (in case of injury to the veins of the neck, rupture of the veins of the uterus after childbirth due to negative pressure in them, during open heart surgery, applying a pneumothorax, accidentally injecting air into a vein along with medications). Air bubbles cause embolism of small blood vessels and sudden death. Gas embolism. Clogging of blood vessels with gas bubbles. This embolism occurs in cases of rapid transition from high pressure to normal (caisson disease in divers, caisson workers). Gas emboli clog the capillaries of the brain and spinal cord, liver, kidneys and other organs, which is accompanied by the appearance of foci of ischemia and necrosis in them. Tissue (cellular) embolism. It develops when tissues are destroyed in connection with an injury or a pathological process, which lead to the impact of pieces of tissues (or cells) into the blood. Emboli can be tumor tissue (in case of decay or metastasis), brain tissue (in the case of a head injury), amniotic fluid in the perineum, etc. Microbial embolism. It develops in those cases when microbes circulate in the blood and obstruct the lumen of the capillaries. At the same time, metastatic abscesses develop at the site of vessel blockage with microbial emboli. Embolism by foreign objects. It is observed when fragments of shells, mines, bullets and other bodies hit the lumen of large vessels. Because foreign objects are heavy, they often travel retrograde. This embolism also includes embolism with lime and cholesterol crystals of atherosclerotic plaques, which crumble into the lumen of the vessel during their ulceration. Embolism by foreign objects. It is observed when fragments of shells, mines, bullets and other bodies hit the lumen of large vessels. Because foreign objects

are heavy, they often travel retrograde. This embolism also includes embolism with lime and cholesterol crystals of atherosclerotic plaques, which crumble into the lumen of the vessel during their ulceration. Embolism by foreign objects. It is observed when fragments of shells, mines, bullets and other bodies hit the lumen of large vessels. Because foreign objects are heavy, they often travel retrograde. This embolism also includes embolism with lime and cholesterol crystals of atherosclerotic plaques, which crumble into the lumen of the vessel during their ulceration.

Disseminated intravascular coagulation syndrome(DVZ-syndrome) is a generalized coagulation of blood in the middle of the vessels, which causes the formation of a large number of microclots and aggregates of cells that disrupt microcirculation in organs and tissues. This syndrome is often described as a catastrophe for the body.

Depending on the causes of development, the following varieties of DVZ-syndrome are distinguished:

1) infectious-septic (develops with sepsis); 2) post-traumatic (with crash syndrome, burn disease, multiple bone fractures);

3) shockogenic (with all types of shock); 4) surgical (after operations with extensive tissue trauma); 5) obstetric (with premature detachment of the placenta, inflow of amniotic fluid into the blood); 6) toxicogenic (with malignant tumor growth); 7) tumor (with immune tissue damage) and others.

The pathogenesis of DVZ-syndrome is based on the so-called "humoral protease explosion", that is, the simultaneous activation of all proteolytic enzymes of the blood plasma, which are part of four extracellular biochemical systems: a) coagulation system; b) fibrinolytic system; c) kallikrein-kinin system; d) complement systems.

2.3. List of questions to check basic knowledge on the subject of the lesson.
1. Classification of circulatory disorders.
2. Concept of hemostasis. Types of circulatory disorders associated with blood flow disorders: stasis, thrombosis, embolism, disseminated intravascular coagulation. General definition and characteristics, causes of development.
3. Thrombosis. Definition, causes of development (general and local factors), morphological characteristics.
4. Thrombus. Types of blood clots, structure of different types of blood clots. Comparative morphological characteristics of thrombus and postmortem blood clot.
5. Complications and consequences of thrombosis, clinical significance.
6. Embolism. Definition, classification, general morphological characteristics, complications and consequences.
7. Morphological characteristics of various types of embolism, conditions and causes of development, diagnostic features.
8 DVZ-syndrome. Definition, causes of development, stages of development. Morphological characteristics. Clinical significance.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues

and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 Test tasks

1. In a 21-year-old patient, in the postoperative period, on the third day after hip amputation, the bandage of the stump leaked blood, shock developed. How to explain such a complication?:
AND Autolysis of thrombus
BVascularization of the thrombus
CCalcification of a thrombus
Ossification of the thrombus
Embolism

2. A 30-year-old woman suffering from thrombophlebitis of the veins of the legs died suddenly due to pulmonary insufficiency. At autopsy of the lungs, 3 hemorrhagic infarcts were found. What is their most likely origin?:
AND Thromboembolism
BHeart failure
Blood stasis
DVenous stasis
Angiospasm

3. During the autopsy of a 40-year-old deceased man who was involved in a car accident and died on the 5th day, the pathologist found a large amount of fat in the microcirculatory channel of the lungs with special staining. What caused the death?:
AND Fat embolism
B Angiospasm
Heart failure
Traumatic shock
General venous stasis

4. Choose one correct answer. A 32-year-old woman suffering from subacute septic endocarditis suddenly lost vision in her right eye. During the examination, the ophthalmologist found a sharp expansion of the lumen of the central artery and the presence of a blood clot in it. Are the detected changes a manifestation?:
AND Phlebothrombosis
B Thromboembolism
CDvs-Syndrome
Sludge Phenomenon
Blood stasis

5. The autopsy revealed: multiple hemorrhagic lung infarcts, brownish-colored dense masses in some lung vessels that are not attached to the vessel wall, varicose veins of the lower extremities with blood clots. What pathological process is it about?
AND Thromboembolism of the pulmonary artery
B Fat embolism of the pulmonary artery
Tissue embolism of the pulmonary artery
D Congestive thrombosis of the pulmonary artery
EHemorrhagic bronchopneumonia

6. The autopsy revealed: multiple hemorrhagic lung infarcts, blood clots, dense brown masses that are not attached to the vessel wall, varicose veins are found in some lung vessels. What is the pathological process in question?:
AND Thromboembolism of pulmonary artery vessels
B Tissue embolism of pulmonary artery vessels
C Hemorrhagic bronchopneumonia

D Congestive thrombosis of pulmonary artery vessels
Fat embolism of pulmonary artery vessels
7. During the accident, the driver was injured in the neck by broken glass. Bleeding was small, but after a few minutes the victim died due to acute shortness of breath. When the deceased's heart is dissected, bubbles are released in the water-filled pericardial cavity. Specify the most likely pathological process?:
AND Air embolism
B Fat embolism
Embolism with foreign bodies
D Thromboembolism
EGas embolism
8. A man with caisson disease died with signs of acute disorders of cerebral circulation in the a. meningea media basin of the left hemisphere of the brain. An autopsy revealed a focus of gray softening of the brain measuring 6x7x3.4 div. Establish the nature of the process that caused the death of a person:
AND Gas embolism
Atherosclerosis of blood vessels
Thrombosis
Fat embolism
Thromboembolism
9. The pilot who died as a result of depressurization of the plane's cabin. A histological examination of the internal organs revealed a large number of bubbles in the vessels, fatty dystrophy in the liver. In the brain and spinal cord - multiple small ischemic foci of gray matter softening. Indicate the most likely reason for such changes:
AND Gas embolism
B Fat embolism
Tissue embolism
D Thromboembolism
Air embolism
10. Histological examination revealed the presence of a thrombus in the artery, which consisted of platelets, fibrin and leukocytes. What kind of blood clot is this?:
AND White
B Mixed
CHyaline
Red
ELayered
11. In a deceased 30-year-old man, blood clots were found in the heart on the valvular and parietal endocardium, between the trabeculae. What are these blood clots called?:
AND Wall-mounted
BWhat is being clogged
C Spherical
Progressive
EMixed

3.2. Algorithm of description of macropreparation and micropreparation

Micropreparations: 1. Fat embolism (Zab. Sudan III, m.zb.) In the preparation of the lungs, all vessels, especially capillaries in the walls of the alveoli contain orange drops of fat emboli. Mark: 1- Fat emboli, 2- alveolar walls.

2. Thrombosis of a vessel (G+E, m.zb.) The cross-section of a vessel clearly shows its wall, a thrombus, which has a typical structure and the place of its attachment to the wall. In this area, the development of granulation tissue that grows into a thrombus can be seen. Mark: 1- obturating thrombus, 2- place of thrombus attachment to the wall, 3- vessel wall.

Macro drug. Represented by the aorta

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not

	demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Inflammation: causes, morphogenesis. Pathomorphology of exudative inflammation".

Suggested topics for essays.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

10. http://moz.gov.ua- Ministry of Health of Ukraine
11. www.ama-assn.org– American Medical Association /American Medical Association
12. www.who.int- World Health Organization
13. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
14. http://bma.org.uk– British Medical Association
15. www.gmc-uk.org- General Medical Council (GMC)
16. www.bundesaerztekammer.de– German Medical Association
17. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
18. http://www.webpathology.com/- Web Pathology

Practical lesson No. 8

Topic: Inflammation: causes, morphogenesis. Pathomorphology of exudative inflammation.

Goal: Familiarize yourself with the topic of "inflammation", its types, stages, and factors that provoke inflammation.

Basic concepts: Inflammation, acute inflammation, chronic inflammation, exudate (serous exudate, hemorrhagic exudate, catarrhal exudate, purulent exudate, fibrinous exudate: diphtheritic and croupous), transudate, phlegmon, abscess, boil, carbuncle, empyema. , GRIP (true and false).

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:

*emphasize the definition or provide an explanation.*Inflammation, acute inflammation, chronic inflammation, exudate (serous exudate, hemorrhagic exudate, catarrhal exudate, purulent exudate, fibrinous exudate: diphtheritic and croupous), transudate, phlegmon, abscess, boil, carbuncle, empyema, CRUP (true and false).

2.2. block diagram on the topic as a list of didactic units of the topic;

Inflammation is a complex vascular-mesenchymal reaction to damage caused by various agents.

Inflammation is a protective and adaptive reaction aimed at limiting the area of damage; destruction (neutralization) of agents that caused inflammation; restoration of damaged tissues (repair).

Inflammation can be caused by various factors: 1) biological (exogenous and endogenous): a) microorganisms and products of their vital activity; b) immune factors: antibodies, immune complexes, sensitized lymphocytes, etc., 2) physical (radiation, electric current, high and low temperatures, trauma), 3) chemical (drugs, toxins, poisons).

Inflammation consists of three phases: alteration, exudation and proliferation.

Alterationrepresented by dystrophy and necrosis. This is the initial phase of inflammation, which leads to the release of mediators that determine the entire subsequent development of the inflammatory reaction.

Exudation- exit of the liquid part of the blood and formed elements outside the vascular bed. Initially, a reaction of the microcirculatory channel with a violation of the rheological properties of blood develops: short-term vasoconstriction; vasodilatation (arterioles, capillaries and post-capillaries) with the development of inflammatory hyperemia; slowing of blood flow and increase in blood viscosity, stasis. In the future,

the permeability of the microcirculatory channel increases: the appearance of pores between endothelial cells due to their contraction and expansion of vessels, as well as due to damage to the endothelium. The above promotes the release of fluid and plasma proteins: interendothelially through interendothelial pores; intraendothelially with increased pinocytosis in the endothelium.

Exudate and inflammatory cellular infiltrate are formed.

Exudate is an inflammatory fluid containing protein (more than 2%) and cellular elements. When cells accumulate in tissues, they speak of an inflammatory cell infiltrate.

The composition of the cells of the infiltrate is different: in the first 6-24 hours, the exudate is dominated by PAL; in the period of 24-48 hours, monocytes-macrophages begin to dominate; in inflammation associated with immediate-type hypersensitivity reactions, eosinophils predominate in the exudate.

Inflammation in the clinic is manifested by 5 classic signs: redness, swelling, pain, temperature rise and functional impairment. Redness - rubor, reflects hyperemia, expansion of all working and auxiliary blood vessels as a result of irritation of vasodilator nerves. At first, blood flow accelerates, and then slows down to peresstasis and stasis.

Proliferation- the final phase of inflammation, which is characterized by:

1. Proliferation of proliferative cells in the center of inflammation: macrophages, cambial mesenchymal cells, smooth muscle cells (SMC), epithelium.

2. Cell differentiation and transformation: a macrophage can transform into an epithelioid and giant cell; B-lymphocyte - into a plasma cell; a cambial mesenchymal cell turns into a fibroblast.

Proliferation of cells in the center of inflammation with the appearance of a large number of fibroblasts is the basis for the restoration of damaged tissues.

Exudative inflammation - xcharacterized by the predominance of exudation and the formation of exudate in tissues and body cavities.

The nature of the exudate depends on the state of vascular permeability and the depth of damage, which is determined by the type and intensity of the damaging factor.

Depending on the nature of the exudate, the following are distinguished: serous, fibrinous, purulent, purulent, hemorrhagic and mixed inflammation; a special type of inflammation can develop on the mucous membranes - catarrhal.

Serous, fibrinous and purulent are independent and main forms of inflammation. Hemorrhagic, catarrhal and ichorous (putrefactive) are not independent forms of inflammation.

*Serous*inflammation is characterized by the release of a watery exudate from the blood, with a low content of protein and cells.

Outwardly, this exudate is similar to stagnant liquid transudate, which appears, for example, in cardiac edema.

The transudate has a low specific gravity and contains no more than 1-2% protein. In the serous exudate there is more protein, up to 6-8%, the specific gravity is higher (1018-1020 p.o.), there are more cells.

Another independent type of exudative inflammation is fibrinous inflammation. Fibrinous inflammation is characterized by the release of exudate containing a large amount of coarsely dispersed proteins and fibrinogen, leukocytes and cells of necrotic tissue. Due to the content of fibrinogen and enzymes released from the necrotic tissue, hyaluronidase and thromboplastin, for example, the exudate coagulates immediately after leaving the vessels. If necrosis with fibrinous inflammation captures only the surface layers of the tissue, then the coagulated fibrin lies on the surface, it is easily removed without damaging the tissue. This subtype of fibrinous inflammation is called croupous inflammation. If the necrosis of the tissue is deep, the fibrinous exudate is released and coagulated in the depth of the tissue itself, often hyalinized with the formation of a tightly bound film. When you try to remove the films, bleeding and ulcers appear. This subtype of fibrinous inflammation is called diphtheritic inflammation.

The type of fibrinous inflammation (croupous or diphtheritic) depends not only on the depth of damage to the underlying tissue, but also on the nature of the epithelium. Where there is a multi-layered flat epithelium (oral cavity, pharynx, tonsils, epiglottis, esophagus, true vocal cords, cervix), films are firmly connected to the epithelium, although necrosis and fibrin shedding are sometimes limited to the epithelial cover. This is explained by the fact that the multi-layered flat epithelium is closely connected with the underlying connective tissue and therefore "firmly holds" the films.

Purulent inflammation -is characterized by the predominance in the exudate of PNAL (preserved and those that have disintegrated).

The most common cause is pyogenic microorganisms (staphylococci, streptococci, gonococci, meningococci, Pseudomonas aeruginosa, etc.).

A characteristic morphological feature is histolysis - tissue melting by proteolytic enzymes of leukocytes (neutral proteases - collagenase, elastase, cathepsin and acid hydrolases).

Purulent inflammation can be limited (abscess) and diffuse (phlegmon), purulent inflammation in pre-existing cavities with accumulation of pus is called empyema.

An abscess is a focal purulent inflammation characterized by the formation of a cavity filled with pus. An abscess, or abscess, develops in those cases when tissue necrosis occurs in the center of inflammation, its impregnation with leukocytes and melting as a result of the proteolytic action of enzymes released from leukocytes upon their death.

Phlegmon is diffuse (diffuse) purulent inflammation, in which purulent exudate spreads diffusely between tissue elements, permeating and delaminating tissues. It most often occurs in the subcutaneous tissue, in the area of the fascia, along the vascular and nerve trunks. Diffuse purulent inflammation can also occur in

parenchymal organs, in soft meninges. Tissues with phlegmonous inflammation swell, seep with pus.

There is a distinction between soft and hard phlegmon. Soft phlegmon is characterized by the absence of necrosis cells in the tissue, hard phlegmon - by the presence of such cells that are not subject to purulent melting, as a result of which the tissue becomes very dense; dead tissue gradually separates.

*Hemorrhagic inflammation*characterized by the presence of a large number of erythrocytes in the exudate. Vascular permeability is of great importance in its development. Occurs with severe infectious diseases: plague, anthrax, flu, in the past – with natural smallpox.

*Purulent inflammation*occurs more often in wounds with extensive crushing of tissues. It is most often associated with anaerobic infection in combination with purulent microorganisms. Large foci of necrosis are characteristic.

*Catarrhal inflammation*occurs on mucous membranes. It is characterized by a large amount of exudate that flows from the surface. Exudate always contains mucus. It can be serous, purulent and mucous. Occurs with infectious diseases of the upper respiratory tract, allergic conditions, etc.

2.3. List of questions to check basic knowledge on the subject of the lesson.
1. Definition of inflammation. Factors leading to the development of inflammation.
2. Classification of inflammation according to various criteria. Phases of inflammation.
3. Acute inflammation. General characteristics, morphological classification.
4. Serous inflammation, morphological characteristics. The concept of exudate. Comparative characteristics of exudate and transudate.
5. Purulent inflammation, classification, general morphological characteristics and characteristics of its individual types. Concept of manure, its composition.
6. Fibrinous inflammation, classification, general morphological characteristics and characteristics of its individual types. The concept of itinous and false croup.
7. Catarrhal inflammation, classification, general morphological characteristics and characteristics of its individual types.
8. Hemorrhagic inflammation, morphological characteristics.
9. Purulent inflammation, morphological characteristics.
10. Mixed inflammation, morphological characteristics.
11. Complications and consequences of inflammation

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 Test tasks

1. A 55-year-old man suffered from chronic glomerulonephritis for a long time. He died of chronic kidney failure. On the surface of the epicardium and pericardium, grayish-whitish villous layers are found. What pathological process took place in the pericardium?

AND Fibrinous inflammation
Organization
Proliferative with burning
DHemorrhagic inflammation
EArterial full blood
2. At the autopsy of a 34-year-old deceased from rheumatism, the surface of the epicardium is woolly, covered with gray films that are easily detached. After their separation, a swollen full-blooded surface is determined. What is the most likely diagnosis?
AND Fibrinous pericarditis
B Purulent pericarditis
CHemorrhagic pericarditis
Proliferative pericarditis
Catarrhal pericarditis
3. A 55-year-old man suffered from chronic glomerulonephritis. Death occurred due to the phenomena of chronic kidney failure. Macroscopically: on the surface of the epicardium and pericardium, there are grayish-whitish villous layers, after removal of which sharply expanded and filled with blood vessels are clearly visible. What process took place in the pericardium?
AND Fibrinous inflammation
Organization
Proliferative inflammation
DHemorrhagic inflammation
EArterial full blood
4. A 40-year-old patient died of cerebral edema. There is a history of facial carbuncles. At the autopsy, hemoptysis and swelling of the brain tissue were noted. In the white matter of the left hemisphere, two cavities measuring 6x5.5 and 5x4.5 cm filled with a yellowish-green creamy liquid were found. The walls of the cavities are nerve tissue with uneven edges. What complication of carbuncle developed in the patient?
AND Acute abscesses
B Chronic abscesses
Empyema
Calculating necroses
Cysts
5. A 40-year-old patient died of cerebral edema. There is a history of facial carbuncles. At the autopsy, hemoptysis and swelling of the brain tissue were noted. Two cavities measuring 6x5.5 cm and 5x4.5 cm were found in the white matter of the left hemisphere, filled with a yellowish-green, creamy liquid. The walls of the cavities are nerve tissue with uneven edges. What complication of carbuncle developed in the patient?
AND Acute abscesses
B Chronic abscesses
Empyema

Calculating necroses
Cysts
6. A large number of different blood cells were found during microscopic examination of a punctate from the center of inflammation in a patient with a skin abscess. Which of these cells are the first to arrive from blood vessels to tissues during inflammation?
AND Neutrophils
BMonocytes
Basophils
DEosinophils
Lymphocytes
7. A 6-year-old child was brought to the hospital in a state of asphyxiation. Greyish-yellow films were found in the larynx, which were easily removed. What kind of inflammation developed?
AND Fibrinous
B Catarrhal
Purulent
D Hemorrhagic
EDesquamative-necrotic
8. The patient has a high temperature, shortness of breath, pain in the right side of the chest. Pleural puncture yielded 700 ml of yellow-green viscous fluid. What pathological process developed in the pleural cavity?:
AND Pleural empyema
Bronchopneumonia
Serous pleurisy
DHemorrhagic pleurisy
E Carcinomatosis of the pleura
9. The mucous membrane of the colon of a person who died from dysentery at autopsy is full of blood, covered with a gray film that comes off with effort. What type of inflammation has developed in the intestines of the patient?:
AND Diphtheritic inflammation
BHemorrhagic inflammation
Catarrhal inflammation
D Serous inflammation
ECrupose inflammation
10. At the autopsy of a deceased patient from chronic renal failure, gray-yellow, tightly attached films were found in the mucous membrane of the colon, which separated with the formation of ulcers. What kind of inflammation is this?:
AND Diphtheritic
BSerious
Catarrhal
D It's big
Purulent

3.2. Algorithm for description of macropreparation and micropreparation

Micropreparations: 1. Fibrinous epicarditis (G.E zab.) Represented by a fragment of a heart. In the field of vision, the epicardium with signs of inflammatory hyperemia, edema and diffuse infiltration. Homogeneous or fibrillar eosinophilic masses of fibrin are layered on the surface of the epicardium. Between the elements of fibrin there are erythrocytes and leukocytes. Mark: 1.- epicardium; 2. - fibrinous exudate.

2. Phlegmon (G.E zab.) It is represented by a fragment of soft tissues (muscles and fatty tissue) between the elements of which there are numerous neutrophils. Elsewhere there are foci of necrosis, where the infiltration is densest and the tissue is disturbed - microabscesses. Mark: 1. - diffuse neutrophilic infiltration; 2.- microabscess.

Macro drug. Represented by heart. The surface of the organ is uneven due to the layering of dirty gray, sometimes brown plates, films and fibers of fibrinous exudate, which gives the heart a specific appearance. Conclusion: hairy heart is fibrinous epicarditis

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of

	participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Proliferative (productive) inflammation: with the formation of acute condylomas, around animal parasites, intermediate productive inflammation, granulomatous inflammation. Specific proliferative inflammation".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association

8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 9

Topic: Proliferative (productive) inflammation: with the formation of acute condylomas, around parasitic animals, intermediate productive inflammation, granulomatous inflammation. Specific proliferative inflammation

Goal:to study the features of the development and morphology of proliferative inflammation. Learn the peculiarities of the structure of polyps, warts and different types of granulomas.

Basic concepts:ANDinterstitial inflammation, inflammatory infiltrate, polyp, condyloma, granuloma (tuberculous granuloma, Pirogov-Langhans cells, syphilitic granuloma, leprosy granuloma, Virchow cells, sclerotic granuloma, Mikulich cells.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:

*emphasize the definition or provide an explanation.*Interstitial inflammation, inflammatory infiltrate, polyp, condyloma, granuloma (tuberculous granuloma, Pirogov-Langhans cells, syphilitic granuloma, leprosy granuloma, Virchow cells, sclerotic granuloma, Mikulich cells.

2.2. Flow chart on the topic as a list of didactic units of the topic.

— **Productive inflammation**characterized by the predominance of cell proliferation of hematogenous and histogenic origin. The causes of productive inflammation are different. It can be caused by biological (microorganisms, animal parasites), physical (radiation) and chemical (drugs) factors; arise as a result of the development of immunopathological processes (immune inflammation). Productive inflammation occurs in the case of persistence of the damaging agent in connection with an imperfect exudative reaction (often caused by defects of the CSF) or in connection with the special properties of the pathogen itself (resistance to the action of phagocytes - incomplete phagocytosis). It is accompanied by the appearance of limited or diffuse infiltrates consisting mainly of macrophages, lymphocytes, and plasma cells. Characteristic transformation of macrophages into epithelioid cells, and the latter have giant cells (foreign bodies or Pirogov-Langhans), as well as increased activity of

fibroblasts. Mediators of productive inflammation arise when monocytes-macrophages interact with lymphocytes.

— A frequent consequence of productive inflammation is sclerosis with the development of atrophy and shrinkage of organs with a violation of their structure - cirrhosis.

— Types of productive inflammation: interstitial, granulomatous and inflammation with the formation of polyps and condylomas.

— *Interstitial inflammation*occurs in the stroma of parenchymal organs - myocardium, liver, kidneys and lungs.

— Consider, for example, interstitial myocarditis, which occurs in many infectious diseases (influenza, diphtheria, typhus, etc.).

— In the stroma of the myocardium, an infiltrate is formed, consisting of macrophages, lymphocytes, plasma cells, single CSF, epithelioid cells, and fibroblasts. Dystrophic, sometimes necrobiotic changes are expressed in cardiomyocytes. Newly formed collagen fibers are visible in the areas of infiltration.

— The result is diffuse small-cell cardiosclerosis.

— *Granulomatous inflammation*characterized by the formation of granulomas - cell nodules, the basis of which are monocytic phagocytes.

— In the development of granulomatous inflammation, the stability of the causative agent (irritant) in relation to phagocytes is of crucial importance.

— Positive value of granuloma: restriction (localization) of the causative agent when it is impossible to eliminate it.

— According to the composition of cells, granulomas are divided into three types: macrophage granuloma, epithelioid cell, giant cell granuloma.

— Depending on the level of metabolism, a distinction is made between granulomas with a low level of metabolism when damaged by inert substances (inert foreign bodies) and consisting mainly of giant cells of foreign bodies and granulomas with a high level of metabolism when damaged by toxic stimuli (mycobacterium tuberculosis, leprosy) and represented by epithelioid-cell nodules .

— By etiology: infectious (associated with bacteria, viruses, rickettsia, protozoa, chlamydia, etc.); non-infectious granulomas (around foreign bodies, particles of organic and inorganic dust: silicosis, talcosis, byssinosis (from the Greek. byssos – flax)). Such granulomas can be the result of medication: granulomatous hepatitis, oleogranulomatous disease; granulomas of unknown nature–with sarcoidosis, Crohn's disease, Horton's disease, Wegener's granulomatosis, etc.

— Diseases accompanied by the development of granulomas are called granulomatous diseases.

— According to the pathogenesis: immune (which more often reflects the GST reaction based on macrophage-T-lymphocyte interaction) - most infectious granulomas or arising from the introduction of dust particles of plant or animal origin are immune; in case of infectious diseases, they reflect the relative resistance of the organism to the causative agent (non-sterile immunity); non-immune (most foreign

body granulomas): most often built from cells of foreign bodies, contain a small number of lymphocytes and plasma cells.

— By morphology: non-specific granulomas do not have specific features. An example would be inflammation around foreign bodies and animal parasites; specific granulomas have a certain structure, which often (but not always) allows establishing the etiological factor.

— They are found in the following diseases: tuberculosis; syphilis; leprosy; scleroma; actinomycosis; sap.

— **Tuberculosis**caused by mycobacterium tuberculosis, which has its own specific properties.

— A center of caseous necrosis is located in the center of the granuloma. It is a structureless mass of disintegrated tissues with the phenomena of karyorrhexis and karyopyknosis, which are quite characteristic of tuberculosis. A mass of all kinds of mesenchymal cells is adjacent to the caseous necrosis from all sides, and these chitins are located in a certain order, as if by zones. Individual very large Pirogov–Langhans giant cells lie closest to the necrosis. The shape of giant cells is round or oval, protoplasm with a large number of round nuclei located on the periphery of the cell in the form of a corolla or horseshoe. Nuclei are well colored, dark, lie under the cell membrane. The widest zone of the so-called epithelioid cells is located outside of the giant cells of Pirogov-Lankhgans. These cells on the preparations have an elongated oval shape and a light, chromatin-poor, blister-like nucleus. Since there is not enough chromatin, the nucleus is pale colored and resembles an air bubble. Epithelioid cells lie in several rows and layers and make up the majority, which is why the tubercle is called epithelioid. Round lymphoid cells are located on the very periphery of the granuloma. They are small in size, have a round core, which is quite compact, well colored. This is the 3rd zone. Finally, plasma cells are scattered around the granuloma in varying amounts, which are also round, the nucleus is located eccentrically, the lumps of chromatin in the nucleus are coarse, dense, well visible, lying in the form of spokes in a wheel. Thin reticulin fibers are located between the cells in the tubercle. In the conditions of treatment of tuberculosis with antibiotics, a granuloma can consist almost entirely of giant cells and is then called a giant cell tubercle. the nucleus is pale in color and resembles an air bubble. Epithelioid cells lie in several rows and layers and make up the majority, which is why the tubercle is called epithelioid. Round lymphoid cells are located on the very periphery of the granuloma. They are small in size, have a round core, which is quite compact, well colored. This is the 3rd zone. Finally, plasma cells are scattered around the granuloma in varying amounts, which are also round, the nucleus is located eccentrically, the lumps of chromatin in the nucleus are coarse, dense, well visible, lying in the form of spokes in a wheel. Thin reticulin fibers are located between the cells in the tubercle. In the conditions of treatment of tuberculosis with antibiotics, a granuloma can consist almost entirely of giant cells and is then called a giant cell tubercle. the nucleus is pale in color and resembles an air bubble. Epithelioid cells lie in several rows and layers and make up the majority, which is why the tubercle is called epithelioid. Round lymphoid cells are located on the very

periphery of the granuloma. They are small in size, have a round core, which is quite compact, well colored. This is the 3rd zone. Finally, plasma cells are scattered around the granuloma in varying amounts, which are also round, the nucleus is located eccentrically, the lumps of chromatin in the nucleus are coarse, dense, well visible, lying in the form of spokes in a wheel. Thin reticulin fibers are located between the cells in the tubercle. In the conditions of treatment of tuberculosis with antibiotics, a granuloma can consist almost entirely of giant cells and is then called a giant cell tubercle. layers and make up the majority, that is why the tubercle is called epithelioid. Round lymphoid cells are located on the very periphery of the granuloma. They are small in size, have a round core, which is quite compact, well colored. This is the 3rd zone. Finally, plasma cells are scattered around the granuloma in varying amounts, which are also round, the nucleus is located eccentrically, the lumps of chromatin in the nucleus are coarse, dense, well visible, lying in the form of spokes in a wheel. Thin reticulin fibers are located between the cells in the tubercle. In the conditions of treatment of tuberculosis with antibiotics, a granuloma can consist almost entirely of giant cells and is then called a giant cell tubercle. layers and make up the majority, that is why the tubercle is called epithelioid. Round lymphoid cells are located on the very periphery of the granuloma. They are small in size, have a round core, which is quite compact, well colored. This is the 3rd zone. Finally, plasma cells are scattered around the granuloma in varying amounts, which are also round, the nucleus is located eccentrically, the lumps of chromatin in the nucleus are coarse, dense, well visible, lying in the form of spokes in a wheel. Thin reticulin fibers are located between the cells in the tubercle. In the conditions of treatment of tuberculosis with antibiotics, a granuloma can consist almost entirely of giant cells and is then called a giant cell tubercle. have a round core, which is quite compact, well colored. This is the 3rd zone. Finally, plasma cells are scattered around the granuloma in varying amounts, which are also round, the nucleus is located eccentrically, the lumps of chromatin in the nucleus are coarse, dense, well visible, lying in the form of spokes in a wheel. Thin reticulin fibers are located between the cells in the tubercle. In the conditions of treatment of tuberculosis with antibiotics, a granuloma can consist almost entirely of giant cells and is then called a giant cell tubercle. have a round core, which is quite compact, well colored. This is the 3rd zone. Finally, plasma cells are scattered around the granuloma in varying amounts, which are also round, the nucleus is located eccentrically, the lumps of chromatin in the nucleus are coarse, dense, well visible, lying in the form of spokes in a wheel. Thin reticulin fibers are located between the cells in the tubercle. In the conditions of treatment of tuberculosis with antibiotics, a granuloma can consist almost entirely of giant cells and is then called a giant cell tubercle. Thin reticulin fibers are located between the cells in the tubercle. In the conditions of treatment of tuberculosis with antibiotics, a granuloma can consist almost entirely of giant cells and is then called a giant cell tubercle. Thin reticulin fibers are located between the cells in the tubercle. In the conditions of treatment of tuberculosis with antibiotics, a granuloma can consist almost entirely of giant cells and is then called a giant cell tubercle.

— **Syphilis**is caused by pale treponema. The disease is chronic, lasts for many years and is characterized by stages depending on changes in the phases of immunity and increased sensitivity to the causative agent and tissue decay products. In the course of syphilis, periods of alternating exudative, alterative and productive reactions are distinguished, just as it happens in tuberculosis. There are basically three such periods.

— The 1st period - the formation of the primary affect at the site of entry of the treponema and regional lymphadenitis. Primary affection in syphilis is called hard chancre. A hard chancre appears 2-4 weeks after infection in the form of a dense copper-red painless infiltrate (nerves die, so there is no pain). This infiltrate is a productive inflammation without any specific features.

— The II period or stage - the stage of cutaneous syphilides or the papular period occurs 6-8 weeks after infection. A copper-red rash appears on the skin or visible mucous membranes in the form of flat red spots - roseolae and dense infiltrative papules that protrude.

— Stage III – the gummy stage occurs 3–6 years after infection. This 3rd period is characterized by the focal character of productive inflammation, the formation of granulomas. A granuloma in syphilis is called a gumma.

— Gumma is built mainly of lymphoid and plasma cells, chaotically mixed with each other, there is no zonation. This is the first difference from tuberculous tubercle. Among the small cells, there are single giant multinucleated cells, similar to Pirogov–Langhans giant cells in tuberculous granuloma. However, the second distinction from the tuberculous tubercle is that the nuclei in the giant cells do not lie under the cell membrane, but are clustered in the center of the protoplasm. Epithelioid cells are also found in the gum, but in small quantities. The third difference between a gumma and a tuberculous granuloma is that there are many blood vessels in the gumma. The walls of these vessels are thickened, and the lumens are narrowed and even completely closed - the phenomenon of obliterating endarteritis. As a result of the obliteration of blood vessels, there are several foci of semi-liquid colliquative necrosis in the gum,

— In addition to gumma, in the tertiary period of syphilis, diffuse productive-necrotic processes, or so-called gummous infiltrates, occur in the aorta and other vessels, as well as in the liver.

— **Actinomycosis.**

— Currently, it has been established that the causative agent of actinomycosis is gram-positive bacteria - microaerophilic, aerobic and anaerobic actinomycetes, which are widespread in nature. In the human body, actinomycetes are also permanent residents, they contaminate the oral cavity, bronchi, gastrointestinal tract, and vagina.

— Actinomycetes, as a rule, lead a saprophytic lifestyle, but some strains can cause disease under certain conditions. It is known that actinomycetes do not penetrate through healthy skin and mucous membranes.

— Actinomycotic granuloma is composed of plasmatic, epithelioid and giant multinucleated cells. All the cells are scattered in disorder. Actinomycotic drusen are found in granuloma in 50% of cases. Drusen stain well according to Gram and Van Gieson. Russel bodies and hyaline spheres, which are hyalinized dead plasma cells, are also found. The presence of large dense sclerotic fields, among which abscesses are scattered, is characteristic. In abscesses, drusen are usually found among dead leukocytes.

— A cluster of xanthoma cells loaded with cholesterol can be found near the border of the abscesses. The word "xanthos" means "yellow"; cells are yellow from cholesterol.

— **Respiratory scleroma - x**chronic disease of the respiratory tract. It is called a Frisch-Volkovich stick. It is characterized by the growth of a peculiar, dense consistency of granulation tissue, built from plasmatic, epithelioid and lymphoid cells. Specific large Mikulich cells with vacuolated cytoplasm, light, as if reticulated. The nuclei, in the number of 1 or 2, are small, compacted, rod-shaped, located near the shell on the periphery of the cell. In the vacuoles of the cytoplasm of Mikulich cells, the causative agents of the disease are located - Frisch-Volkovich bacilli. They have a mucilaginous capsule, and therefore the cytoplasm of the cells, becoming mucilaginous, becomes light, reticulate. During the development of the process, part of the plasma cells ages and undergoes hyalinosis, turning into Russel bodies and hyaline spheres. There are quite a lot of blood capillaries in the granuloma. The growth of connective tissue narrows the lumen of the respiratory tract. This causes breathing problems and can cause death from asphyxiation.

— **Leprosy -**a chronic infectious disease that usually affects the skin and peripheral nerves.

— The disease is caused by mycobacterium Hansen. The source of infection is a sick person. There are three types of leprosy: lepromatous, tuberculoid, and intermediate.

— Most often, the skin, upper respiratory tract and peripheral nerves are affected. Specific granulomas are formed - lepromas.

— *Lepromatous form of leprosy*most often develops in the skin, is characterized by the appearance in it of nodules of various sizes and nodules (leproma) of a soft consistency, located in the surface layers of the skin.

— Histologically active lepromatous process is represented by the development of nodules. They merge with each other and consist mainly of macrophages with an admixture of lymphocytes, plasma cells, and histiocytes. Leprosy contains a huge amount of leprosy mycobacteria. According to Binford, 1 g of flowering leproma contains 5·109 mycobacteria. Such a powerful and unstoppable reproduction of the causative agent of leprosy is explained by the fact that their phagocytosis by macrophages is incomplete. Vacuoles, fatty inclusions, very characteristic of leprosy, gradually appear in them. Macrophages changed in this way are called Virchow's leprosy cells. Masses of bacteria in the macrophage stick together in the form of "balls", when the cells die, they are released from it and are located

freely in the tissue. In the future, the spheres are phagocytosed by giant cells of foreign bodies. Lepromatous infiltration in the skin is often diffuse. The tuberculoid form of leprosy is characterized by the proliferation of epithelioid cells, the formation of giant Pirogov–Langhans cells, and the accumulation of lymphocytes. Cellular infiltrates in the tubercular form of leprosy are located in the papillary layer under the epidermis itself. Leprosy mycobacteria are found in very small quantities. With the tuberculoid form of leprosy, small nerves of the skin are constantly involved in the process, which are destroyed. Nerve damage is accompanied by loss of skin sensitivity as one of the early symptoms of leprosy. Based on the nature of the tissue reaction, it can be assumed that the tuberculoid form shows a high resistance of the macroorganism to infection.

— *An intermediate form of leprosy*manifested by the appearance of a nonspecific cellular reaction in the skin around blood vessels and appendages of the skin, and sometimes small nerve trunks. With this form of mycobacterium leprosy, sometimes it is found in intact nerves. The intermediate form of leprosy is very difficult for clinical and morphological diagnosis.

— **Sap -**zoonotic infectious disease, which proceeds according to the type of septicopyemia with the formation of specific granulomas, abscesses in various tissues and organs.

— With acute phlegm, nodules appear, which consist of epithelioid cells with an admixture of neutrophilic leukocytes. These nodules very quickly undergo necrosis and purulent melting. Karyorrhexis is very characteristic; nuclei turn into small lumps that are intensively stained with hematoxylin. In addition to granuloma, abscesses can occur in organs and skin.

— Nodules are formed in case of chronic sputum. Nodules appear in various organs, including the lungs, and are very similar to tuberculous tubercles. With chronic asthma, sclerotic changes can occur in the organs, in particular in the lungs.

— *Productive inflammation with the formation of polyps and condylomas.*It is observed on the mucous membranes and in the flat epithelium bordering them. It is characterized by the simultaneous involvement of the epithelium and stroma of the mucous membrane in the process. The growth of the glandular epithelium together with the cells of the underlying connective tissue leads to the formation of polyps. Such polypous growths are observed with long-term inflammation of the mucous membrane of the nose, stomach, rectum, uterus, vagina, etc. It should be noted that polyps on some mucous membranes often have an inflammatory origin, while on others - tumor origin. In the areas of the flat epithelium, which is located near the prismatic one (anus, genitals), exudate during chronic inflammation constantly irritates them, which causes the growth of the stroma and epithelium with the formation of papillary formations - condylomas. Similar papillary growths of the epithelium are observed in syphilis,

On histological examination, classic condylomas are characterized by papillomatosis, acanthosis, elongation and expansion of papillae, parakeratosis, and the presence of koilocytes. Flat condylomas are located in a multi-layered flat epithelium with

acanthosis. There is usually a clear demarcation between the unchanged basal and parabasal layers and the more superficial layers of the epithelium, which contain koilocytes. Sometimes in the superficial parts of the damage there is a layer of different thickness of dyskeratocytes with pyknotic nuclei and other dystrophic changes. Condylomas are always removed, regardless of their type, location and size, spontaneous recovery (disappearance) never occurs, and benign condylomas can sometimes turn into carcinoma.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Definition of inflammation, classification, macro- and microscopic signs of inflammation.
2. Causes of the development of chronic inflammation. (Or why acute inflammation became chronic?)
3. Classification (types) of chronic inflammation.
4. Interstitial inflammation, morphological characteristics, consequences.
5. What is a polyp, morphological characteristics of a polyp (macro- and microscopic structure), examples?
6. What is a condyloma, morphological characteristics (macro- and microscopic structure), examples?
7. Granulomatous inflammation. morphological characteristic, consequences.
8. What is granuloma, types of granuloma, types of giant cells.
9. Structure of tuberculous granuloma.
10. Structure of syphilitic granuloma.
11. The structure of a leprosy granuloma.
12. Structure of rhinoscleral granuloma.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1. Test tasks

1. Microscopic examination of the infiltrate, which was removed from the submandibular area of the skin of a 30-year-old patient, revealed: foci of purulent melting surrounded by maturing granulations and mature connective tissue, drusen in the pus consisting of numerous short rod-like elements end to a homogeneous center. Determine what disease the patient has?

AND Actinikoz
B Tuberculosis
C Sifilis
Candida
E-

2. A 53-year-old man complains of redness and a small abscess on the skin of his right cheek. On cross-section, the focus is dense, yellow-green in color. Yellow-white grains - drusen grains - are observed in the pus. Microscopically, the abscess is surrounded by

maturing granulation tissue and mature connective tissue, where plasmatic, xanthoma, and epithelioid cells are found. Name a possible mycosis:
AND Actinomycosis
Aspergillosis
Sporotrichosis
Streptotrichosis
EBlastomycosis
3. Epithelioid cells, plasma cells, Mikulich cells, and Roussel's eosinophilic bodies were found in the biopsy of the nasal mucosa. What is the most likely diagnosis?
AND Rhinoscleroma
Syphilis
Tuberculosis
Respiratory syncytial infection
Allergic rhinitis
4. The patient has been suffering from chronic sialoadenitis for a long time. Histological examination of a biopsy of a salivary gland reveals infiltrates consisting of histiocytes, large epithelioid cells, giant cells of the type of cells of foreign bodies in the stroma. Name the type of inflammation?:
AND Intermediate
Alternative
Purulent
DFibrinous
EGranulomatous
5. Microscopic examination of the autopsy material revealed granulomas consisting of macrophages, lymphocytes, and plasma cells. Macrophage cells with large fat vacuoles and a large number of bacteria were very characteristic. Your diagnosis?:
AND Leprosy
B Tuberculosis
Syphilis
Banal inflammation
ESap
6. When examining the postoperative material sent with suspicion of a tumor, a large area of necrosis with preserved outlines from preexisting tissues, surrounded by a cellular infiltrate of lymphocytes, plasma cells, and epithelioid cells, was revealed. Among these cells are many blood vessels with proliferating endothelium. Your diagnosis?:
AND Syphilis
B Tuberculosis
Banal inflammation
Leprosy
Angioma
7. An autopsy of a deceased man of 40 years of age, who had worked in a coal mine for 20 years, revealed atrophy and sclerosis in the mucous membrane of the larynx and

trachea. The lungs are enlarged, dense, and have a large number of rounded, oval or irregularly shaped nodes. Your diagnosis?:
AND Anthracosis
B Silicosis
Tuberculosis
Sclerosis
Emphysema
8. Histological examination of the tissues of a 40-year-old deceased man revealed granulomas consisting of epithelioid cells and lymphocytes with admixtures of macrophages and plasma cells in the lungs. Giant cells with horseshoe-shaped nuclei are located between them. Your diagnosis?:
AND Tuberculosis
Banal inflammation
Syphilis
Leprosy
EScleroma
9. A 46-year-old patient complained of difficulty breathing through the nose. Mikulich cells, clusters of epithelioid cells, plasma cells, lymphocytes, and hyaline spheres were found in the biopsy of the thickened nasal mucosa. Your diagnosis?:
AND Scleroma
Allergic rhinitis
Meningococcal nasopharyngitis
Rhinovirus infection
Adenovirus rhinitis
10. In a patient who died of heart failure, the pathomorphological examination revealed: that the leaflets of the mitral valve were deformed, thickened, and fused at the edges; in the connective tissue of the myocardium - diffusely scattered nodes that are formed from foci of fibrinoid necrosis, near which accumulate macrophages that resemble giant multinucleated cells. Similar foci are surrounded by lymphocytes and individual plasma cells. Which of the listed granulomas occurs in this patient?:
AND Rheumatic
Syphilitic
CLepronosis
DTuberculosis
Actinomyotic
11. Microscopic examination of a kidney biopsy revealed foci in the center of which are granular eosinophilic masses surrounded by an infiltrate of lymphocytes, epithelioid cells, and single Pirogov-Langhans cells. Choose the pathological process that most fully corresponds to the specified changes?:
AND Granulomatous inflammation
Alterative inflammation
Proliferation and differentiation of macrophages
Coagulation necrosis

Caseous necrosis

3.2. Algorithm of description of macropreparation and micropreparation

Micropreparations: 1. Actinomycosis (Note G.+E.) Represented by liver tissue. In the center of the field of vision is a focus of pus with a large number of basophilic inclusions — drusen (clusters of colonies of microorganisms), which have an irregular shape. Mark: 1- accumulation of leukocytes, 2.- actinomycotic drusen.

2. Epithelioid cell granuloma(Note G.+E.) The material is lung tissue. In the field of vision, we see a homogeneous center of caseous necrosis of pink color, which is surrounded by a layer of epithelioid cells. Single multinucleated Pirogov-Langhans giant cells are found between them. There are many lymphocytes on the periphery of the focus. Mark: 1.- caseous necrosis; 2.- epithelioid cells; 3.- giant cells of Pirogov-Langhans.

Macro drug. Represented by a fragment of the wall of the large intestine. The intestine is open, the mucous membrane is smooth. In the center of the fragment, a soft, shiny gray-pink protrusion (like a nodule) measuring 0.5 cm on a wide base is observed on the mucous membrane. Conclusion: Colon polyp.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .

"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Final lesson. (Subsection Disorders of blood and lymph circulation. Inflammation). Practical experience".

Suggested topics for essays.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association

8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 10

Topic: Final lesson. (Subsection Disorders of blood and lymph circulation. Inflammation). Practical experience.

Goal: Check and evaluate the level of mastery of the material on the topics of the unit "Violation of blood and lymph circulation. Inflammation".To find out the ability to apply theoretical knowledge in practice, namely micro- and macroscopic diagnostics.

Basic concepts:provided in the materials of the corresponding topics.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:

*Highlight the definition or provide an explanation.*Hyperemia: physiological, pathological, arterial, venous, edema, anasarca, cyanotic compaction of the spleen, cyanotic compaction of the kidney, brown compaction of the lungs, nutmeg liver; bleeding and hemorrhages: epistaxis, hemoptysis, hemoptysis, cyclic and acyclic uterine bleeding, blood in feces, urine, hematomas, hemorrhagic inclusions, ecchymoses, purpura, pithecia, hemopericardium, hemoperitoneum, hemothorax, hydrothorax, pyothorax, hemarthrosis; thrombosis, thrombus (white, red mixed, occlusive, parietal, moraine, axial, spherical, agonal), embolism (thromboembolism, gas, air, tissue, embolism with foreign bodies, fat, amniotic fluid) Inflammation, acute inflammation, chronic inflammation, exudate (serous exudate, hemorrhagic exudate, catarrhal exudate, purulent exudate, fibrinous exudate: diphtheritic and croupous), transudate, phlegmon, abscess, boil, carbuncle, empyema, CRUP (true and false); interstitial inflammation, inflammatory infiltrate, polyp, condyloma, granuloma (tuberculous granuloma, Pirogov-Langhans cells, syphilitic granuloma, leprous granuloma, Virchow cells, sclerotic granuloma, Mikulich cells).

2.2. Flow chart on the topic as a list of didactic units of the topic:
provided in the relevant previous topics.

2.3. List of questions to check basic knowledge on the subject of the lesson.
Provided in previous related topics.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 Test tasks: provided in the relevant previous topics.

3.2. Algorithm of description of macropreparation and micropreparation.

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the

	discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Molecular-pathomorphological bases of the immune response. The immune system in the prenatal and postnatal period. Pathology of immune processes: amyloidosis, hypersensitivity reactions, transplant rejection. Immune deficiency. Autoimmune diseases".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association

3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 11

Topic: Molecular and pathomorphological bases of the immune response. The immune system in the prenatal and postnatal period. Pathology of immune processes: amyloidosis, hypersensitivity reactions, transplant rejection. Immune deficiency. Autoimmune diseases.

Goal:to learnbasics of immune response and pathomorphology of hypersensitivity reactions. Morphological changes in immune processes: amyloidosis, transplant rejection, as well as immune deficiency and autoimmune diseases.

Basic concepts: Hypersensitivity (anaphylaxis, atopy, granulomatosis,cytotoxic type of hypersensitivity, immunocomplex type of hypersensitivity) Autoimmune diseases (organ-specific autoimmune diseases, organ-nonspecific autoimmune diseases) Immunodeficiency, primary immunodeficiency syndromes, secondary immunodeficiency syndromes, accidental involution of the thymus, transplant rejection, transplant against the host, thymomegaly (sudden infant death syndrome);

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:

*emphasize the definition or provide an explanation.*Hypersensitivity (anaphylaxis, atopy, granulomatosis, cytotoxic type, hypersensitivity, immune complex type of hypersensitivity). Autoimmune diseases (organ-specific autoimmune diseases, non-specific autoimmune diseases). Immunodeficiency, primary immunodeficiency syndromes, secondary immunodeficiency syndromes, accidental involution of the thymus, transplant rejection, "graft against host", thymomegaly (sudden infant death syndrome).

2.2. Flow chart on the topic as a list of didactic units of the topic.

Immunity—it is a complex of reactions aimed at protecting the body from infectious agents of substances that differ from it in biological (antigenic) properties.

*Cellular immunity*is the function of T-lymphocytes; with cellular immunity, effector cells are formed - T-killers, capable of destroying cells that have an antigenic structure, through direct cytotoxicity and through the synthesis of certain substances called lymphokines, they participate in the processes of interaction between cells (macrophages, T cells, B- cells) during an immune response. In addition, two subtypes of T cells take. participation in the regulation of the immune response: T-helpers enhance the immune response; T-suppressors have the opposite effect.

Humoral immunity -it is a function of B cells and is characterized by the transformation of B cells into plasma cells that secrete immunoglobulins (antibodies) that have specific activity against the antigen that has entered the body.

The immune response is characterized:

specificity (reactivity is aimed only at a certain agent, which is called an antigen);

potentiation (the ability to produce an enhanced response when the body receives the same antigen continuously);

immunological memory (the ability to recognize and produce an enhanced response against the same antigen upon repeated exposure to the body, even if the first and subsequent exposures occur after long intervals of time).

These features distinguish the immune response from other non-specific host responses (acute inflammation and non-immune phagocytosis).

*Tolerance to own antigens.*The concept of "own" and "other" is central to immunological reactivity. A large number of molecules in the body are antigens, that is, they cause an immune response when introduced into another body, but are not recognized as antigens by the host. The inability to respond to one's own antigens is called natural tolerance. This phenomenon prevents the host's immune system from destroying its own tissues. Tolerance to one's own antigens develops in the embryonic period, and this is a manifestation of the specificity and memory of the immune response.

*Cellular immunity*is the function of T-lymphocytes; with cellular immunity, effector cells are formed - T-killers, capable of destroying cells that have an antigenic structure, through direct cytotoxicity and through the synthesis of certain substances called lymphokines, they participate in the processes of interaction between cells (macrophages, T cells, B- cells) during an immune response. In addition, two subtypes of T cells take. participation in the regulation of the immune response: T-helpers enhance the immune response; T-suppressors have the opposite effect.

Humoral immunity -it is a function of B cells and is characterized by the transformation of B cells into plasma cells that secrete immunoglobulins (antibodies) that have specific activity against the antigen that has entered the body.

The immune response is characterized:

1) specificity (reactivity is aimed only at a certain agent, which is called an antigen);

2) potentiation (the ability to produce an enhanced response when the body receives the same antigen continuously);

3) immunological memory (the ability to recognize and produce an enhanced response against the same antigen upon repeated exposure to the body, even if the first and subsequent exposures occur after long intervals of time).

These features distinguish the immune response from other non-specific host responses (acute inflammation and non-immune phagocytosis).

*Tolerance to own antigens.*The concept of "own" and "other" is central to immunological reactivity. A large number of molecules in the body are antigens, that is, they cause an immune response when introduced into another body, but are not recognized as antigens by the host. The inability to respond to one's own antigens is called natural tolerance. This phenomenon prevents the host's immune system from destroying its own tissues. Tolerance to one's own antigens develops in the embryonic period, and this is a manifestation of the specificity and memory of the immune response.

Types of immune response

Based on whether the immune system was previously familiar with the antigen or not, two types of immune response are distinguished: primary and secondary.

Primary immune response"occurs upon first encounter with a specific antigen. Although the antigen is recognized almost immediately after exposure, it takes several days before sufficient immunoglobulin is produced to detect an increase in serum immunoglobulin levels. During this latent period, those B cells with receptors that have reacted with a specific antigen go through six to eight successive cycles of division before a large enough clone of antibody-secreting plasma cells is formed.IgM is the first immunoglobulin produced during the primary response, followed by IgG.Switching from IgM to IgG synthesis or other immunoglobulins occurs as a normal phenomenon during the activation of B cells and is carried out as a result of heavy chain gene switching.

*Secondary immune response*occurs when re-meeting with an antigen. Re-recognition occurs immediately and the production of serum immunoglobulins, which is detected by laboratory tests, occurs faster (after 2-3 days) than with the initial response. IgG is the main immunoglobulin that is secreted during the secondary response. In addition, the peak level is higher and the decline is slower than in the primary response.

Abnormalities of the immune response

Basically, immune reactions develop covertly, while they lead either to the complete destruction of the antigenic aggressor; or to partial inhibition of its pathogenic action, ensuring a state of immunity in the body.

However, under some circumstances, these reactions may develop unusually.

In some cases; when a foreign agent is introduced into the body, they are so intense that they lead to tissue damage and are accompanied by the phenomenon of inflammation, then we speak of a reaction (or disease) of hypersensitivity.

Sometimes, under certain conditions, body cells acquire antigenic properties or antibodies are produced in the body that can react with normal cell antigens. In these cases, they talk about diseases due to autoimmunization, or autoimmune diseases.

Finally, there are conditions in which, despite the arrival of antigenic material, immune reactions do not develop. Such conditions are referred to as immune failure or immunodeficiency.

Thus, the immune system, which is normally involved in maintaining homeostasis, can be the source of pathological conditions caused by excessive reaction or insufficient response to aggression, which are designated as immunopathological processes.

*Immunopathological*such processes are called, the development of which is associated with a violation of the function of immunocompetent (lymphoid) tissue. Immunopathological processes form the basis of immunopathology - a branch of medicine that studies all pathological diseases that arise as a result of immunological conflict and violations of immunological homeostasis. In addition to such a broad interpretation of immunopathology, there is another, more narrow one. According to him, immunopathology includes autoimmunization, autoallergy, and autoaggression.

The morphology of immunopathological processes includes the structural reflection of immunogenesis disorders (antigenic stimulation or immune deficiency) and local immune reactions that occur in a sensitized organism - hypersensitivity reactions.

Morphology of disorders of immunogenesis

The morphology of disorders of immunogenesis (immunological homeostasis) can affect both the thymus and peripheral lymphoid tissue and is associated with two types of immune reactions - humoral and cellular.

Changes in the thymus gland (thymus) that occur with disorders of immunogenesis

The thymus belongs to the central organs of the immune system, at the same time it is also a gland of internal secretion, therefore this gland is called a connecting chain - a "switch" between the immune and endocrine systems.

The main functions of the thymus (lymphopoietic, immunoregulatory, and endocrine) are performed primarily thanks to the secretion of hormones, mainly of a polypeptide nature - thymosin, thymopoietin, thymic serum factor, etc. by its epithelial cells. The influence of the thymus on the processes of immunogenesis is also mediated by the endocrine system and regulatory T-lymphocytes - T-effectors, helpers, suppressors.

During a person's life, the thymus is subject to age-related involution, which is characterized by the slow replacement of its tissue with fatty tissue. However, at any age, islands of thymus parenchyma remain in the adipose tissue of the anterior mediastinum, and the secretion of thymic hormones and the production of T-

lymphocytes are partially preserved. Age-related involution of the thymus is one of the reasons for a decrease in the activity of cellular immunity and an increase in the frequency of infectious, autoimmune, and oncological diseases in the elderly. Thymus pathology is associated with aplasia, hypo- and dysplasia, accidental involution, atrophy, thymomegaly and hyperplasia with lymphoid follicles. The development of a number of immunodeficiency syndromes, autoimmune diseases and some endocrine disorders are associated with thymus pathology.

*Aplasia, hypo-, dysplasia of the thymus*are congenital abnormalities of the development of the thymus and are accompanied by a deficiency of the cellular chain of immunity or a combined immune deficiency. Thymic hormones are not produced at all or their production is minimal. In aplasia (agenesis), the thymus is completely absent; with hypo- and dysplasia, its size is reduced; distribution to the cortex and medulla is disturbed, the number of lymphocytes is sharply reduced.

*Accidental involution of the thymus*is a rapid decrease in its mass and volume under the influence of primarily glucocorticosteroids in various stressful situations, including infectious diseases, intoxications, and injuries. At the same time, the production of thymic hormones progressively decreases, the emigration of T-lymphocytes from the thymus increases, although the main mass of them is subject to disintegration on the spot (apoptosis). The functional significance of acute involution of the thymus is unknown, but its delay ("immobile" thymus) is accompanied by a decrease in the activity of the cellular and humoral links of immunity. Accidental involution of the thymus can be reversed, but in cases of an adverse outcome leads to atrophy of the thymus.

*Atrophy of the thymus*develops as an adverse consequence of accidental involution of the thymus and may be the cause of some acquired immunological syndromes (in chronic infectious diseases, immunosuppressive therapy). As a result of the decrease in lymphocytes and the collapse of the network of epithelial cells, the lobes of the thymus parenchyma decrease in volume, the thymic bodies become calcified, and connective and adipose tissue grows in the perivascular spaces. At the same time, the production of thymic hormones is significantly reduced.

*Thymomegaly*characterized by an increase in the mass and volume of the thymus parenchyma above the age norm while maintaining its normal structure. It can be congenital or acquired. Congenital thymomegaly occurs more often in children, less often in adults, and is quite often associated with defects in the development of the nervous and cardiovascular systems, congenital dysfunction of the endocrine system, first of all, chronic insufficiency of the kidneys and gonads. Congenital thymomegaly, especially in infectious diseases, is accompanied by generalized hyperplasia of lymphoid tissue. At the same time, the production of thymic hormones is reduced, violations of mainly the cellular link of immunity are noted, which are close to congenital immunodeficiency syndrome.

The cause of death of patients with thymomegaly can be infectious and infectious-allergic diseases. In connection with endocrine disorders under the influence

of stress factors (medical manipulations, surgical interventions), sudden death is possible.

Previously, cases of thymomegaly were united by the concept of "thymic-lymphatic condition", the basis of which was considered congenital hyperfunction of the thymus. This interpretation is inherently incorrect, therefore the term "thymic-lymphatic condition" is not used in medical everyday life. Nowadays, this condition has acquired a different meaning and reflects immunoendocrine dysfunction of various origins.

*Thymus hyperplasia with lymphoid follicles*characteristic of autoimmune diseases. B-lymphocytes, plasma cells accumulate in the sharply expanded intralobular perivascular spaces of the thymus parenchyma, and lymphoid follicles appear, which are not normally found there. The production of thymic hormones can be increased or decreased. Until recently, the significance of thymus hyperplasia with lymphoid follicles in the pathogenesis of autoimmune diseases was still unknown. It is assumed that damage to the thymus can be one of the reasons for the development of the autoimmune process, but secondary damage to this gland is possible.

Changes in peripheral lymphoid tissue that occur when immunogenesis is impaired

Changes in peripheral lymphoid tissue are most characteristic of antigenic stimulation and its hereditary deficiency.

During antigenic stimulation (sensitization) of the body, the changes in the peripheral lymphoid tissue are unambiguous and are manifested by a macrophage reaction, hyperplasia of lymphocytes with their successive plasmacytic transformation. These changes are complemented by an increase in the permeability of microvessels, swelling of the interstitium and accumulation of protein-polysaccharide (HIK-positive) substances in it (tissue dysproteinosis). The degree of macrophage-plasmacytic transformation of lymphoid tissue reflects the stress of immunogenesis and, above all, the level of antibody (immunoglobulin) formation by plasmacytic cells.

Especially vivid changes with antigenic stimulation are manifested in the lymph nodes (primarily regional in relation to the place of arrival of the antigen) and the spleen.

A large number of plasmablasts and plasma cells appear in the lymph nodes, which increase in size, become full-blooded and swollen, in their cortical layer, in the light centers of the follicles, and in the medullary layer. They displace lymphocytes. The proliferation and desquamation of cells of the sinuses, the formation of a significant number of macrophages and protein-polysaccharide substances in the stroma is noted. The spleen increases, looks full-blooded and juicy; large follicles are clearly visible on the dissection surface. There is hyperplasia and plasmatization of both the red pulp and its follicles, the peripheral zone of which consists entirely of plasmablasts and plasma cells. Along with plasmablasts, there are many macrophages in the red pulp.

If mainly cellular immune reactions develop in response to antigenic stimulation, then mainly sensitized lymphocytes proliferate in the lymph nodes and spleen, and not

plasmablasts and plasma cells. At the same time, there is an expansion of T-dependent zones.

The same changes in the form of cellular hyperplasia and macrophage-plasmacytic transformation, and in some cases myeloma hyperplasia, are found in the bone marrow, portal tracts and sinusoids of the liver, in the interalveolar septa, perivascular and peribronchial tissue, in the interstitium of the kidneys, pancreas and intestines, intermuscular layers, adipose tissue, etc.

*Hereditary deficiency*peripheral lymphoid tissue is characterized by changes in both the spleen and especially the lymph nodes. In the spleen, the size of the follicles is significantly reduced, light centers and plasma cells are absent. Lymph nodes lack follicles and the cortical layer (B-dependent zones), only the cortical layer (T-dependent zone) is preserved. Such changes are characteristic of hereditary immunodeficiency syndromes associated with a defect in humoral immunity.

Immunological hypersensitivity

*Hypersensitivity is a pathological excessively strong immune reaction to a foreign agent,*which leads to damage to body tissues. There are four different types of hypersensitivity. All forms, except for type IV, have a humoral mechanism (that is, they are mediated by antibodies); Type IV hypersensitivity has a cellular mechanism. In all forms, primary exposure to a specific antigen (sensitizing dose) causes a primary immune response (sensitization). After a short period (one or more weeks) during which the immune system is activated, a hypersensitive response occurs to any subsequent exposure to the same antigen.

TypeAnd hypersensitivity (immediate) (atopy; anaphylaxis)

Mechanism of development: the first arrival of an antigen (allergen) activates the immune system, which leads to the synthesis of antibodies - IgE (reagins), which have a specific; reactivity against this antigen. After that, they are fixed on the surface membrane of tissue basophils and blood basophils due to the high affinity (affinity) of IgE to Fc receptors. Synthesis of antibodies in sufficient quantity to develop hypersensitivity continues for one or more weeks. At the next introduction of the same antigen, the interaction of the antibody (IgE) and the antigen occurs on the surface of tissue basophils or blood basophils, which causes their degranulation. From the cytoplasmic granules of tissue basophils, vasoactive substances (histamine and various enzymes involved in the synthesis of bradykinin and leukotrienes (see "Inflammation") that cause vasodilation,

Local manifestations -atopy, a congenital predisposition, familial to a pathological response against certain allergens.

Skin - sudden redness, swelling (hives) and itching occurs when the allergen hits the skin; in some cases – acute dermatitis and eczema.

Mucous membrane of the nose– when inhaling an allergen (for example, plant pollen, animal wool), vasodilation and mucus hypersecretion occur in the nasal mucosa (allergic rhinitis).

Lungs -inhalation of allergens (plant pollen, dust) leads to contraction of bronchial smooth muscles and mucus hypersecretion, which leads to acute airway obstruction and suffocation (allergic bronchial asthma).

Intestine -oral ingestion of an allergen (for example, nuts, shellfish, crabs) causes muscle contraction and fluid excretion, which manifests itself in the form of spastic abdominal pain and diarrhea (allergic gastroenteritis).

Systemic manifestations- anaphylaxis. A rare but extremely life-threatening systemic type I hypersensitivity reaction. The entry of vasoactive amines into the bloodstream causes a contraction of smooth muscles, widespread vasodilation and an increase in vascular permeability with the release of fluid from the vessels into the tissues. The resulting peripheral vascular insufficiency and shock can lead to death within minutes (anaphylactic shock). In less severe cases, the increase in vascular permeability leads to allergic edema, which has the most dangerous manifestation in the larynx, because it can cause fatal asphyxiation.

Systemic anaphylaxis mainly occurs with the injection of allergens (for example, penicillin, foreign serum, local anesthetics, radiopaque substances). Rarely, anaphylaxis can occur when allergens are ingested orally (shellfish, crabs, eggs, berries) or when allergens hit the skin (bee and wasp stings). In sensitized people, even a small amount of allergen can trigger the development of fatal anaphylaxis (penicillin hypersensitivity test).

Type II hypersensitivity

Mechanism of development: type II hypersensitivity is characterized by the reaction of an antibody with an antigen on the surface of a host cell, which causes the destruction of this cell. The antigen can be one's own, but for some reason recognized by the immune system as foreign (at the same time, an autoimmune disease occurs). An antigen can also be external and can accumulate on the cell surface (for example, a drug can be a hapten when combined with a cell membrane protein and thus stimulate an immune response).

A specific antibody, mainly IgG or IgM, that is synthesized against an antigen, interacts with it on the cell surface and causes cell damage in several ways:

*1.*Cell lysis – activation of the complement cascade leads to the formation of the "membrane attacking" complex C5b6789, which causes cell membrane lysis.

2. Phagocytosis -the cell that carries the antigen is engulfed by phagocytic macrophages that have Fc or C3b receptors, which allows them to recognize antigen-gene-antibody complexes on the cell.

*3.*Cellular cytotoxicity - the antigen-antibody complex is recognized by unsensitized "null" lymphocytes (K cells; see Immunity), which destroy the cell. This type of hypersensitivity is sometimes classified separately as type VI hypersensitivity.

4. Change in cell function -an antibody can react with cell surface molecules or receptors to cause either enhancement or inhibition of a particular metabolic response without causing cell necrosis (see Stimulation and Inhibition in Hypersensitivity, below). Some authors classify this phenomenon separately as type V hypersensitivity.

Manifestations of type II hypersensitivity reactionsdepend on the type of cell that carries the antigen. Note that transfusion reactions are actually normal immune responses against foreign cells. They are identical in the mechanism of the type II hypersensitivity reaction and also have an adverse effect on the patient, in connection with which hemotransfusion complications are often considered together with disorders that occur with hypersensitivity.

Type II hypersensitivity occurs in hemotransfusion reactions (antibodies in the patient's serum react with antigens on erythrocytes, causing either indirect intravascular hemolysis by complement or delayed hemolysis as a result of immune phagocytosis by splenic macrophages); hemolytic disease of newborns; hemolytic reactions caused by drugs, infectious diseases (mycoplasma pneumonia, infectious mononucleosis).

Immune complex damage.

The third mechanismassociated with the toxic effect on cells and tissues of circulating immune complexes, which leads to the activation of complement components and the development of reactions of immune complexes (immunocomplex reaction). Accumulation of immune complexes activates complement and causes acute inflammation and necrosis (Reactions such as the Artus phenomenon – with repeated administration of the rabies vaccine; reactions of the serum sickness type – repeated intake of a large amount of antigen, foreign serum proteins, drugs, viral, microbial agents).

The fourth mechanism is caused by the effect on tissues of effector cells of sensitized T-lymphocytes, which exhibit cytotoxicity either directly or through the secretion of lymphokines. Type IV hypersensitivity reactions generally occur 24-72 hours after administration of the antibody. Histological examination of tissues in which a type IV hypersensitivity reaction occurs reveals cell necrosis and pronounced lymphocytic infiltration.

Direct cytotoxicity plays an important role in contact dermatitis, in the response against tumor cells, virus-infected cells, transplanted cells, in some autoimmune diseases.

So, the first immunological mechanisms are a manifestation of humoral immunity (antibodies, complement components, circulating antigen-antibody complexes) and others of cellular immunity (lymphocytes, macrophages). This determines the nature of hypersensitivity reactions and the principle of their classification. Reactions associated with immunopathological mechanisms, which are manifestations of humoral immunity, are called immediate-type hypersensitivity reactions (IHRT), and those associated with immunopathological mechanisms, which are manifestations of cellular immunity, are called delayed-type hypersensitivity reactions (HST). In addition, transplant immunity reactions (rejection reactions) are distinguished.

Hypersensitivity reactions are morphologically reflected in immune inflammation. It is called immune because the trigger for the development of this inflammation is an immune reaction. Immune inflammation can be acute or chronic.

*Immediate type hypersensitivity reaction*morphologically, it is a manifestation of acute immune inflammation. It is characterized by the speed of development, the advantage of alterative and vascular-exudative changes, and the slow course of reparative processes. Alterative changes concern mainly the walls of blood vessels, the main substance and fibrous structures of connective tissue. They are represented by plasma leakage, mucoid and fibrinoid swelling, fibrinoid necrosis. The appearance of coarsely dispersed proteins, fibrin, neutrophils, "digesting" immune complexes, and erythrocytes in the center of immune inflammation is associated with pronounced plasmarrhagic and vascular-exudative reactions. In this regard, fibrinous or fibrinous-hemorrhagic exudate becomes the most characteristic of GNT. Proliferative-reparative reactions in HNT develop later and are poorly expressed. They are manifested by the proliferation of cells of the endothelium and perithelium (adventitia) of vessels and over time coincide with the appearance of mononuclear-histiocytic elements, which reflects the elimination of immune complexes and the beginning of reparative processes. Evaluation of morphological changes in HNT, their belonging to an immune reaction requires evidence using the immunohistochemical method.

The most typical dynamics of morphological changes in HNT is reflected in the Arthus phenomenon, which occurs in sensitized animals upon local administration of a resolving dose of the antigen. In human pathology, GNT is the essence of many bacterial infections, allergic diseases and processes. Manifestations of GNT with the advantage of alteration are constant in tuberculosis, syphilis, they are the basis of vascular changes in rheumatism, systemic lupus erythematosus, glomerulonephritis, nodular periarteritis, etc. Vascular-exudative manifestations of HNT are pronounced in case of croupous inflammation of the lungs.

GNT reactions are similar to so-called reagin reactions, i.e. reactions involving allergic antibodies, or reagins (IgE), fixed on cells. They are distinguished by the surface alteration of cells and tissues, which explains the lack of participation of complement in the reaction and the prevalence of vascular-exudative changes associated with massive degranulation of tissue basophils (labrocytes) and the release of histamine; the infiltrate is dominated by eosinophils - inhibitors of basophils. An example of a reagin reaction can be changes in atopic bronchial asthma.

Delayed type hypersensitivity reaction(GST). Two types of cells participate in this reaction - sensitized lymphocytes and macrophages. Lymphocyte and macrophage infiltration in the focus of immune conflict is a reflection of chronic immune inflammation, which is the basis of GST.

Destruction of the target cell, that is, immunologically determined cellular cytolysis, is, of course, associated with the activation of lysosomal enzymes of killer lymphocytes. At the same time, macrophages enter into a specific reaction with the antigen with the help of mediators of cellular immunity - lymphokines and cytophilic antibodies adsorbed on the surface of these cells. At the same time, contacts appear between lymphocytes and macrophages in the form of cytoplasmic bridges, which may serve to exchange information between cells about the antigen. Immunologically

determined cellular cytolysis can be connected with cellular antibodies, that is, with NK- and K-cells.

Inflammation in the form of lymphohistiocytic and macrophage tissue infiltration in combination with vascular-plasmatic and parenchymatous-dystrophic processes can be recognized as immune, that is, reflecting HST, only if there is evidence of the connection of infiltrated cells with sensitized lymphocytes. This evidence can be found in histochemical and electron microscopic examination.

Clinical and morphological manifestations of HST include the following: a tuberculin-type reaction in the skin in response to the introduction of an antigen, contact dermatitis (contact allergy), autoimmune diseases, immunity to many viral and some bacterial infections (viral hepatitis, tuberculosis, brucellosis). Morphological manifestation of HST is granulomatosis.

Reactions of HNT and HST often combine or change each other, thus reflecting the dynamics of the immunopathological process.

Manifestations of transplant immunity are represented by the reaction of the recipient's body to the donor's genetic transplant, that is, the reaction of transplant rejection. Antigens of the transplant induce the formation of specific antibodies that circulate in the blood, and the production of sensitized lymphocytes, which carry out cellular invasion of the transplant. The main role in the rejection reaction is played by sensitized lymphocytes, so the manifestations of transplant immunity are similar to HST.

The morphological manifestations of the rejection reaction are reduced to the growing infiltration of the transplant mainly by lymphocytes, as well as by histiocytes due to the invasion of these cells and their reproduction in place. Cellular infiltration is accompanied by impaired blood circulation and swelling of the graft. In the end, many neutrophils and macrophages appear among the cells of the infiltrate. It is believed that immune lymphocytes that destroy transplant cells are able to saturate with its antigens, so humoral antibodies directed against transplant antigens not only bind to transplant cells, but also lyse lymphocytes. Enzymes released from activated lymphocytes destroy the transplant cells, which leads to the release of new transplant antigens. This is how the growing enzymatic destruction of the transplant is carried out. Clinical types of transplant rejection: a rapid reaction that occurs within a few minutes after transplantation; acute rejection lasts from several days to months; chronic rejection, characterized by progressive deterioration of organ function over many months or years. The rejection reaction can be suppressed using a number of immunosuppressive agents. This makes it possible to use not only an isograft (recipient and donor - twins), but also an allograft (recipient and donor - foreign), both from a living person and from a corpse, when transplanting organs and tissues. characterized by progressive deterioration of organ function over many months or years. The rejection reaction can be suppressed using a number of immunosuppressive agents. This makes it possible to use not only an isograft (recipient and donor - twins), but also an allograft (recipient and donor - foreign), both from a living person and from a corpse, when transplanting organs and tissues. characterized by progressive deterioration of organ function over

many months or years. The rejection reaction can be suppressed using a number of immunosuppressive agents. This makes it possible to use not only an isograft (recipient and donor - twins), but also an allograft (recipient and donor - foreign), both from a living person and from a corpse, when transplanting organs and tissues.

Autoimmunization and autoimmune diseases

Autoimmunization(autoallergy, autoaggression) is a condition characterized by the appearance of a reaction of the immune system to normal antigens of one's own tissues.

Autoimmunization is closely related to the concept of immunological tolerance (from Latin tolerare – to bear, tolerate), which is characterized by a state of reactivity ("tolerance") of lymphoid tissue in relation to antigens that can cause an immune response. During the maturation of lymphoid tissue, immunological tolerance to antigens of all organs and systems occurs, except for the tissues of the eye, thyroid gland, gonads and adrenal glands, brain and nerves. It is taken into account that the antigens of these organs and tissues are separated from the lymphoid tissue by physiological barriers, which explains the lack of tolerance to them by the immunocompetent system. The immune system begins to recognize "own" and "foreign" tissue antigens in a newborn a few weeks after birth. At the same time, the production of autoantibodies in a small amount constantly occurs throughout life, and autoantibodies are believed to be involved in the regulation of various body functions. their action is under the control of T-suppressors and anti-idiotypic antibodies, which prevents the development of an autoimmune process.

Among the etiological factors of autoimmunization, chronic viral infections, radiation, and genetic disorders occupy a significant place. Etiology is closely related to pathogenesis. In the pathogenesis of autoimmune diseases, there are causative, initiating and contributing factors. The causative factors should include some genes of the HLA system, which determine the quantitative and qualitative individual abilities of the immune response; the hormonal background, related, first of all, to gender (autoimmune diseases are 6-9 times more common in women than in men) and genetically determined features of the cells of the target organs of the autoimmune process. An unfavorable combination of these factors determines 50% of the risk of developing the disease. Initiating factors can be viral and bacterial infections, physical and chemical damage to both immune system organs and target organs.

Autoimmune diseasesare diseases that arise as a result of autoimmunization, that is, the aggression of autoantibodies, circulating immune complexes containing autoantigens, and effector immune cells (killer lymphocytes) against antigens of the body's own tissues. Therefore, autoimmune diseases are also called autoaggressive.

Guided by the mechanism of autoimmunization, two groups of autoimmune diseases are distinguished. The first group is organ-specific autoimmune diseases that arise in connection with damage to the physiological barriers of immunologically separated organs, which allows the immune system to respond to their unchanged antigens by producing autoantibodies and sensitized lymphocytes. At the same time,

morphological changes occur in the organs, which are characteristic mainly for GST - the tissue of the organs is infiltrated by lymphocytes, parenchymal elements die, and in the end, sclerosis develops. This group includes thyroiditis (Hashimoto's disease), encephalomyelitis, polyneuritis, multiple sclerosis, idiopathic Addison's disease, aspermatogenia, sympathetic ophthalmia.

The second group is organ-specific autoimmune diseases. The leading cause of these diseases is a violation of control of immunological homeostasis by the lymphoid system. At the same time, autoimmunization develops in relation to antigens of many organs and tissues, which do not have organ specificity and are not able to cause the production of antibodies during parenteral administration. Organs and tissues develop morphological changes that are characteristic of both delayed-type and especially immediate-type hypersensitivity reactions. This group of autoimmune diseases should include systemic lupus erythematosus, rheumatoid arthritis, systemic scleroderma, dermatomyositis (a group of rheumatic diseases), secondary thrombotic thrombocytopenic purpura (Moshkovich's disease).

Autoimmune diseases of the intermediate type are also known, that is, they are close to autoimmune diseases of the first or second type. These are myasthenia gravis, type 1 diabetes, thyrotoxicosis, Sjogren's and Goodpascher's syndromes, etc.

In addition to autoimmune diseases, there are diseases with autoimmune disorders. The appearance of autoantigens in these diseases is associated with a change in the antigenic properties of tissues and organs - denaturation of tissue proteins (in case of burns, irradiation, trauma, chronic inflammation, viral infections); the formation of an autoantigen is possible under the influence of a bacterial antigen, especially a cross-reactive one (eg, in glomerulonephritis, rheumatism). In the formation of autoantigen, great importance is attached to the hapten mechanism, and the role of a hapten can be the products of the body's metabolism, as well as microorganisms, toxins, and medicinal substances. Autoimmunization in these conditions causes not the occurrence of the disease, but the progression of local (organ) changes characteristic of it, which reflect the morphology of delayed and immediate types of hypersensitivity reactions.

Immunodeficiency syndromes

Immunodeficiency syndromesrepresent an extraordinary manifestation of the insufficiency of the immune system. They can be primary, due to underdevelopment (hypoplasia, aplasia) of the immune system - hereditary and congenital immunodeficiency syndromes, or secondary (acquired), which arise in connection with disease or treatment.

Primary immunodeficiency syndromes

Primary immunodeficiency syndromes can be a manifestation of insufficiency:

1) cellular and humoral immunity (combined)

2) cellular immunity;

3) humoral immunity.

*Syndromes of insufficient cellular and humoral immunity are called combined.*Most patients have an autosomal recessive form,occur in children and

newborns (Swiss-type agammaglobulinemia, or Glanzmann-Riniker syndrome, Louis-Bar ataxia-telangiectasia). In these syndromes, hypoplasia of both the thymus and peripheral lymphoid tissue is found, the number of lymphocytes is reduced in the thymus, as well as in the lymph nodes, spleen, and peripheral blood. There are no immunoglobulins in the serum, which determines the defect of cellular and humoral immunity. In connection with insufficient immunity, such children often develop infectious diseases (viral, fungal, bacterial), which recur and cause severe complications (pneumonia, meningitis, sepsis), and physical development is delayed. In combined immunodeficiency syndromes, malformations and malignant mesenchymal tumors often occur.

*Syndromes of insufficient cellular immunity*in some cases, of course, they follow the autosomal dominant type (immunodeficiency with achondroplasia, or Mac-Cusick syndrome), in others, they are congenital (agenesis or hypoplasia of the thymus, or Dy-George syndrome). The syndrome is characterized by a lack of T-lymphocytes in the blood, in the thymus-dependent areas of the lymph nodes and spleen. Signs of insufficient cellular immunity are manifested in the form of severe viral and fungal infectious diseases in childhood. Children die from developmental defects or from complications of infectious diseases.

*Syndromes of humoral immunity deficiency*have a hereditary nature, and their linkage with the X-chromosome has been established. Children of the first five years of life are sick. Some syndromes (X-linked agammaglobulinemia or Bruton's syndrome) are characterized by the loss of the ability to synthesize all immunoglobulins, which is morphologically confirmed by the absence of B-dependent zones and cells of the plasmacytic line in the peripheral lymphoid tissue, primarily in the lymph nodes and spleen. Observed in boys, infectious diseases break up mainly in the second half of the first year of life after the level of passively transmitted maternal antibodies falls.

Isolated InA deficiency is the most common immunodeficiency, resulting from a defect in terminal differentiation of plasma cells secreting InA. In most patients, InA deficiency is asymptomatic. Only some patients are prone to liver and intestinal infections.

Secondary immunodeficiency syndromes

Secondary (acquired) immunodeficiency syndromes differ from primary ones in that they arise in connection with the disease or as a result of drug therapy.

Among the diseases that lead to the development of the immune system deficiency, acquired immune deficiency syndrome, or AIDS, which is widespread in many countries of the world, is an independent disease, the causative agent of which is a virus (see Viral diseases). The development of secondary immunodeficiency syndromes can also be caused by various infections, leukemias, malignant lymphomas (lymphogranulomatosis, reticulosarcoma), thymoma, sarcoidosis. In these diseases, there is a deficiency of humoral and cellular immunity due to a defect in the population of both B and T lymphocytes, and possibly their precursors.

Radiation therapy, the use of corticosteroids, and immunosuppressants after organ transplantation occupy a significant place among the types of treatment that lead to secondary insufficiency of the immune system.

Insufficiency of the immune system, which occurs in connection with the treatment of one or another disease, is considered as a pathology of therapy (iatrogeny).

Immunodeficiency is always accompanied by the development of opportunistic infections and at the final stage, most often Kaposi's sarcoma and malignant B-cell lymphomas. The occurrence of infectious diseases depends on the type of immunodeficiency. A decrease in the number of T cells predisposes to the development of infectious diseases caused by viruses, mycobacteria, and fungi. Deficiency of B cells predisposes to the development of purulent bacterial infectious diseases

The emergence of malignant neoplasms can be associated with either a violation of the immune response aimed at removing malignant cells, or with the nominal stimulation of the damaged immune system, when the normal control of cell proliferation is disturbed.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. The concept of immunity, its types. Morphological features of central and peripheral organs of immunogenesis.
2. Involution of the thymus, age-related and accidental, causes of development, morphological characteristics.
3. Characteristics of immunological reactions according to the degree of activity: hypoergic, normergic, hyperergic.
4. Hypersensitivity reactions of immediate and delayed type. Morphological characteristics.
5. The concept of immunodeficiency, congenital and acquired. Immunodeficiencies: Classification, examples.
6. Autoimmune diseases. Definition, classification, features of development, morphological characteristics.
7. Transplant rejection reaction: definition, types, morphological characteristics.
8. The "graft against the host" reaction.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

1. The 38-year-old patient died duringan attack of bronchial asthma that could not be stopped. During the histological examination, accumulation of mucus was found in the lumen of the bronchi, in the wall of the bronchi there are many mast cells (labrocytes), many of them in a state of degranulation, as well as many eosinophils. What is the pathogenesis of these changes in the bronchi?

AND Atopy, anaphylaxis

BCytotoxic, cytolytic effect of antibodies
CIimmunocomplex mechanism
DCellularly determined cytolysis
Granulomatosis
2. An animal sensitized by tuberculin, intraperitoneally administered tuberculin. After 24 hours, at laparotomy, venous hyperemia and edema of the peritoneum were revealed. There are a large number of lymphocytes and monocytes in smears-prints from the peritoneum. What is the pathological process in the animal?
AND Allergic inflammation
B Serous inflammation
Purulent inflammation
DFibrinous inflammation
Aseptic inflammation
3. In a 31-year-old patient, after the treatment of phlegmon of the cheek (due to a cut during shaving), the regional submandibular lymph nodes were enlarged for a long time. A biopsy of one of them revealed full blood, swelling in the cortical and brain layers, and in the expanded follicular centers - macrophage-plasmacytic infiltration. The immunogram shows an increased content of B-lymphocytes with a decrease in the level of T-lymphocytes. What is the pathology in the lymph nodes?
AND Lymphadenitis
B Lymphogranulomatosis
Lymphosarcoma
Hyperplasia of the lymph node
E-
4. A 23-year-old patient developed a urinary syndrome (hematuria, proteinuria, leukocyturia) after having angina. A puncture biopsy of the kidneys revealed a pattern of intracapillary proliferative glomerulonephritis, and electron microscopy revealed large subepithelial deposits. What is the pathogenesis of this disease?
AND Immune complex mechanism
Atopy, anaphylaxis with the formation of IgE and their fixation on mast cells
Cytotoxic, cytolytic effect of antibodies
DCellularly determined cytolysis
Granulomatosis
5. A 10-year-old child underwent a Mantoux test (with tuberculin). After 48 hours, a papule up to 8 mm in diameter appeared at the site of tuberculin injection. What type of hypersensitivity reaction developed after tuberculin administration?:
AND Artyus phenomenon
BAtopic reaction
Serum sickness
Hypersensitivity type IV (HRT)
Anaphylaxis
6. An 8-year-old child developed swelling of the limbs, increased blood pressure, and urine in the form of "meat slops" 2 weeks after a severe acute respiratory syndrome.

Poststreptococcal glomerulonephritis with nephrotic syndrome was diagnosed. What pathological process is the basis of kidney pathology?:
AND Deposition of immune complexes between the basal membrane and endothelial cells of kidney glomeruli
BDamage of kidney tubules by streptococcal toxins
CDeposition of immune complexes in tubules between the basal membrane and epithelial cells
Formation of antibodies against the basement membrane
The toxic effect of acetylsalicylic acid on the kidneys
7. A 30-year-old patient turned to the doctor with complaints about the presence of a rash, redness and swelling of the skin, which appears after eating certain products. Urticaria is this?:
AND Local manifestation of type I hypersensitivity
BLocal manifestation of type II hypersensitivity
Type IV hypersensitivity reaction
Systemic type I hypersensitivity reaction
Posthemotransfusion reaction
8. The patient developed allergic angioedema after taking the analgin tablet. The most dangerous complication of such edema is?:
AND Asphyxia due to swelling of the larynx
BAcute airway obstruction
CSpastic abdominal pains
Hypersecretion of mucus
Heart failure
9. An 8-year-old child developed nephrotic syndrome 10 days after a sore throat, acute poststreptococcal glomerulonephritis was diagnosed. Such a reaction is a manifestation of type III hypersensitivity, which is the result?:
AND Immune complex damage
BAutoimmune damage
Antibody damage
Cytotoxicity of T-Lymphocytes
Reaction of antibodies with antigens on the surface of host cells
10. In a patient with bronchial asthma, a viral infection provoked an asthmatic status with a fatal outcome. Histological examination of the lungs revealed spasm and swelling of the bronchioles, infiltration of lymphocytes, eosinophils, and other leukocytes, as well as degranulation of labrocytes, was revealed in their walls. What is the mechanism of hypersensitivity underlying the described changes?:
AND Reagin reaction of hypersensitivity
BImmune-induced cell cytolysis
Hot
DAutoimmune
EImmunocomplex

11. A 43-year-old man underwent a kidney transplant. The function of the graft progressively worsened, and a month later a graft-nephrectomy was performed. Acute vasculitis, necrosis of renal tubules and lymphocytic infiltration of the interstitial tissue were revealed during microscopic examination of the extracted graft. Are these changes the result?:
AND Reaction of acute transplant rejection
BCyclosporin nephrotoxicity
Reaction of the most acute graft rejection
Atrophy of the kidney
Reaction of chronic transplant rejection
11. In order to close the tissue defect, a patient who received burns was transplanted with his own skin, taken from the unaffected areas of the thigh. This type of transplantation is called?:
AND Autotransplantation
Isotransplantation
Allotransplantation
Xenotransplantation
EHeterotransplantation
12. When examining biopsy material taken from a patient with autoimmune gastritis, lymphocytic and macrophage infiltration was noted in the gastric mucosa. An expansion of T-dependent zones, proliferation of sensitized lymphocytes in the paracortical zone was found in the lymph nodes. The identified morphological changes indicate development?:
AND IV (delayed) type hypersensitivity
B Hypersensitivity type II
Hypersensitivity type III
Hypersensitivities of the I (immediate) type
Hypersensitivity type V
13. At the autopsy of the corpse of a 10-month-old child who died of a severe form of pneumonia, a complete absence of the thymus was found, the size of the follicles in the spleen was significantly reduced, and there were no light centers in them; Lymphoid follicles and cortical layer are absent in lymph nodes. Could the cause of the identified structural changes be:
AND Thymus aplasia
B Hypoplasia of the thymus
C Hyperplasia of the thymus
D Agenesis of the thymus
E Atrophy of the thymus

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

8. Specify the name of the organ or ego part;
9. Specify the dimensions of the body (length, width, thickness);

10. Specify the surface of the organ, the condition of the capsule, overlap;
11. Specify the consistency of the organ;
12. The type and structure of the organ at autopsy;
13. Indicate the presence of a pathological formation (if any);
14. Conclude.

Description of the micropreparation:
1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the

	discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Regeneration. Structural basis of physiological adaptation of organs and cells. Morphology of cell accommodation processes. Compensatory and adaptive processes".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 12

Topic: Regeneration. Structural basis of physiological adaptation of organs and cells. Morphology of cell accommodation processes. Compensatory and adaptive processes

Goal: To study the types, mechanisms, and stages of adaptive and adaptive processes that develop normally and especially in pathological conditions. Learn the types and stages of regeneration, definition and essence of organization, sclerosis, wound healing.

Basic concepts: Hypertrophy, hyperplasia, false hyperplasia (hypertrophy), atrophy, cachexia, brown cardiac atrophy, brown liver atrophy, metaplasia, dysplasia, scar, granulation tissue, pathological regeneration.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:

*emphasize the definition or provide an explanation.*Hypertrophy, hyperplasia, false hyperplasia (hypertrophy), atrophy, cachexia, brown atrophy of the heart, brown atrophy of the liver, metaplasia, dysplasia, scar, granulation tissue, pathological regeneration.

2.2. block diagram on the topic as a list of didactic units of the topic.

Adjustment (adaptation) is the processes of vital activity, thanks to whichm is the relationship between the organism and the external environment. The device is aimed at preserving the species, therefore it covers both health and disease.

Compensation is a private manifestation of adaptation, aimed at correcting the impaired function during illness ("self-preservation" in a critical situation).

Adaptive and compensatory reactions of the whole organism, which vary quantitatively and change qualitatively, take place in all its diseases.

All compensatory and adaptive processes are based on two basic principles of living systems: 1) constancy of preservation of form and function under changing conditions of existence (homeostasis); 2) mobility and variability of form and function in the process of adaptation to environmental conditions.

These two properties are fixed in the process of phylo- and ontogenesis at different levels of organization (molecular, cellular, tissue, organ, system, organismic).

At the molecular level of organization, compensatory and adaptive reactions are ensured by: 1) high-quality stability of protein metabolism; 2) its wide quantitative fluctuations; 3) the presence of a general plastic reserve of cells (stock of structurally

organized proteins, stock of RNA, ability for rapid synthesis of RNA, DNA reproduction); 4) reversibility of differentiation of structure and function.

Mechanisms of regulation of the molecular level of the organization of compensatory and adaptive processes: 1) enzymatic; 2) humoral (selective action of a set of metabolites, hormones); 3) nervous.

At the cellular level of the organization, compensatory and adaptive reactions are provided by: 1) the general plastic reserve of the cell; 2) a large supply of cells and their ability to alternate between work and rest; 3) the ability of the cell to reproduce; 4) their ability to metaplasia.

At the organ level: 1) a reserve of functional units of the organ; 2) heterogeneity of their functions; 3) the ability to regenerate functional units.

At the systemic level: quantitative wide fluctuations of structure and function.

All the above-listed levels of organization of compensatory and adaptive reactions ensure the adaptation of the organism as a whole to the action of the environment.

Three phases were identified in their development:

1. The genesis of compensation or the phase of formation of compensation. At this moment, the functions are sharply strained, the metabolism is excited, the reserves may not be enough, and dystrophic changes, hypoxia, develop at first. This phase is sometimes called the emergency phase.

Fixation phase. Compensatory and adaptive reactions are fully developed. Dystrophic changes decrease, protein synthesis begins to increase, the weight and mass of organs increases, function sometimes increases by 50-100-150%.

Exhaustion phase. Exhaustion always occurs after increased work. Sooner or later, but always. Since ATP resynthesis is weakened. When all ATP is used, the function stops completely.

Adaptation in pathology can reflect various functional states: functional tension, reduction or disruption of tissue (organ) function. In this regard, it can be manifested by various pathological processes: 1) atrophy; 2) hypertrophy (hyperplasia); 3) organization; 4) reconstruction of tissues; 5) metaplasia; 6) dysplasia.

Atrophy- lifelong decrease in the volume of cells, tissues, organs with suspension or termination of their function, but not every decrease in the organ refers to atrophy. In connection with a violation in the process of ontogenesis, the organ may be completely absent - agenesis; preserve the appearance of the early embryo - aplasia; not reaching full development - hypoplasia. If there is a decrease in all organs and a general underdevelopment of all body systems, then they speak of dwarfism.

Physiological and pathological atrophy are distinguished. Physiological atrophy is observed throughout a person's life. Thus, after birth, the umbilical arteries atrophy and are subject to obliteration, the gonads atrophy in the elderly, the bones and intervertebral cartilages in the elderly. Pathological atrophy occurs for various reasons; among which the most important are insufficient nutrition, disorders of blood circulation and activity of endocrine glands, central and peripheral nervous system, intoxication. After eliminating the causes that caused atrophy, if it has not reached a

high degree, a complete restoration of the structure and function of the organ or system is possible.

Pathological atrophy can be both general and local. General atrophy, or exhaustion, occurs in the form of alimentary exhaustion (with starvation or impaired absorption of food); exhaustion with cancer cachexia; exhaustion with pituitary cachexia; with cerebral cachexia (damage to the hypothalamus), as well as with other diseases (chronic infections, such as tuberculosis, dysentery, brucellosis, etc.). The characteristic appearance of patients with exhaustion is a sharp weight loss, a decrease in body weight, there is no subcutaneous adipose tissue; where it is preserved, it has a brownish color (accumulation of lipochrome pigment). Muscles are atrophic, the skin is dry, flabby; internal organs are reduced in size. Brown atrophy (accumulation of lipofuscin pigment in cells) occurs in the liver and myocardium. In endocrine glands, atrophic and dystrophic changes differ in their intensity depending on the cause of exhaustion; in bones - osteoporosis; in the cerebral cortex - cells of dead nerve cells.

Local atrophy occurs for various reasons. The following types are distinguished: dysfunctional; caused by insufficient blood supply; from squeezing; neurotic; under the influence of physical and chemical factors.

Dysfunctional atrophy - occurs as a result of a decrease in the function of an organ - muscle atrophy in case of bone fractures, joint diseases, when movements are limited; optic nerve after surgical removal of the eye; edges of the tooth cell after tooth extraction. At the same time, the intensity of metabolism in the tissues is reduced, they receive an insufficient amount of blood and nutrients.

Atrophy from insufficient blood supply occurs as a result of narrowing of arteries; insufficient blood flow leads to hypoxia, as a result of which the activity of parenchymal organs decreases, the size of cells decreases. Hypoxia stimulates the proliferation of fibroblasts with subsequent development of sclerosis. Such a process is observed in the myocardium, when atrophy of myocardiocytes and diffuse cardiosclerosis occur with progressive atherosclerosis of the coronary arteries; with sclerosis of kidney vessels, atrophy and shrinkage of the kidneys develop.

Atrophy from compression can develop even in organs consisting of dense tissue. With long-term compression, tissue integrity disorders occur (wrinkles in the bodies of the vertebrae and in the sternum when in contact with an aneurysm of the aorta. Atrophy from compression occurs in the kidneys when the outflow of urine is difficult. The basis of atrophy from compression is insufficient blood flow to cells and hypoxia that occurs in the in connection with this.

Neurotic atrophy is due to a violation of the connection between the organ and the nervous system, which occurs when the nerve conductors are destroyed. Most often, this type of atrophy occurs in striated muscles due to the death of motor neurons of the anterior horns of the spinal cord or nerve trunks that innervate these muscles (in poliomyelitis, inflammation of the facial nerve).

Atrophy under the influence of physical and chemical factors is quite common. Under the influence of radiation energy, atrophy is especially pronounced in the bone

marrow and genitals. With long-term use of ACTH and corticosteroids, atrophy of the cortex of the adrenal glands may occur and their insufficiency may develop.

The appearance of organs with local atrophy is diverse. In most cases, the size of organs decreases, the surface is smooth (smooth atrophy). Less often, organs, such as kidneys, liver, acquire a granular appearance (granular atrophy). With hydronephrosis, hydrocephalus, pseudohypertrophy, the organs are enlarged, but not due to parenchymal elements, but due to the accumulation of fluid or the growth of fatty tissue. Sometimes this fiber grows around the atrophied organ (kidney).

The value of atrophy for the body is determined by the degree of reduction of the organ and decrease in its function. If atrophy and sclerosis do not reach a significant degree, then after eliminating the cause that caused the atrophy, it is possible to restore the structure and function, which was discussed above. Under certain conditions, an atrophied organ may even undergo hypertrophy over time.

Hypertrophy (hyperplasia) can have an adaptive nature - an increase in the volume of a cell, tissue, or organ due to cell proliferation or an increase in the number and size of intracellular ultrastructures. Two types of hypertrophies should be classified as adaptive: neurohumoral hypertrophy (hyperplasia) and hypertrophic growths.

Neurohumoral hypertrophy and hyperplasia occur when the function of the endocrine glands is disturbed (hormonal or correlative hypertrophy and hyperplasia). The physiological prototype of such hypertrophy and hyperplasia, which have an adaptive value, can be the hypertrophy of the uterus and mammary glands during pregnancy and lactation. In conditions where ovarian dysfunction occurs, hyperplasia of the glands develops in the mucous membrane of the uterus, sometimes with cystic expansion of their lumen - the so-called glandular-cystic hyperplasia of the endometrium, which is accompanied by irregular uterine bleeding. With atrophic processes in the testicles in the chest gland of men, hyperplasia of the glandular lobes develops, which leads to an increase in the size of the entire gland - gynecomastia. Hyperfunction of the anterior lobe of the pituitary gland, which occurs with its adenoma, accompanied by an increase in organs and protruding parts of the skeleton - occurs. Correlative hypertrophies and hyperplasias, which arise as a reaction to certain hormonally determined stimuli, are often the basis for the development of the tumor process.

Hypertrophic growths, which lead to an increase in the size of tissues and organs, arise due to various reasons. They are quite common in chronic inflammation (e.g., in mucous membranes with the formation of polyps), in disorders of lymphatic circulation in the lower extremities and lymph stagnation, which leads to the growth of connective tissue (elephantitis). Hypertrophic growth of fat and connective tissue occurs with partial or complete atrophy of the organ (false hypertrophy). Thus, with muscle atrophy, fatty tissue grows between their fibers; with atrophy of the kidney, the growth of adipose tissue around it increases; with brain atrophy, the bones of the skull thicken; when the blood pressure in the vessels decreases, the intima grows and thickens.

All the processes of hypertrophic growth of supporting tissue listed above, filling the place previously occupied by an organ or tissue, are called vacant hypertrophy.

Organization,as one of the forms of manifestation of adaptation, it represents the replacement of the center of necrosis or thrombus by connective tissue, as well as encapsulation. The replacement of the center of necrosis or thrombotic masses by connective tissue (organization itself) occurs when the masses are subject to resorption and at the same time young connective tissue grows in them, which then turns into a scar. They talk about encapsulation in those cases when dead masses, animal parasites, foreign bodies are not resorbed, but become covered with connective tissue and are separated from the rest of the organ by a capsule. Masses of necrosis are permeated with lime; petrifications occur. Sometimes bone tissue is formed in the inner layers of the capsule by metaplasia. Multinucleated giant cells (giant cells of foreign bodies) are formed around foreign bodies and animal parasites in the granulation tissue.

Hyperplasia, regeneration and accommodation are the basis of adaptive tissue remodeling. An example of reconstruction can be collateral blood circulation, which occurs when the flow of blood in the main vessels is obstructed. With it, there is an expansion of the lumen of the veins and arteries that depart from the affected main vessel, thickening of the walls due to muscle hypertrophy and neoplasms of elastic fibers. The structure of small vessels takes on the character of larger ones. Reconstruction of the spongy substance in the bones is observed when the direction of the load on the bone is changed (after a fracture, with rickets, joint diseases). Tissue remodeling occurs in some tissues under changed conditions of their existence. For example, in the lungs, in the foci of atelectasis, the compacted alveolar epithelium acquires a cubic shape due to the cessation of air flow. Nephrothelium, which covers the cavity of the capsule of the renal glomerulus, when it is excluded from its function, it becomes cubic. Such changes in the epithelium are called histological accommodation.

Metaplasia- the transition of one type of tissue to another, related to it. Metaplasia is most often found in the epithelium and connective tissue, less often in other tissues. Reconstruction of one tissue into another is possible within one germ layer and develops during the proliferation of young cells (during regeneration, neoplasms). Metaplasia always occurs in connection with the previous proliferation of undifferentiated cells, that is, it is indirect. Heterotopia or heteroplasia, when the epithelium does not appear in the usual place due to a developmental defect, should not be taken for metaplasia.

Epithelial metaplasia is most often manifested as the transformation of a prismatic epithelium into a flat one with keratinization (epidermal or squamous epithelial metaplasia). It is observed in the respiratory tract with chronic inflammation, with a deficiency of vitamin A in the pancreas, prostate, mammary, and thyroid glands, in the appendix of the testicle with inflammation and hormonal influences. Metaplasia begins with the proliferation of cambial cells, which differentiate in the direction of not prismatic, but multilayered flat epithelium. The transition of a multi-layered flat epithelium without cornification into a cylindrical one is called prosoplasia. Metaplasia

of the stomach epithelium into the intestinal epithelium (intestinal metaplasia or enterolization of the gastric mucosa) is possible, as well as metaplasia of the intestinal epithelium into the gastric epithelium (gastric metaplasia of the intestinal mucosa).

The term "dysplasia" as a kind of adaptive process is often used in oncomorphology. It is characterized by significant violations of the proliferation and differentiation of the epithelium with the development of cellular atypia and a violation of histoarchitectonics. Cellular atypia is represented by a different size and shape of cells, an increase in the size of the nuclei and their hyperchromia, an increase in the number of mitotic figures, and the appearance of atypical mitoses. Violation of histoarchitectonics in dysplasia manifests itself as a loss of the polarity of the epithelium, and sometimes of its properties that are characteristic of a given tissue or a given organ (loss of histo- or organ-specificity of the epithelium). The basement membrane is not disturbed. Thus, dysplasia is not a cellular concept, but a tissue concept.

Depending on the degree of proliferation and the state of cell and tissue atypia, three stages (degrees) of dysplasia are distinguished: I - mild (minor), II - moderate (medium), III - severe (significant). Most often, dysplasia occurs during inflammatory and regenerative processes and reflects a violation of cell proliferation and differentiation.

Compensation- the reaction of the organism (system, organ, tissue, cell), the manifestation of which is the correction of impaired functions during the disease. The compensatory process proceeds in stages; three phases are distinguished in it: formation, consolidation and exhaustion. The phase of formation of compensation, which is also called the "emergency" phase, is characterized by the inclusion of all structural reserves and changes in the metabolism of the organ (system) in response to the pathogenic influence. In the fixation phase, the compensatory possibilities are revealed to the fullest - there is a restructuring of the structure and exchange of the organ (system), which ensures their function under conditions of increased load. This phase can last quite a long time. However, depending on many conditions (age of the patient, duration, severity of the disease, nature of treatment, etc.), the insufficiency of compensatory capabilities develops, which characterizes the exhaustion phase of compensation or decompensation (eg, decompensated heart disease, decompensated liver cirrhosis). The development of the phases of the compensated process (establishment, consolidation and depletion of decompensation) is determined by a complex system of reflex acts of the nervous system, as well as humoral influences. In this regard, during decompensation, it is very important to look for its cause not only in the diseased organ, but also outside it, among those mechanisms that regulate its activity.

Morphologically, compensation is mainly manifested by hypertrophy. At the same time, the organs increase in size, but retain their configuration. The cavity of the organ either becomes wide (eccentric hypertrophy) or decreases (concentric hypertrophy). In the cells of a hypertrophied organ, structural and functional changes are observed, which indicate an increase in the intensity of metabolism. The enhanced

function of the hypertrophied organ occurs due to an increase in the number of its specific intracellular formations; and in some cases this process unfolds on the basis of pre-existing cells and leads to an increase in their volume (hypertrophy), in others it is accompanied by the formation of new cells (cellular hyperplasia).

There are two types of compensatory hypertrophy: working (compensatory) and vicarious (replacement).

Working (compensatory) hypertrophy develops with increased work of the organ, while there is an increase in the volume (number) of cells that determine its specialized function. Work-related hypertrophy is observed under increased load and in physiological conditions (hypertrophy of the heart and hypertrophy of skeletal muscles in athletes and people engaged in physical labor). In diseases, increased work of the organ is necessary in cases of defects in it, which are compensated by increased work of parts of the organ that have preserved their structure and function. Working hypertrophy occurs in the heart, gastrointestinal tract, urinary tract and other organs. Hypertrophy of the heart is the most vivid example of compensatory hypertrophy and reaches the highest degrees in congenital and acquired heart defects, which are accompanied by stenosis of the atrioventricular openings and peripheral vascular tracts of the ventricles, with arterial hypertension, narrowing of the aorta, sclerosis of pulmonary vessels, etc. The myocardium is subject to hypertrophy mainly, which performs the main work in specific conditions of impaired blood circulation (left ventricle with aortic valve defects; right ventricle with mitral valve defect). At the same time, the weight of the heart can exceed the normal weight by 3-4 times and reach 900-1000 g. The size of the heart also increases. Hypertrophy of the myocardium occurs due to an increase in the mass of the sarcoplasm of myocardiocytes, the size of their nuclei, the number and size of myofibrils and mitochondria, that is, hyperplasia of intracellular ultrastructures occurs. At the same time, the volume of muscle fibers increases. Simultaneously with the hypertrophy of the myocardium, there is a concomitant hyperplasia of the fibrous structures of the stroma, intramural vascular branches and the nervous system of the heart. So, at the heart of myocardial hypertrophy are the processes that run together in muscle fibers, stroma of the myocardium, its vascular system and intramural nervous system. Each of them is an integral part of the concept of "hypertrophied heart" and ensures its participation in the development and maintenance of the increased work of the heart over a long, sometimes multi-year period.

With compensated myocardial hypertrophy, the length of the heart increases due to the distal tract (from the base of the semilunar valves of the aorta to the most distant point of the apex of the heart); the supply tract (from the apex of the heart to the place of attachment of the back sail of the bicuspid valve) does not change. There is an expansion of the heart cavities, which is defined as active compensatory or tonogenic.

The development of compensatory hypertrophy is facilitated not only by mechanical factors that impede blood flow, but also by neurohumoral influences. The full implementation of compensatory hypertrophy requires a certain level of innervation of the heart and hormonal balance. Hypertrophy of the wall of the stomach

or intestine occurs above the area of narrowing of their lumen. The smooth muscle layer of their wall hypertrophies, functional capacity is preserved. The lumen of the cavity above the narrowing is widened. After some time, the compensation phase is replaced by decompensation due to the failure of the hypertrophied muscle layer. Bladder wall hypertrophy occurs with prostate hyperplasia (adenoma), which narrows the urethra, and other complications of bladder emptying. The bladder wall thickens, muscular trabeculae are visible on the side of the mucous membrane (trabecular hypertrophy).

Vicarious (substitute) hypertrophy is observed when one of the paired organs (lungs, kidneys) dies due to disease or after surgery. Compensation of the impaired function is provided by increased work of the preserved organ, which is subject to hypertrophy. Vicarious hypertrophy is similar to regenerative hypertrophy in its pathogenetic essence and significance for the body. A complex of reflex and humoral influences plays a significant role in its occurrence, as in the case of compensatory hypertrophy.

Regeneration- restoration of tissue structural elements instead of dead ones. In the biological sense, regeneration is an adaptive process developed in the course of evolution and characteristic of all living things. Restoration of structure and function can occur with the help of cellular or intracellular hyperplastic processes. On this basis, cellular and intracellular forms of regeneration are distinguished. The cellular form of regeneration is characterized by mitotic and amitotic cell reproduction; for the intracellular form of regeneration, which can be organoid and intraorganoid - an increase in the number (hyperplasia) and size (hypertrophy) of ultrastructures (nuclei, nucleolus, mitochondria, ribosomes, Golgi complex) and their components. The intracellular form of regeneration is universal, because it is characteristic of all tissues and organs. However, the structural and functional specialization of organs and tissues in phylo- and ontogenesis "selected" for some the predominantly cellular form, for others - predominantly or exclusively intracellular, for the third - equally both forms of regeneration. The advantage of one or another form of regeneration in the relevant organs and tissues is determined by their functional purpose, structural and functional specialization.

The morphogenesis of the regenerative process consists of two phases - proliferation and differentiation. These phases are especially well expressed in the cellular form of regeneration. In the proliferation phase, young, undifferentiated cells multiply. These cells are called cambial cells, stem cells and precursor cells. Each tissue is characterized by its own cambial cells, which differ in the degree of proliferative activity and specialization; however, one stem cell can be the progenitor of several types of cells (stem cell of the hematopoietic system, lymphoid tissue, some cellular representatives of connective tissue).

In the differentiation phase, young cells mature, their structural and functional specialization occurs. The same change in hyperplasia of ultrastructures by their differentiation is the basis of the mechanism of intracellular regeneration.

The development of the regenerative process depends on a number of general and local conditions or factors. General factors should include age, constitution, nature of nutrition, state of metabolism and hematopoiesis; to local ones - the state of innervation, blood and lymph circulation in the tissue, the proliferative activity of its cells, the nature of the pathological process.

There are three types of regeneration: physiological, reparative and pathological.

*Physiological regeneration*occurs throughout life and is characterized by constant renewal of cells, fibrous structures and the main substance of connective tissue. There are no such structures that are not subject to physiological regeneration. Where the cellular form of regeneration predominates, cell repair takes place. This is how the covering epithelium of the skin and mucous membranes, the secretory epithelium of the exocrine glands, cells that line the serous and synovial membranes, cellular elements of connective tissue, erythrocytes, leukocytes and blood platelets, etc., are constantly changing. In tissues and organs, where the cellular form of regeneration is lost, intracellular structures are restored. Together with the restoration of cells and subcellular structures, there is constant biochemical regeneration, that is, restoration of the molecular composition of all body components.

*Reparative or restorative regeneration*observed in various pathological processes that lead to damage to cells and tissues. The mechanisms of reparative and physiological regeneration are the same; reparative regeneration is nothing more than enhanced physiological regeneration. However, due to the fact that reparative regeneration is stimulated by pathological processes, it has qualitative morphological differences. Reparative regeneration can be complete or incomplete. Full regeneration, or restitution, is characterized by filling the defect with tissue identical to the dead tissue; it develops mainly in tissues where cellular regeneration prevails. In case of incomplete regeneration, i.e. tissue healing with a scar, hypertrophy occurs as an expression of the regenerative process, therefore it is called regenerative; it contains the biological meaning of reparative regeneration.

We are talking about pathological regeneration in cases where, for one reason or another, there is a distortion of the regenerative process, a violation of the change in the phases of proliferation and differentiation. A manifestation of pathological regeneration is the excessive or insufficient formation of regenerative tissue (hyper- or hyporegeneration), as well as the transformation of one type of tissue into another during the regeneration process, excessive regeneration of peripheral nerves and excessive formation of bone callus during fracture growth, slow wound healing and epithelial metaplasia in the cell chronic inflammation. Pathological regeneration mostly develops in the event of violations of general and local conditions of regeneration (innervation disorders, protein and vitamin starvation, chronic inflammation, etc.).

Pathological regeneration is observed in cases where there is a violation of the phases of cell proliferation and differentiation.

Pathological regeneration is manifested either in excess or insufficient formation of regenerating tissue, as well as transformation during regeneration of one type of

tissue into another (metaplasia). Examples of pathological regeneration can be the formation of a colloid scar on the skin, the formation of a bone callus during the fusion of fractures, sluggish wound healing, metaplasia (transformation) of the gastric epithelium into intestinal epithelium in chronic gastritis, etc.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Concept of adaptation and compensation as additive processes.
2. Stages (phases) of adaptation, morphological characteristics.
3. Classification of adaptation processes, reasons for their development.
4. Hypertrophy (hyperplasia), definition, classification, morphological characteristics (physiological, pathological, certain types; true, false).
5. Atrophy, definition, classification, morphological characteristics (physiological, pathological, individual types).
6. Metaplasia, definition, classification, morphological characteristics (separate types).
7. Concept of dysplasia. Clinical significance.
8. Concept of regeneration, classification (physiological, reparative, pathological, full, partial).
9. Regeneration of certain types of tissues (muscle, epithelial (glands, kidneys, liver, etc.), nervous (CNS, PNS), bones, etc.)
10. Wound healing: types, stages; granulation tissue: concept, structure, clinical significance.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

1. Women suffering from diabetesabet, turn to the surgeon with complaints about the presence of ulcers on the skin of both lower legs that do not heal for a long time. The surgeon decided that the process is a manifestation of:

And Atrophy
B Reparative regeneration
C Pathological regeneration
D Metaplasia
E Organization

2. The patient has a deep torn wound with uneven edges, covered with pus. Marginal areas of juicy granulation tissue that does not rise above the level of damage. What wound healing:

And Healing under the scab.
B Primary tension healing.
C Focal agglutination
D Immediate closure of the epithelial tissue defect.
E Organization of the wound.

3. In the bronchus biopsy of a patient who abused smoking, chronic inflammation and transformation of a single-layered ciliated epithelium into a multilayered flat epithelium was found in the thickened mucosa. Which process is most likely?:
ANDMetaplasia
B Epithelial hypertrophy
C Squamous cell carcinoma
D Epithelial hyperplasia
E Leukoplakia
4. When examining from the oral cavity to the mucous membrane of the cheek, a dense whitish spot with a diameter of about 1 cm was found, which is slightly raised above the level of the mucous membrane. What is the name of this painful process?:
And Leukoplakia
B Erythroplakia
C Organization
D Mucoid edema
E Carnification
5. After a traumatic injury to the liver, the structure and function of the liver was completely restored over time. What do you call this type of regeneration?:
And Restitution
B Pathological regeneration
C Transformation
D Normal physiological regeneration
E Replacement
6. A 35-year-old man developed a keloid scar on the inner surface of his right hand after electrosurgery, and non-healing ulcers on his forearm. Both of these processes are examples of:
A Pathological regeneration
B Reparative regeneration
C Vicari hypertrophy
D Protective hyperplasia
E Metaplasia
7. During a microscopic examination of the lung tissue of a child who died as a result of pneumonia, it was established that the mucous membrane of the bronchi is largely represented by a multi-layered non-keratinized flat epithelium. What process occurs in the bronchi? :
ANDMetaplasia
In Protective hyperplasia
C Pathological regeneration
D Pathological hypertrophy
E Organization
8. When microscopically examining a bronchobioptate of a patient who had chronic bronchitis for a long time, it was found that the mucous membrane of the bronchial

tubes is represented by multilayered flat epithelium in some areas. This is a manifestation of:
ANDMetaplasia
B Hypertrophy
C Regeneration
D Atrophy
E Norma
9. Microscopic examination of a gastric mucosa biopsy revealed the presence of glandular foci of intestinal metaplasia in the pyloric section. The most likely cause of these changes is:
ANDReflux of intestinal contents
In Alcohol abuse
C Irregular nutrition
D Reflux of gastric contents
E Liver disease
10. After a fall, a child developed a small bruise on his knee, which completely epithelized after a while. What form of regeneration was observed in this case? :
ANDReparative
B Pathological
C Physiological
D Intracellular
E Renewable

3.2. Algorithm of description of macropreparation and micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluatedtheoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests

- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Oncogenesis. Anatomical and microscopic features and types of growth of benign and malignant tumors. Morphological characteristics of the main stages of development of malignant tumors. Clinical and morphological nomenclature of tumors. Benign and malignant non-epithelial (mesenchymal) tumors. Sarcoma: features of development and metastasis. Tumors of fibroblastic, myofibroblastic and fibrohistiocytic origin. Tumors from adipose and muscle tissue, tumors from blood vessels."

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 13

Topic: Oncogenesis. Anatomical and microscopic features and types of growth of benign and malignant tumors. Morphological characteristics of the main stages of development of malignant tumors. Clinical and morphological nomenclature of tumors. Benign and malignant non-epithelial (mesenchymal) tumors. Sarcoma: features of development and metastasis. Tumors of fibroblastic, myofibroblastic and fibrohistiocytic origin. Tumors from adipose and muscle tissue, tumors from blood vessels.

Goal: To learn the theories of oncogenesis and to recover from aanatomical and microscopic features and types of growth of benign and malignant tumors. To know the morphological characteristics of the main stages of the development of malignant tumors. Know and be able to apply the clinical and morphological nomenclature of tumors. To study benign and malignant non-epithelial (mesenchymal) tumors; sarcoma:

features of development and metastasis. To be able to differentiate tumors of fibroblastic, myofibroblastic and fibrohistiocytic origin, as well as tumors from adipose and muscle tissue, tumors from blood vessels.

Basic concepts:tumor; invasive growth, appositional growth, expansive growth, unicentric growth, multicentric growth, exophytic, endophytic growth; atypism, morphological atypism, tissue atypism, cellular atypism, anaplasia; metastases; sarcoma, fibrosarcoma, liposarcoma, malignant hibernoma, angiosarcoma, osteosarcoma, chondrosarcoma, myosarcoma (rhabdomyosarcoma, leiomyosarcoma); fibroma, desmoid, leiomyoma, rhabdomyoma, lipoma, hibernoma, chondroma, osteoma, angioma (lymphangioma, hemangioma), hemopericytoma, glomus angioma.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:

*emphasize the definition or provide an explanation.*Tumor; invasive growth, appositional growth, expansive growth, unicentric growth, multicentric growth, exophytic growth, endophytic growth; atypism, morphological atypism, tissue atypism, cellular atypism, anaplasia; metastases; sarcoma, fibrosarcoma, liposarcoma, malignant hibernoma, angiosarcoma, osteosarcoma, chondrosarcoma, myosarcoma (rhabdomyosarcoma, leiomyosarcoma); fibroma, desmoid, leiomyoma, rhabdomyoma, lipoma, hibernoma, chondroma, osteoma, angioma (lymphangioma, hemangioma), hemopericytoma, glomus angioma.

2.2. Flow chart on the topic as a list of didactic units of the topic.

*Tumor*is a newly formed heterogeneous tissue, the basis of which is the endless reproduction of cells due to changes in their genetic apparatus. The formed tumor is distinguished by the peculiarities of its growth, metabolism, interaction with other tissues, is characterized by a certain autonomy in relation to the whole organism, which can be considered as a certain independence of its further development.

A tumor can arise from any tissue that is capable of proliferation processes, but it does not arise from highly specialized, mature cells. The level of morbidity and mortality from tumors is different, which is explained by the state of ecology, ethnic customs, heredity, etc. The most important arethe following theories of tumorgrowth: physicochemical theory (theory of carcinogens), virogenetic theory, dysontogenetic, polyetiological theory.

According to the physicochemical theory, the main role belongs to the action of physical and chemical carcinogens, that is, substances capable of causing tumors.

Physical carcinogens include: solar, cosmic, ionizing radiation, radioactive substances. Physical carcinogens exert their effect through damage to the cell's genome. The carcinogenic effect of the indicated factors can also be potentiated by the action of other carcinogenic agents - chemical (tobacco smoking, aniline, asbestos) and viral (human papilloma virus, Abstein-Barr virus, hepatitis B and C virus). Proponents of the viral-genetic theory believe that tumors can be caused by so-called oncogenic viruses, which contain DNA and RNA. DNA viruses cause malignant transformation of cells in 1:107 cases, most often they cause infectious diseases (adenovirus, chicken pox virus, hepatitis B virus). RNA viruses more often cause malignant transformation of cells because, leaving the cell, they do not damage its membrane and it does not die, as in the case of DNA viruses. According to the dysontogenetic theory, tumors arise from embryonic cell-tissue shifts and defectively developed tissues under the influence of various provoking factors. The polyetiological theory unites all the remaining existing factors that can change the genome of a cell and cause its malignant transformation.

Patho- and morphogenesis of tumors can be formulated on the basis of the indicated theories. Pathogenesis (carcinogenesis) considers the mechanisms of tumor genesis, types and mechanisms of blastomatous action of various pathogenic agents.

The following stages are distinguished: the initiation stage involves changes in the genome of a somatic cell under the influence of pathogenic (carcinogenic) agents; the intermediate stage is the activation of proto-oncogenes (normal cell genes) with their transition into oncogenes, which code for the production of oncoproteins as a result of the disruption of the function of regulatory genes. Activation of proto-oncogenes is accompanied by suppression of anti-oncogenes; the promotion stage - there is tumor transformation of cells with unlimited, uncontrolled growth and tumor formation.

Mechanisms of activation of proto-oncogenes: insertion mechanism - the appearance of viral genes in the genome of a somatic cell activates nearby proto-oncogenes; chromosomal translocations - observed in Burkitt's lymphoma, chronic myelogenous leukemia; point mutations; amplification - increase in the number of gene copies.

Morphogenesis is the process of formation and development of a tumor in a morphological display. There are two morphogenetic options for the occurrence of tumors:

The appearance of a tumor does not happen by chance, but gradually, step by step, according to separate stages of changes in the mother's tissue: precancerous stage; diffuse or focal hyperplasia, dysplasia; stage "cancer in situ" - non-invasive cancer, when the integrity of the basement membrane is preserved; the stage of invasive growth - the stage of a formed malignant tumor; metastasis

Some of the tumors can pass the stage of a benign tumor (cancer of the stomach, colon).

The appearance of the tumor is diverse. Most often, it has the appearance of a node with an uneven surface or has a diffuse form, in the form of a thickening of the mother's tissue, differing from it only in color and consistency.

The size of the tumor depends on its "age", although both the nature of the tumor itself and the structure of the parent tissue are important. If the tumor does not have a noticeable harmful effect on the body, it can reach significant sizes, in other cases the body dies much earlier.

The consistency of the tumor is also different: sometimes it is denser (tumors made of bone, cartilage, fibrous tissue), then it is looser, when the parenchyma prevails over the stroma in the tumor.

One of the characteristics of tumors is the autonomy of their development, which has a relative character, because the tumor tissue constantly receives from the macroorganism various nutrients, oxygen, hormones, and cytokines that arrive with the blood stream. In addition, its growth is influenced by the immune system. In other words, tumor autonomy must be understood not as a certain independence of tumor cells from the body, but as their acquisition of self-regulation properties. Autonomy is more pronounced in malignant tumors (cancer, sarcoma). They grow quickly, destroying the parent tissue; it is less pronounced in benign tumors, some of them are subject to regulatory actions of the body, grow slowly, without destroying the parent tissue. In both cases, the cells switch to the autocrine mechanism of regulating their development, producing growth factors or oncoproteins - analogs of growth factors.

The structure of tumors is diverse depending on the tissue from which they are formed, the nature and direction of their growth. Organoid and histoid types of structure are distinguished. In the first case, the tumor consists of two clearly defined elements: parenchyma and stroma. They are not isolated from each other, but closely related to each other biologically and histogenetically. Nutrition of the parenchyma depends on the state of blood supply through the vessels of the stroma, on the other hand, the parenchyma affects the state of the stroma (the amount and nature of the stroma depend on the nature and state of the parenchyma). Otherwise, in some tumors, the stroma may not be sharply expressed and is represented only by vessels with a small amount of connective tissue (histoid type of structure).

Tumor development is characterized by considerable diversity. However, it always grows by itself, that is, due to the reproduction of its own cells, no matter what size and distribution it reaches. As a rule, no new cells are involved in the growth process. In some cases, neoplastic transformation occurs within the tumor field. Different types of tumor growth are distinguished - expansive, infiltrative, exophytic, endophytic, unicentric, multicentric. In case of expansive growth (characteristic of benign tumors), there is no destruction of the surrounding tissues, growth occurs with gradual separation of the tumor and the appearance of a capsule that separates the tumor from the parent tissue. Invasive (infiltrative) growth, on the contrary, is characteristic of malignant tumors. It is characterized by the destruction of surrounding tissues (histolysis). However, invasive growth does not always coincide with tumor malignancy - there is a group of so-called semi-malignant tumors that grow infiltratively, but do not metastasize, but are morphologically mature forms. As a result of invasive growth, malignant tumors are fused with surrounding tissues and are therefore clinically immobile. Their border with the mother part is unclear.

The rate of tumor growth depends on its type. Immature (malignant) tumors, which mainly consist of parenchyma, grow quite quickly, while mature ones, as well as tumors with a relatively developed stroma, grow quite slowly. The speed of tumor growth is one of the most important signs of tumor malignancy, because the degree of germination and destruction of surrounding tissues depends on it. Therefore, it can be said that the fastest growing malignant swellings, the elements of which are the least mature. Such factors as inflammation, the period of puberty, pregnancy, stress, etc. can have some importance for the growth rate. Invasion is most often observed in the direction of least resistance: along interstitial gaps, along nerve fibers, blood and lymphatic vessels. Exophytic growth is the expansive growth of a tumor into an organ cavity. At the same time, it can fill a significant part of it (cancer of the stomach, intestines, bronchus). Endophytic growth is the infiltrative growth of a tumor deep into the organ wall. At the same time, it can be imperceptible from the outside and can be seen only in the section, in the form of tissue that grows through the wall of the organ. When a tumor arises from a single germ, it is said that its growth is unicentric, when a tumor grows from several tumor germs, it is said to be a multicentric growth. In the latter case, several tumor nodes appear in one organ (chondroma of the fingers), in other cases we can talk about tumors of the same type that arise simultaneously or gradually in different parts of the body, completely independently of each other. Such tumors almost always have a systemic nature, that is, they arise in certain body systems (numerous skin tumors - lipomatosis; of the nervous system - neurofibromatosis or Recklinghausen's disease, hemoblastosis, etc.). In addition, simultaneous formation of several tumors in the same patient is also possible (dimorphic tumors).

One of the important signs of tumors is their progression, i.e., a tendency in the direction of constant clonal evolution of tumor cells. It was determined that most tumors arise from one cell of one tumor embryo, that is, they have a monoclonal growth pattern. Over time, the tumor becomes increasingly heterogeneous, that is, various cell clones appear that "provide" various signs of a growing malignant tumor (recurrences, metastases, invasive growth, atypism).

*Atypicalism*is one of the most important signs of a tumor, which determines the origin of the tumor, its morphology, place in the qualification scheme, features of clinical manifestations and prognosis. This feature is the basis of such manifestations of tumor growth, which were previously united by the terms anaplasia and cataplasia, which are used to this day.

Morphological atypismtumors can be tissue and cellular. Tissue atypism is characterized by a violation of tissue interactions characteristic of normal tissues or organs. It is based on violations of the ratio of parenchyma and stroma, as well as changes in the size and shape of tissue structures. Cellular atypism at the light-optical level is characterized by polymorphism or, on the contrary, monomorphism of cells, nuclei and nucleoli, hyperchromatosis, a violation (increase) of the karyoplasmic index due to an increase in the size of nuclei, asymmetric hypo- and hyperchromic mitoses, etc. Cellular atypism is sometimes so pronounced that it is impossible to establish the histogenesis of the tumor, and when it reaches the extreme degree of cataplasia, tumor

cells become monomorphic. The appearance of unlimited proliferation of cells in the tumor,

At the ultrastructural level, morphological atypism is characterized by changes in the nucleus and cytoplasm of the tumor cell. In the nucleus, there is a violation of the structure and arrangement of chromatin in the form of clusters under the karyolemma: the amount of heterochromatin (containing inactive DNA) increases in relation to euchromatin (containing active DNA). In the nuclei there are various inclusions (vesicles, intussusceptions of the karyolem), the size of the nucleoli increases. In the cytoplasm, the number of mitochondria decreases, large organelles appear, the number of ribosomes increases, and the number of contacts of the karyolemma with the membranes of the organelles increases.

Biochemically atypismmanifested by a number of metabolic features in tumor cells. Tumor tissues are rich in cholesterol, glycogen and nucleic acids, glycolytic processes prevail over oxidative ones, which is accompanied by the accumulation of lactic acid.

Histochemical atypismreflects both morphological and biochemical features of the tumor. It is characterized by the fact that various histochemical changes in the activity of various enzymes, accumulation and redistribution of glycoaminoglycans, proteins and lipids appear in the cells. Specific enzymes were found in some tumors, which is important for differential morphological diagnosis.

Antigenic atypismcharacterized by the antigenic diversity of the antigenic composition of the tumor. There are: antigens of viral tumors, antigens of tumors caused by carcinogens, tumor-specific antigens, embryonic antigens, heterogeneous antigens.

Functional disordersin tumor cells depend on the degree of morphological and biochemical atypia. More differentiated tumors retain the functional features of the cells of the parent tissue. low-differentiated, as a rule, lose the functions of the mother tissue (organ), which can have adverse consequences (tumors of the adrenal glands, pancreas).

Any tumor first forms a so-called primary node. Benign tumors remain in the form of a slowly growing nodule. Malignant tumors, on the contrary, due to invasive growth penetrate into lymphatic and blood vessels, their cells are transferred to other organs, where secondary nodes (metastases) are formed, which are hematogenous, lymphogenic, implantation, perineural. The process of metastasis has a cascade character and manifests itself in the form of separate stages: invasion of tumor cells into the vessel lumen; tumor embolus transport; adhesion of cells on the surface of the endothelium and exit into the perivascular space (extravasation); formation of secondary nodes (metastases).

All tumors can be classified according to the two most common principles: clinical and anatomical and histogenetic.

*According to the clinical and anatomical principle*distinguish between mature, homologous or benign tumors and immature, heterologous, or malignant tumors (cancers and sarcomas). Benign tumors consist of more differentiated tissue with signs

of tissue (rather than cellular) atypism, grow mainly expansively (the exception is the so-called tumors with locally destructive growth, or semi-malignant tumors from blood vessels, cartilage, fibrous tissue, etc.), not metastasize As a rule, necrosis (decay) is rarely observed in these tumors, but sometimes amyloidosis or hyalinosis of the stroma, hemorrhages occur. Malignant tumors consist of undifferentiated tissue with signs of cellular and tissue atypism, characterized by infiltrative growth, occurrence of metastases, recurrences, as well as necrosis (disintegration) with the development of bleeding and hemorrhages.

On the basis of the histogenetic principle, benign and malignant tumors are distinguished:

Organ non-specific epithelial tumors
Organ-specific epithelial tumors
Mesenchymal tumors
Tumors of melanin-forming tissues
Tumors of the nervous system and meninges
Tumors of the blood system
Teratomas.

Thus, the problem of tumors is one of the most relevant in modern medicine. This is due to the high frequency of their distribution and the lack of clear ideas in various aspects of the problem (etiology, patho- and morphogenesis, classification, etc.).

In recent years, certain trends have been identified regarding the epidemiology of various tumors. So, for example, there is an increase in the incidence and mortality rates from cancer in all countries of the world; cancer diseases begin to appear in all age groups, although most often - after 50 years; revealed gender differences in the incidence of certain forms of cancer among men and women; and also the structure of morbidity and mortality from cancer is constantly changing due to the increase in the frequency of some diseases and the decrease in the frequency of others.

Non-epithelial tumors

Non-epithelial tumors include neoplasms of mesenchymal and neuroectodermal origin. This is the most numerous and most diverse group of tumors in terms of histological structure. At the end of the 40s of the last century, according to the assumptions of the outstanding American oncologist APStout, a significant part of these tumors, located between the epidermis and the bone system, was separated into a separate group called "soft tissue tumors". After 20 years, this term was adopted in all countries of the world and taken by the WHO as the basis of the international classification of tumors. Today, this group of soft tissue tumors includes 115 separate nosological forms of tumors and tumor-like processes.

The group of tumors of mesenchymal origin is distinguished by a special number of different histological variants of structures. Mesenchyme in ontogenesis gives rise to connective tissue, blood vessels, muscles, tissues of the musculoskeletal system, serous membranes, which under certain conditions can be the source of tumors.

Tumors of soft tissues.

Classifications of soft tissues differ in complexity and ambiguity. Like all tumors, neoplasms of soft tissues are classified according to histogenesis, degree of maturity and clinical course:

1. Tumors of fibrous tissue: mature, benign (fibroma, dermoid); immature, malignant (fibrosarcoma).

2. Tumors from adipose tissue: mature, benign (lipoma, hibernoma); immature, malignant (liposarcoma, malignant hibernoma).

3. Tumors of muscle tissue (from smooth and striated): mature, benign from smooth muscles (leiomyoma); mature, benign from striated muscles (rhabdomyoma); immature, malignant from smooth muscles (leiomyosarcoma); immature, malignant from striated muscles (rhabdomysarcoma).

4 Tumors of blood and lymphatic vessels: mature, benign (hem-, lymphangioma, hemangiopericytoma, glomusangioma); immature, malignant (hem-, lymphangioendothelioma, malignant hemangiopericytoma).

5. Tumors of synovial tissues: mature, benign (benign synovial oma); immature, malignant (malignant synovioma).

6 Tumors of mesothelial tissue: mature, benign (benign mesothelioma); immature, malignant (malignant mesothelioma).

In addition to soft tissue tumors, non-epithelial tumors include neoplasms of melanin-forming tissue and bones, which are divided into bone-forming and cartilage-forming: mature, benign - chondrosteoma, immature, malignant - chondrosteosarcoma.

Mature, benign tumors of the connective tissue itself.

A fibroma is a mature tumor of fibrous connective tissue. It occurs in all age groups with the same frequency in men and women. It is localized more often between the epidermis and the bone in the subcutaneous fatty tissue, in the tendons and fascia of the upper and lower limbs, trunk. In internal organs, this tumor is extremely rare.

Fibroma has the appearance of a node with clear boundaries, dense or soft consistency depending on the histological structure, pink-white on section with pronounced fibrousness.

Microscopically, the fibroma is represented by bundles of connective tissue fibers, which have different lengths and thicknesses, located in different directions. The polymorphism of fibroblasts is weakly expressed, the nuclei are hyperchromic.

Depending on the predominance of cellular or fibrous components, two types of fibroma are distinguished: dense with a dominance of collagen bundles over cells and soft, which consists of loose fibrous connective tissue with a large number of cells.

Clinically, fibroma grows slowly, does not have a general effect on the body, if it is not localized in vital organs, then its course is benign. The probability of malignancy is small. The exception is soft fibroids, which often recur. Some authors classify soft fibroma as differentiated fibrosarcoma.

Desmoid (desmoid fibroma) is a connective tissue neoplasm that, according to the histological picture, resembles a fibroma. Differs infiltrative growth. Tissue and cellular atypism are weakly expressed. It occurs mostly in women after childbirth. In rare cases, it is observed in men and children. Depending on the localization, the

following are distinguished: abdominal desmoid (when localized in the thickness of the anterior abdominal wall); extra-abdominal desmoid.

Abdominal desmoid is relatively benign, not prone to malignancy. Extra-abdominal desmoid or aggressive fibromatosis is often observed at a young age in both men and women. It is localized in places of aponeurosis and fascia on the limbs, in the shoulder girdle, buttocks. It is distinguished by rapid aggressive infiltrative growth, despite the absence of a large number of mitoses. It often recurs and often becomes malignant.

Malignant tumors of the connective tissue itself

Fibrosarcoma is an immature malignant tumor of fibrous connective tissue. Fibrosarcomas are relatively rare tumors. In the past, they ranked first in frequency among non-epithelial malignant neoplasms. After Stout's proposal, only those malignant tumors that produce mature collagen types I or III and do not form other structures were considered fibrosarcomas. Many tumors that were considered fibrosarcomas were classified as synovial sarcomas, malignant histiocytomas, leiomyosarcomas. Tumors are localized most often on the thigh, shoulder, trunk.

Fibrosarcoma can grow in the form of a node and in the form of an infiltrate

Microscopically, it consists of immature fibroblast-like cells and collagen fibers. There are differentiated and poorly differentiated fibrosarcomas.

Differentiated fibrosarcomas are characterized by pronounced polymorphism and hyperchromia of nuclei. Low-differentiated fibrosarcomas are characterized by monoformism, dyschromia and hypochromia of the nuclei, many atypical mitoses. The two most unfavorable prognostic features of fibrosarcoma are hypochromic nuclei and foci of myxomatosis. Fibrosarcomas metastasize mostly hematogenously to the lungs, less often to the liver, and then lymphogenously to regional lymph nodes. The prognosis for poorly differentiated fibrosarcomas is much worse (about 50% of patients die in the first five years).

Mature, benign tumors from adipose tissue.

Lipoma is one of the most common soft tissue tumors. It occurs more often in women in all age groups. Can occur anywhere there is adipose tissue. It can rarely be localized in internal organs. Often there are multiple.

A lipoma often has the appearance of a node of a partial structure (many layers of connective tissue), soft-elastic consistency, yellow in appearance, resembling adipose tissue in appearance. When localized between muscles, it can be vaguely separated, simulating infiltrative growth. It can reach large sizes (more than 20 cm in diameter), especially with retroperitoneal localization.

Microscopicallythe tumor is basically built like normal adipose tissue and differs from it in different sizes of lobules and fat cells. Due to the presence of a large number of layers of dense fibrous connective tissue, it indicates a fibrolipoma. A sufficient number of vessels in the tumor in some cases allows us to talk about an angiolipoma.

Clinically, in most cases, lipoma has a benign course. However, in connection with multicentric growth, relapses may occur due to incomplete removal of the tumor field. With retroperitoneal localization, malignancy of the tumor is often noted.

Hibernoma- mature benign tumor from brown fat. It is more common in women in all age groups. Brown fat is usually found in humans in the embryonic period. Microscopically, brown fat cells differ in the presence of many fat vacuoles in the cytoplasm, which give it a foamy appearance, the nuclei are located in the center of the cell.

Hibernoma is localized most often on the neck, back, hips, abdominal wall, in the mediastinum, that is, in places where brown fat is normally found during embryogenesis.

It has the form of a node of a lobular structure, brown in color.

Microscopicallyconsists of polygonal and round cells, they form particles that are separated by thin layers of connective tissue. Cell nuclei are centrally located and contain one nucleolus. Cytoplasm is fine-grained, eosinophilic or foamy (multilocular fat cells). The chemical composition of fat differs even in one cell. Cholesterol is often detected, which is clearly visible in polarized light.

Hibernoma does not recur and does not metastasize.

Immature, malignant tumors from adipose tissue.

Liposarcoma- an immature malignant tumor from adipose tissue. The tumor is more common in men in all age groups. Most often, it occurs in the soft tissues of the thigh, lower leg, and retroperitoneal region. The tumor can reach large sizes, and its mass can reach several kilograms.

Liposarcoma has the form of a node or a conglomerate of nodes with infiltration of surrounding tissues. The consistency is dense, the cut surface is juicy, variegated - with foci of oozing, hemorrhages, and necrosis. It is often white, juicy, reminiscent of "fish meat".

Microscopicallypronounced tissue and cellular polymorphism. It consists of lipoblasts of various degrees of maturity, there are giant cells with chimeric nuclei. Based on the dominance of certain cell forms that make up the tumor, the following are distinguished: highly differentiated liposarcoma; polymorphic (low-differentiated) liposarcoma.

The latter has the most malignant course. Because liposarcomas can often be multiple, developing simultaneously or sequentially in one or different areas of the body. Most variants of liposarcoma clinically progress slowly and rarely metastasize. Some of them, for example, round cell liposarcoma, do not differ in course from other sarcomas - they grow quickly, recur and metastasize mainly hematogenously in the lungs.

Malignant hibernoma- an immature, malignant tumor of brown fat. Tumor localization, sex and age of patients coincide with similar indicators for hibernoma.

Macroscopicallymalignant hibernoma resembles liposarcoma. When localized under the skin, it is often covered with ulcers.

Microscopicallycharacterized by sharply expressed polymorphism of multilocular cells that have a polygonal shape. There are many giant uninucleate and multinucleate cells with basophilic homogeneous and fine-grained cytoplasm. There are few mitoses.

It very rarely metastasizes - mainly in the lungs by the hematogenous route.

Tumors of muscle tissue (smooth and striated).

Leiomyoma– a mature, benign tumor of smooth muscles. It occurs at any age in both men and women.

Leiomyoma is localized in the skin (from the muscles that raise the hair, from the vessel wall), in the uterus, in the muscular shell of the gastrointestinal tract.

Macroscopicallythe tumor is a clearly separated nodule of dense consistency, fibrous on section. The size of the tumor is very variable, sometimes a leiomyoma can reach a size of 30 cm or more. Leiomyomas are often multiple or isolated, or form a conglomerate of nodes.

Microscopicallyleiomyoma formed from spindle-shaped tumor cells that form bundles going in different directions. With special research methods, myofibrils are detected in the cytoplasm. Sometimes the nuclei in myoma form rhythmic structures, the so-called polysad structures, which are an indicator of tumor growth. When the connective tissue component predominates, it is called fibromyoma. The more connective tissue in a tumor, the slower it grows. With a sufficient number of vessels, the tumor is called an angioleiomyoma. Epithelioid leiomyoma is distinguished by the shape of the cells. All variants of leiomyoma are benign. Uterine fibroids have the greatest clinical significance. Uterine leiomyomas often occur in women aged 30-50 years. According to the histological picture, they more often have the structure of fibromyoma.

Depending on the localization in the uterus, leiomyomas are distinguished: submucosal; intramural (in the thickness of the muscle wall); subserous

Intramurally located fibroids are practically asymptomatic, with submucosal localization, frequent minor bleeding is often observed in the clinic, sometimes pronounced uterine bleeding is possible, requiring surgical intervention. Subserosally located nodes can squeeze the ureters with the development of hydronephrosis, pyelonephritis. In the post-menopausal period, the reverse development of tumor nodes is described. It is necessary to know that the rapid growth of the tumor during this period indicates the possible malignancy of the neoplasm.

Leiomyosarcoma (malignant leiomyoma)- an immature malignant tumor of smooth muscle tissue. It is localized more often in the gastrointestinal tract, mostly in the colon, after that - in the retroperitoneal space, in the soft tissues of the limbs, and in the uterus. It occurs more often at a young age, very rarely in children.

Macroscopicallymore often has the form of a node, which can reach more than 30 cm in diameter. Infiltrating growth is not always obvious.

Microscopicallythere are two types of leiomyosarcoma - highly differentiated and poorly differentiated. Highly differentiated are very difficult to distinguish microscopically from leiomyomas. The most important differential feature is the presence of many atypical mitoses. Low-differentiated leiomyosarcomas are characterized by sharp cataplasia of tumor cells, the appearance of giant cells, and significant polymorphism.

Leiomyosarcomas metastasize early and widely, mainly by the hematogenous route, giving multiple metastases to the liver, lungs, and often to the brain. Sometimes metastases can be detected in the clinic earlier than the main tumor. Especially with its retroperitoneal localization and localization in the large intestine.

Rhabdomyoma is a mature, benign tumor of striated muscles. It is rare. Described in all age groups, more often in children and newborns. It is localized on the head, neck, trunk, upper and lower limbs. Separate rhabdomyomas of the tongue, heart, and female genital organs.

Macroscopicallycan have the form of a node and an infiltrate.

Microscopicallytumor cells copy different degrees of differentiation muscle elements of different shapes - large oval, striated. Transverse striation is difficult to detect, mainly in elongated striatal cells. Glycogen is detected in the cytoplasm of cells. Figures of mitosis are absent.

Clinicallyare benign, with the exception of rhabdomyomas of the heart and tongue, which are the cause of death of patients.

Rhabdomyosarcoma- an immature, malignant tumor of striated muscles. It occurs more often than rhabdomyomas. In children, rhabdomyosarcoma is one of the most common tumors, second only to nephroblastoma (Wilms tumor) and neuroblastoma. It is localized in the thickness of the muscles of the lower, less often - the upper limbs, in the retroperitoneal tissue, mediastinum, on the face, neck, in the nasopharynx, in the genitourinary organs.

Macroscopicallythe tumor is a node with a diameter of up to 20 cm or more.

Microscopicallya characteristic polymorphism due to the fact that tumor cells copy embryonic muscle cells in their structure at various stages of embryogenesis and differ in significant cataplasia. To make a diagnosis, methods are used to detect transverse striations in the cytoplasm of cells, electron microscopy to detect myofibrils, and immunohistochemical typing using monoclonal antibodies.

Rhabdomyosarcoma is characterized by a high degree of malignancy. It often recurs, gives multiple hematogenous metastases in the liver and lungs.

Tumors of blood and lymphatic vessels.

Hemangioma- a mature, benign tumor from blood vessels. Some of these tumors refer to malformations of the vascular system of a tumor-like nature, some to true blastomas. Depending on which vessels copy the neoplasm, the following types of hemangioma are distinguished: capillary; venous; cavernous; arterial

Capillary hemangioma is a neoplasm with the proliferation of endothelial cells and the formation of atypical capillaries. It is localized most often in the skin, mucous membranes of the gastrointestinal tract. It is often multiple. It is more common in female children.

Macroscopicallyit is represented by a red or bluish node with a smooth or bumpy surface, it has a porous structure on the cross section. If the tumor is localized in the skin, the node becomes white when pressed.

Microscopicallythe tumor consists of branched capillary-type vessels with a narrow lumen that is not always filled with blood. The endothelium is swollen,

hyperchromic. Capillaries can form indistinctly separated particles, which gives the impression of infiltrating growth.

Cavernous hemangioma – neoplasm, which consists of chimeric cavities of the type of sinusoids of different sizes, which are interconnected. It is most often found in the liver, gastrointestinal tract, brain.

Macroscopicallyhas the appearance of a purplish-bluish node clearly separated from the surrounding tissues, which resembles a sponge in cross-section.

Microscopicallyconsists of thin-walled caverns (cavities), covered with a single layer of endothelial cells and filled with blood.

Arterial angioma- is a conglomerate of developed vessels of the arterial type, among which there are areas resembling a capillary hemangioma.

Venous hemangioma- microscopically represented mostly by vessels of the venous type, next to which there are vessels of the capillary and atrial type. It is located in the depth of soft tissues, between muscles.

Glomusangioma(Barre-Masson tumor)– mature benign tumor of vascular origin (myoarterial glomus). They occur with the same frequency in men and women, mostly of mature age.

Macroscopicallytwo types are distinguished: solitary glomusangioma; multiple disseminated (familial glomusangioma).

It occurs more often in the form of a single nodule with a diameter of 0.3–0.8 cm, soft consistency, grayish-pink color. Favorite localization in the hands and feet, mostly on the fingers, in the area of the nail bed. It is clinically manifested by sharp pain due to a large number of nerve endings.

Microscopicallyconsists of slit-like vessels of the sinusoidal type, which are covered with endothelium and surrounded by cuffs of epithelioid cells and resemble glomus cells.

Hemangiopericytoma– a tumor of vascular origin, in which, along with the formation of vessels, the proliferation of perivascular cells (Zimmerman pericytes) occurs. It occurs at any age, often in children. As a rule, it has a benign course. It can recur after several years. With a certain localization, for example, in the retroperitoneal region, on the upper limbs, head and neck, regardless of the maturity of the cells that make up the tumor, it can metastasize. Therefore, Stout and other authors suggest considering different variants of hemangiopericytoma as "potentially malignant tumors".

Lymphoangioma- a tumor from lymphatic vessels. It occurs more often in children as a developmental defect. It is localized mostly in the mucous membrane of the oral cavity, retroperitoneal space, mesentery. Cystic and cavernous variants of the tumor structure are more common. The microscopic structure is similar to the structure of hemangiomas.

Hemangioendothelioma- considered by many authors as the most malignant tumor. It occurs more often at the age of 30–50 years, but can often occur in childhood. It is localized most often in the skin, soft tissues of the limbs, trunk, head, less often in internal organs.

Macroscopicallyis a node up to 10 cm in diameter, lobular structure, in places with infiltrative growth. The nodules are soft, juicy, pink or red in color with centers of necrosis.

Microscopicallythe tumor is built of atypical, randomly anastomosing vessels lined with several layers of atypical endothelial cells. Pronounced cellular polymorphism, nuclear hyperchromia. Hemangioendothelioma metastasizes widely, mostly by the hematogenous route to the lungs, bones, and liver. Metastases to regional lymph nodes can be observed.

Lymphangioendothelioma- similar in structure to hemangioendothelioma. It often occurs against the background of chronic lymphostasis.

Tumors of synovial tissue.

*Synoviomas*they occur more often at the age of 30-40, mainly in men.

Macroscopically, it looks like a dense nodule measuring 5 cm or more, uniform in section, white-pink in color. It is localized on the limbs in the area of the joints (the area of the knee, forearm, fingers and toes).

Microscopically, the tumor is polymorphic, it contains fissures and cysts of various sizes, lined with oval, cubic, prismatic cells that resemble cells of the glandular epithelium. In addition, there are spindle-shaped cells that form the swollen stroma. They are also polymorphic. There are single giant multinucleated cells. Since the morphological and biological features of synoviomas often do not coincide, and a morphologically mature tumor may turn out to be malignant, today most authors believe that all synoviomas should be considered malignant, regardless of the degree of maturity.

Tumors from the mesothelium.

Mesothelioma- a mature, benign tumor, occurs relatively rarely, has the structure of a fibroma, rich in cellular elements, therefore it is called fibrous mesothelioma.

Macroscopically, it is a clearly separated nodule, which grows slowly, most often in the visceral pleura, dense, layered in section.

*Malignant mesothelioma*is a rare neoplasm that develops from mesothelial cells, mainly in the pleura, but can also be observed in the peritoneumand pericardium. Almost all patients with malignant mesothelioma have a history of working with asbestos.

Macroscopicallythe tumor has the form of a dense infiltrate, 2-3 cm thick or more, on the serous membranes. In the pericardium and omentum, it may have the appearance of vaguely separated nodes with a villous surface.

Microscopicallythe tumor resembles adenocarcinoma or hemangioendothelioma. Epithelioid mesothelioma of tubular or papillary structure is most common.

Histological verification of both mature and immature mesothelioma is very difficult. An accurate diagnosis can be made with the help of immunohistochemical typing using monoclonal antibodies, as well as the tissue culture method.

Cyst-forming and cartilage-forming tumors.

Chondroma – a mature benign tumor that mimics the morphology of mature hyaline cartilage. It is more often localized in the phalanges of the fingers of the hand,

bones of the wrist, but can also be found in large tubular bones (thigh, shoulder, tibia) and in the lungs. It occurs in all age groups, but more often in children. Clinically, it grows slowly over the years.

Macroscopicallychondroma is a node of lobular structure, dense, blue and white in color, resembling cartilage.

Microscopicallythe tumor has the structure of mature hyaline cartilage. Cellular atypism is weakly expressed. Cartilaginous cells vary slightly in size, with one, or sometimes two, small nuclei, located randomly in typical lacunae, separated from each other by a greater or lesser amount of the main substance of the hyaline type. The significance of the tumor is determined by its location. For example, when located in the bronchi, it can cause atelectasis of the lung.

Osteoma– mature, benign bone tumor. The predominant localization of osteomas is the bones of the skull, especially the sinuses. Osteoma in tubular bones is rare. Most often, it appears in childhood.

Macroscopicallyhas the appearance of a node, in consistency is denser than normal tissue. In the accessory sinuses of the skull, they are sometimes multiple, growing in the form of a polyp on a stem. In relation to the bone, the osteoma can be periosteal, cortical or endosteal. In most cases, osteomas are diagnosed accidentally during an X-ray examination.

Microscopicallyosteomas are divided into compact and spongy. Compact osteoma consists almost entirely of bone mass, thin fibrous or lamellar structure with very narrow vascular channels. A spongy osteoma is represented by a clear mesh of bone beams, but arranged randomly. Interbeam spaces are filled with cellular and fibrous tissue. It does not have clear boundaries with the surrounding bone tissue. The combination of multiple osteomas localized in the lower jaw, skull vault and long tubular bones with intestinal polyposis and soft tissue tumors was named Gardner's syndrome.

*Osteosarcoma*is a collective concept that includes immature malignant tumors of bone and cartilage-forming tissue, such as periosteal chondrosarcoma, peri- and intracortical osteogenic sarcoma, malignant osteoblastoma. It is necessary to know that an X-ray examination is mandatory for the verification of osteogenic tumors. Thus, the diagnosis is X-ray morphological. The age of patients varies from 6 to 60 years, 50% are patients younger than 30 years. Radiologically, thinning and destruction of the cortical bone layer is noted.

Macroscopicallytumor of a variegated appearance - from white-gray to brown-red color, loose consistency, despite the presence of focal calcification.

Microscopicallythe main tissue component of the tumor is represented by bone and osteoid structures lined with atypical osteoblasts, with the presence of thin-walled vessels, many atypical figures of mitosis are found. Metastasis is carried out mainly by the hematogenous route, mainly in the lungs.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Definition of a tumor as a pathological process.
2. Theories of oncogenesis, factors contributing to the development of tumors.

3. Types of tumor growth.
4. Tumor structure, tumor progression, tumor heterogeneity. Concept of atypism (atypia), types of atypism, morphological atypism (morphological characteristics of tissue and cellular atypism).
5. The effect of the tumor on the body of the host. Secondary changes of the tumor.
6. The concept of metastasis, the path of metastasis, the difference between metastases and a primary tumor.
7. Classification of tumors formed from mesenchyme.
7.1. Connective tissue tumors, morphological characteristics.
7.2. Tumors from adipose tissue (white and brown tissue, morphological characteristics.
7.3. Bone tissue tumors, morphological characteristics.
7.4. Tumors from cartilaginous tissue, morphological characteristics.
7.5. Tumors of muscle tissue, characteristics.
7.6. Tumors from tissue vessels, morphological characteristics.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

1. A 40-year-old man was found to have a tumor-like mass measuring 8x7 cm on his neck, which the surgeon removed incompletely due to its intimate connection with large vessels. Microscopically, it has pronounced tissueand cellular atypism, lipoblast-type cells of various degrees of maturity, with polymorphism, hyperchromia of nuclei, pathological mitoses, and foci of necrosis. Determine the histological form of the tumor:

AND Liposarcoma
Lipoma
CFibroma
DFibrosarcoma
Hibernoma

2. A 57-year-old woman underwent an operation to extirpate the uterus with appendages. Macroscopically: in the thickness of the myometrium, a nodule of dense consistency, rounded shape with clear contours, 5x6 cm in size, on a section of the fibrous structure. Histologically, it is represented by randomly arranged bundles of smooth muscle cells and excessively developed stroma. What is the most likely diagnosis?

AND Fibromyoma
Rhabdomyosarcoma
Rhabdomyoma
DFibroma
ELeiomyosarcoma

3. During an ultrasound scan, a retroperitoneal tumor was detected in a young woman, which has the shape of a node up to 25 cm in diameter. When examined microscopically, the tumor consists of spindle-shaped cells that form bundles going in different directions. With special research methods, myofibrils are detected in the cytoplasm. Cells differ in pronounced polymorphism, there are giant cells and many atypical figures of mitosis. Your diagnosis:
AND Rhabdomyosarcoma
BLeiomyoma
C Leiomyosarcoma
Rhabdomyoma
Liposarcoma
4. A 38-year-old man was diagnosed with a large tumor in the retroperitoneal space. Macroscopically, it had the form of a conglomerate of nodes with infiltration of surrounding tissues. The consistency is dense, the cut surface is white, juicy, reminiscent of "fish meat", variegated in places - with foci of oozing, hemorrhages and necrosis. Microscopically, tissue polymorphism is sharply expressed. It consists of lipoblasts of various degrees of maturity, there are giant cells with chimeric nuclei. Your diagnosis:
AND Liposarcoma
BMalignant hibernoma
Lipoma
Hibernoma
EFibroma
5. During computer tomography, a tumor was found in the retroperitoneal space of a young woman, which was surgically removed. Macroscopically, it looked like a nodule with dimensions of 20x15x13 cm, lobular structure (due to the large number of connective tissue layers), soft-elastic consistency of yellow color, resembling adipose tissue in appearance. When examined microscopically, the tumor is built according to the type of adipose tissue, and the sizes of particles and fat cells vary widely. Your diagnosis:
AND Lipoma
BDesmoid
CMalignant hibernoma
Hibernoma
Liposarcoma
6. A dense, mobile tumor clearly separated from the surrounding tissues was found in the skin. In section, it is white in color, represented by a fibrous fabric. Microscopically: chaotically intertwined collagen fibers, few cells. What kind of tumor is this?:
AND Fibroma
B Dermatofibroma.
CDesmoid
DMyoma

EHistiocytoma
7. Histological examination of the skin neoplasm revealed: the parenchyma is formed from the covering epithelium with an increased number of layers. Stroma together with epithelial growths form papillae. Specify the type of atypism?:
AND Fabric
B Metabolic
C Histochemical
D Cellular
Functional
8. The uterus removed for surgery was delivered for histological examination. Under the mucous membrane, numerous rounded nodes are identified, which are clearly separated from the surrounding tissue. Microscopically, the tumor is composed of bundles of smooth muscles with tissue atypism. Your diagnosis?:
AND Myoma
BFibromyoma
C Leiomyosarcoma
Chorionepithelioma
Uterine cancer
9. Upon microscopic examination of the tumor of the upper lip, it was found that it is made of numerous slit-like cavities, the wall of which is lined with flattened endothelium, filled with liquid blood and clots. What is the diagnosis?:
AND Cavernous hemangioma
BVenous hemangioma
Capillary hemangioma
DHemangiopericytoma
Glomus-Angioma.
10. In a 17-year-old patient, intraoperatively, a tumor measuring 4.5x5.0x3.5 cm was found on the lower surface of the liver. It was subserosal, dark red in color, and on section was represented by cavities with significant blood content. Make a preliminary diagnosis?:
AND Cavernous hemangioma
B Lymphangioma.
Capillary hemangioma
DHemangiopericytoma
EHemangioendothelioma
11. A dense, mobile tumor was macroscopically found in the thickness of the skin. Under microscopy, it is represented by randomly arranged bundles of collagen fibers with a small number of spindle-shaped cells. What is this tumor?:
AND dense fibroma
B Lipoma
C Glomus-Angioma.
DMelanoma
EMyoma

12. A 6-month-old child has a flat red nodule on the skin of the neck, the nodule turns pale when pressed with a glass. What is the most likely diagnosis?:
AND Hemangioma
B Lymphangioma
Melanoma
DPigmented nevus
ELeiomyoma

3.2. Algorithm of description of macropreparation and micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the organ(length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the

	subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Tumors from the epithelium: benign organ-nonspecific epithelial tumors, cancer (features of development, metastasis, histological forms)".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine

2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 14

Topic: Epithelial tumors: benign organ-nonspecific epithelial tumors, cancer (features of development, metastasis, histological forms).

Goal: To study the pathomorphology of tumors from the epithelium: benign organ-nonspecific epithelial tumors, cancer (learn the features of development, metastasis, characteristics of various histological forms).

Basic concepts: Papilloma (solid, soft), adenoma (cystadenoma, trabecular, acinar, trabecular, papillary adenoma), cancer (adenocarcinoma, signet ring cell carcinoma, solid cancer, mucosal carcinoma, scirr, medullary carcinoma, squamous cell carcinoma.)

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:

*emphasize the definition or provide an explanation.*Papilloma (solid, soft), adenoma (cystadenoma, trabecular, acinar, trabecular, papillary adenoma), cancer (adenocarcinoma, signet ring cell carcinoma, solid cancer, mucosal carcinoma, scirr, medullary carcinoma, squamous cell carcinoma.)

2.2. Flow chart on the topic as a list of didactic units of the topic.

Epithelial tumors are the most common among tumors. The basis of their classification is the features of histogenesis (type of epithelium), degree of differentiation and organ specificity.

Depending on the histogenesis, tumors of the covering epithelium (multilayered flat and transitional) and glandular are distinguished.

According to the course, which is mainly determined by the degree of differentiation, epithelial tumors can be benign or malignant.

Depending on the organ specificity, organ-specific and epithelial tumors without specific localization are distinguished.

Organ-specific tumors arise only in certain organs and have characteristic morphological features, sometimes functional (synthesis of characteristic hormones), which distinguish the tumor from other tumors and easily allow (even in the presence of metastases) to establish its origin from a specific organ.

Benign tumors without characteristic localization

*Papilloma.*Benign tumor of the integumentary epithelium (multilayered flat and transitional). It often occurs in the skin, oral cavity, bladder, etc. Macroscopically, it has a spherical shape on a wide base or on a leg, soft or elastic consistency, mobile.

Microscopically, the tumor is a papillary formation of multilayered flat or transitional epithelium, which is separated by the basement membrane from the connective tissue stroma with vessels. The polarity and complexity of the epithelium is preserved, but there is a thickening of the layers, increased keratinization.

Papilloma can be multiple - papillomatosis of the larynx.

Occasionally, the papilloma recurs and becomes malignant (larynx, bladder).

*Adenoma.*Benign tumor of the glandular epithelium. It develops on mucous membranes covered with glandular epithelium and in organs. Adenomas of mucous membranes in the form of a polyp are called adenomatous polyps. Adenomas of the mucous membrane of the stomach and colon often become malignant. The following morphological variants of adenoma are distinguished: acinar (alveolar), tubular, trabecular, solid, papillary cystadenoma, villous adenoma, fibroadenoma.

Papillary cystadenoma. Macroscopically, the tumor has the appearance of a cystic formation (it can be much larger than the ovary) with thin walls and a transparent liquid. The inner surface of the cyst (cyst) is covered with multiple white-pink papillae. The tumor is located within the ovary.

Microscopically, the tumor consists of cystically stretched lumens of the glands. Cubic or cylindrical epithelium, which lines the inner surface of cysts, forms papillary protrusions, preserves the basement membrane, polarity and complexity. In the ovaries, the tumor has a propensity for malignization, infiltrative growth and malignant course.

Benign tumors with characteristic localization.

*Villous adenoma of the colon.*Macroscopically, it looks like a large polyp (more than 1 cm) on a stalk or a wide base with a villous surface.

Microscopically, the adenoma is formed by elongated numerous papillae, which are formed by a highly differentiated epithelium with a large number of goblet cells. Dysplasia is often noted, multilineage of the epithelium is determined, atypia appears, and goblet cells disappear. In 30% of cases, the tumor becomes malignant.

*Breast fibroadenoma.*Benign tumor, often found in women aged 25-35. In the case of pregnancy, the tumor increases (it has progesterone receptors), regresses with

age. It rarely becomes malignant, carcinoma in situ is diagnosed in 0.1% of cases. Macroscopically - a dense, mobile, well-demarcated, painless node, usually up to 3 cm with slit-like cavities on the section. Sometimes it reaches large sizes - a giant fibroadenoma.

Microscopically, the tumor consists of glandular structures (ducts) of various shapes and sizes. The epithelium preserves the basement membrane, complexity and polarity. The stroma is well developed and dominates the parenchyma. Intracanalicular fibroadenoma is distinguished - the stroma is loose, rich in cells, grows into the ducts, compressing them, and pericanalicular fibroadenoma - the fibrous stroma surrounds the ducts and, as a result, they have the appearance of round tubes. Both variants are often found in the tumor.

*Phylloid (leaf-shaped)*the tumor belongs to stromal tumors and can be benign, borderline and malignant.

Adenomas of endocrine organs.

They are characterized by pronounced organ specificity. They can be hormonally active and manifest as a specific hormonal syndrome or without hormonal activity. Tumors that arise from endocrine cells that belong to the APUD system (amine precursor uptake decarboxylation) and produce biogenic amines or polypeptide hormones are called apudomas.

Apudoms are diverse and are named according to the hormones they produce. Apudomas include adenomas of endocrine glands (pituitary gland, pineal gland, pancreas), paragangliomas (chromaffinous and non-chromaffinous (hemodectoma)), carcinoid. Apudomas have a malignant course, the probability of malignancy increases with the growth of the tumor, so they are classified as potentially malignant.

*Carcinoid.*Traditionally, the term is applied to tumors that arise from enterochromaffin cells of the gastrointestinal tract and produce serotonin (a biogenic amine). Tumors of other locations (lungs, pancreas, etc.) are also called carcinoids. It is most often found in the appendix and small intestine (30%). It can be accompanied by carcinoid syndrome: reddening of the skin, watery diarrhea, bronchospasm, non-infectious thromboendocarditis of the valves of the right half of the heart. Macroscopically, the tumor without clear boundaries is up to 1 cm, it can be larger. It is yellow on section, located in the submucosal layer, and occasionally ulcerates.

Microscopically, the tumor consists of polygonal cells that are located around capillaries and are delimited by groups of connective tissue layers. The cells have a positive argentophine reaction. Occasionally, a carcinoid becomes malignant and can metastasize.

Pituitary adenomas.

*Somatotropic adenoma*consists of eosinophilic cells, produces somatotropin (growth hormone). Gigantism develops in children, and acromegaly in adults (enlargement of arms, legs, jaws, nose, and internal organs; accompanied by hyperglycemia, osteoporosis, and hypertension).

*Corticotropic adenoma*consists mainly of basophilic cells, produces adrenocorticotropic hormone (ACTH). Causes the development of Itsenko-Cushing's disease, which is accompanied by hypercorticism.

*Prolactinoma*consists mainly of chromophobe cells, causes amenorrhea and galactorrhea in women, impotence and sometimes galactorrhea in men.

Adenomas of the pancreasarise from islet cells.

*Insuloma*develops from beta cells, produces insulin, has a trabecular or tubular structure, is accompanied by hypoglycemic syndrome.

*Glucagonoma*develops from A-cells, produces glucagon, has a trabecular structure, causes hyperglycemic states and secondary diabetes.

*Gastrinoma*develops from G cells, produces gastritis (causes hyperplasia of parietal cells of the gastric mucosa and stimulation of hydrochloric acid production, has a trabecular structure, is accompanied by Zollinger-Ellison syndrome, which is characterized by multiple recurrent ulcers of the stomach and duodenum. It has a malignant course in 70% of cases .

*Note*develops from D cells, produces vasoactive intestinal peptide, has a solid-trabecular structure, develops watery diarrhea, hypoglycemia and achlorhydria (pancreatic cholera or Werner-Morrison syndrome. Malignant course in 80% of cases.

Pheochromocytoma(chromaffin paraganglioma) arises from chromaffin cells of the adrenal medulla, if the tumor arises from extra-adrenal chromaffin tissue, it is called a paraganglioma. The tumor produces adrenaline and norepinephrine, causes secondary hypertension, and in 10% of cases it becomes malignant.

Multiple endocrine neoplasia syndrome (MEN)- a number of genetic syndromes, which are accompanied by the development of multiple endocrine tumors, mainly apud.

Malignant epithelial tumorscalled cancer or carcinoma. Diagnosed most often among tumors. Usually associated with precancerous conditions, previous changes in the epithelium: metaplasia, dysplasia, hyperplasia. Epithelial dysplasia progresses from mild to moderate to severe, causing carcinoma in situ and subsequently invasive cancer that grows into surrounding tissues. Carcinoma in situ is an intraepithelial tumor that does not extend beyond the basement membrane. As for the cervix, in many cases it is not possible to distinguish severe dysplasia from carcinoma in situ, so these conditions were combined under the name CIN 3 (cervical intraepithelial neoplasia 3) and a single treatment strategy was chosen. Carcinoma in situ does not metastasize.

Cancer metastasizes mainly lymphogenously, the first metastases occur in regional lymph nodes, later hematogenous and implantation metastases may occur.

Squamous cell cancer. It develops from a multi-layered flattened epithelium. It occurs in the lungs as a result of metaplasia of the bronchial epithelium. There may be different degrees of differentiation. The formation of "cancer pearls" is characteristic of highly differentiated cancer, keratin is absent in poorly differentiated cancer, and keratin is determined intracellularly in a moderately differentiated form.

*Adenocarcinoma (glandular cancer).*It develops from the prismatic epithelium of mucous membranes and organs, the presence of glands is characteristic. It has a

different degree of differentiation. The lower the degree of differentiation, the fewer glands are determined in the tumor. A special form of low-differentiated adenocarcinoma is a sclerotic adenocarcinoma with an abundant stroma and a nested cluster of hyperchromic cells with a pronounced atypism. The consistency of the tumor is cartilaginous.

Undifferentiated cancer(it is impossible to determine from which epithelium the tumor originates without special diagnostic methods). Small cell cancer is found in the stomach, lungs (hormonally active, so it can be attributed to apudom) and other organs. Large cell carcinoma occurs in the stomach and lungs. Signet cell carcinoma is most often found in the stomach. Medullary cancer is most common in the breast. The stroma is sparse, the atypical cells are large, the nuclei are well-defined nucleoli, the cell boundaries are not defined, there are necrosis. Atypical mitoses are defined among numerous mitoses. The tumor reaches large sizes, soft, white-pink color on the section, the surface of the tumor is smooth. Undifferentiated cancer with a scirrhous type of growth occurs mainly in the stomach.

Cancer with specific localization in organs. An example of cancer with pronounced organ specificity can be clear cell kidney cancer and chorionic carcinoma.

Clear cell kidney cancer. The most common form of renal cell carcinoma, which develops from the tubular epithelium. Men aged 40-60 years are more often affected. It metastasizes hematogenously, the first metastases are determined in the lungs. It is characteristic that the tumor grows into the renal vein and spreads along the vena cava to the heart. The tumor has the appearance of a node with clear borders, which are formed by a pseudocapsule. On section, the tumor is variegated, yellow with hemorrhages. Microscopically, the tumor is made up of atypical cells with small hyperchromic nuclei and optically empty (light) cytoplasm, which form solid-alveolar structures, has many sinusoidal vessels, hemorrhages. When staining with sudan 3, lipids in the cytoplasm of cells are determined.

Chorion carcinoma. A malignant tumor that develops from a trophoblast. Occurs in women after childbirth, abortions, against the background of destructive vesicular snow. It is localized in the uterus, but an ectopic location outside the uterus is possible and vision develops in men. The tumor metastasizes hematogenously in the lungs, liver, brain, etc. Hemorrhages occur in metastases, which explains hemoptysis when the tumor is located in the lungs. The tumor has the appearance of a soft nodule of dark red color. The tumor consists of atypical small cytotrophoblast cells and large syncytial formations (Langhans cells), the stroma is not determined, there are many hemorrhages. The tumor is hormonally active, produces chorionic gonadotropin (HCG), an increase in its level in urine and blood is a diagnostic criterion. Immunohistochemically, HCG can be detected in tumor cells.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Histogenetic (ontogenetic) classification of tumors, classification by level of differentiation, morphological characteristics.

2. Classification of epithelial tumors. Rules for naming epithelial tumors (nomenclature).
3. Benign epithelial tumors from the covering epithelium - papillomas (classification, morphological characteristics)
4. Benign epithelial tumors of the glandular epithelium - adenomas (classification, morphological characteristics).
5. Malignant epithelial tumors from the covering epithelium - squamous cell carcinomas (classification, morphological characteristics)
6. Malignant epithelial tumors from the glandular epithelium - adenocarcinomas (classification, morphological characteristics of different types).
7. Tumors of individual organs.
3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs with subsequent sketching of micropreparations and opisom of the macropreparation in the album).
3.1 test tasks
1. A 57-year-old patient developed periodic uterine bleeding. For diagnostic purposes, the uterus was scraped. In the obtained material, among the elements of the blood, glandular components are observedleks of different sizes and shapes, formed by atypical cells with hyperchromic nuclei with numerous mitoses (including pathological ones). What is the most likely diagnosis?
AND Adenocarcinoma
B Ffibromyoma of the uterus
C Choirionepithelioma
Glandular hyperplasia of the endometrium
Endometritis
2. During the microscopic examination of the surgical material (part of the lip with an ulcer), near the edges and under the bottom of the ulcer defect in the connective tissue of the mucous membrane, epithelial complexes of atypical multilayered epithelium with figures of pathological mitosis were found. In the center of the complexes is an accumulation of bright pink concentric formations. What pathology developed?
AND Squamous cell carcinoma with keratinization
BSquamous cell carcinoma without keratinization
Transitional cell cancer
Basal cell cancer
EPapilloma
3. Microscopic examination of a colon biopsy revealed a tumor of prismatic epithelium forming atypical glandular structures of various shapes and sizes. The basal membrane of the glands is destroyed. The tumor cells are polymorphic, the nuclei are hyperchromic, and a large number of pathological mitoses are noted. What is the most likely diagnosis?
AND Adenocarcinoma
B Basal cell cancer

Solid cancer
D. Mucosal cancer
Undifferentiated cancer
4. During the examination, a tumor was found on the hard palate of the patient in the form of a small dense nodule of gray color without clear boundaries. After removal, the tumor was examined histologically. It is made of small, cubic-shaped cells with a hyperchromic nucleus, which form alveoli, trabeculae, solid and cribrose structures. Tumor growth is invasive. Name the tumor:
AND Adenocystic carcinoma
BMalignant pleomorphic adenoma
Mucoepidermoid cancer
Adenolymphoma
Monomorphic adenoma
5. In a 69-year-old patient, a small plaque-like formation appeared on the skin in the area of the lower eyelid, with subsequent ulceration, which was surgically removed. Upon microscopic examination of the formation: in the dermis of the skin there are complexes of atypical epithelial cells, on the periphery of the formations the cells are located perpendicular to the basement membrane. Cells are dark, prismatic polygonal; nuclei are hyperchromic with frequent mitoses. Sometimes there are formations similar to a hair follicle. What is the histological form of cancer in the patient?
AND Basal cellular
B Flat-celled with cornification
Flat-celled without cornification
Adenocarcinoma
Undifferentiated
6. A 46-year-old woman had a tumor removed from the right parotid area, which had gradually increased over the course of 5 years. Macroscopically: an encapsulated nodule with a diameter of 60 mm, elastic consistency, whitish-gray tissue with multiple small cysts containing mucus. Microscopically: ductal structures or cells of a solid structure consisting of monomorphic polygonal and cubic cells, areas of myxoid and chondroid matter are located between them. Diagnose the tumor:
AND Pleomorphic adenoma
BMonomorphic adenoma
CMucoepidermal tumor
Adenolymphoma
Adenocystic carcinoma
7. During histological examination of a micropreparation of a malignant lung tumor, it was found that it consists of lymphocyte-like cells that do not form any structures. The stroma is poorly expressed, many mitoses and necrosis are observed. What kind of tumor is this?
AND Small cell cancer
B Fibroma
Squamous cell non-keratinous cancer

D. Squamous cell keratinous cancer
Adenocarcinoma
8. During the histological examination of the wall of the cyst localized in the area of the upper jaw, it was established that the wall of the cyst is lined from the inside with a multilayered flat epithelium with underlying granulation tissue with lymphocytic infiltration. The outer layer is represented by loose fibrous connective tissue surrounded by fibrous scar tissue. These data are the basis for establishing such a diagnosis:
AND Cystogranuloma
BSimple granuloma
Epithelial granuloma
D Keratocyst
Ameloblastoma
9. A biopsy from the right main bronchus of a 63-year-old man, a smoker, revealed a tumor consisting of groups of atypical epithelial cells that penetrate beyond the boundaries of the basal membrane of the mucous layer, forming "nests" and strands, in the central parts of which concentric, bright - eosinophilic masses - "cancer pearls". Diagnose the disease:
APloskokl squamous cell carcinoma
B Flat glassretinal keratin papilloma
C Flat glassepithelial nonkeratinous cancer
D Adenocarcinoma
E DrIbnocellular cancer
10. Microscopic examination of a colon biopsy revealed a tumor of prismatic epithelium forming atypical glandular structures of various shapes and sizes. The basal membrane of the glands is destroyed. The tumor cells are polymorphic, the nuclei are hyperchromic, and a large number of pathological mitoses are noted. What is the most likely diagnosis?
AND Adenocarcinoma
B Basal cell cancer
Solid cancer
D. Mucosal cancer
Undifferentiated cancer
11. At the autopsy of the deceased, a 46-year-old man with a history of viral hepatitis C, a liver tumor was found in the form of a nodule, the tumor tissue was green in section. Microscopically: the tumor is made up of atypical hepatocytes forming acini. The stroma is poor, with thin-walled blood vessels. Your diagnosis?:
AND Hepatocellular carcinoma
BHepatoadenoma
Sarcoma of the liver
Liver angioma
Steatohepatosis

12. During the examination of the kidney removed during the operation, a tumor was found in the form of a soft variegated nodule. Microscopically, the tumor consists of tubular and papillary structures, its cells are atypical with hyperchromic nuclei. Diagnose a tumor?:
AND Renal adenocarcinoma
BHypernephroid cancer
Dark cell adenoma
DNephroblastoma
Clear cell cancer

3.2. Algorithm of description of macropreparation and micropreparation

Description of macropreparation:

1. Specify the name of the body of the ababout the ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

10. Specify the name of the body;
11. Specify the color;
12. Specify what changes in cells;
13. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical

	skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Nomenclature and morphological features of tumors of nervous tissue. Features of tumors of the central nervous system. Nomenclature and morphological features of tumors originating from melanin-producing tissue".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 15

Topic: Nomenclature and morphological features of tumors of nervous tissue. Features of tumors of the central nervous system.

Goal: Learn and be able to freely apply nnomenclature and morphological features of nerve tissue tumors. Know the features of the structure and growth of tumors of the central nervous system.

Basic concepts: Nevus (intradermal nevus, border nevus, mixed nevus, blue nevus, juvenile nevus), glioma (astrocytoma, oligodendroglioma, ependymoma), meningioma, schwannoma, ganglioma, glioblastoma;

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic: emphasize the definition or provide an explanation. Nevus (intradermal nevus, border nevus, mixed nevus, blue nevus, juvenile nevus), glioma (astrocytoma, oligodendroglioma, ependymoma), meningioma, schwannoma, ganglioma, glioblastoma;

2.2. Flow chart on the topic as a list of didactic units of the topic.

Tumors of the nervous system differ from tumors of other origin in a wide variety, because they arise from different elements of the nervous system: central, autonomic, peripheral, as well as elements of the mesenchymal system, which are part of the nervous system. According to the degree of differentiation, they can be more or less mature, that is, benign and malignant. However, when localized in the brain or

spinal cord, they always turn out to be malignant, because even with slow growth, they exert pressure on vital centers and cause disruption of their functions

Tumors of the nervous system are divided into:

Tumors of the nervous system and meninges.

Output cell	Benign tumors	Malignant tumors
Tumors of central nervous tissue Neuroectodermal tumors *Astrocytic tumors*		
Astrocyte, astroblast	Astrocytoma	Astroblastoma
Oligodendroglial tumors		
Oligodendrogliocyte oligodendroglioblast	Oligodendroglioma	Oligodendroglioblastoma
Ependymal tumors and tumors of the choroid epithelium		
Ependymocyte, Ependymoblast, Choroidepithelium	Ependymoma Choroid papilloma	Ependymoblastoma Choroid carcinoma
Neuronal tumors		
Ganglioneurocyte Ganglioneuroblast	Ganglioneuroma (gangliocytoma)	Ganglioneuroblastoma Neuroblastoma
Low differentiation and embryonal tumors		
Medulloblast Glioblast		MedulloblastomaGlioblastoma
Meningovascular tumors		
Meningothelioma	Meningioma	Meningeal sarcoma
Tumors of the autonomic nervous system		

Sympathogonia Ganglioneuroblast Ganglioneurocyte Cells of non-chromaffin paraganglia	Ganglioneuroma Two-grade non-chromaffin paraganglioma (glomus tumor, chemodectoma)	Sympathoblastoma (sympathogonioma) Ganglioneuroblastoma Malignant non-chromaffin paraganglioma (hemodectoma)
Tumors of the peripheral nervous system		
Lemocyte (the so-called Schwann cell)	Neurolemoma (schwannoma), Neurofibromatosis (Recklinghausen's disease)	Malignant neurilema (neurogenic sarcoma)

Neuroectodermal(neuroepithelial) tumors of the brain and spinal cord are built from neuroectoderm derivatives. More often than tumors of other organs, they have a dysontogenetic origin, that is, they develop from the remnants of precursor cells of mature elements of the central nervous system, so their histological affiliation is sometimes difficult to establish. The cellular composition of these tumors corresponds to certain phases of development of neuronal and glial elements of the nervous system. Neuroectodermal tumors include: astrocytic, oligodendroglial, ependymal and choroidal epithelium tumors; neuronal, poorly differentiated and embryonic. Malignant neuroectodermal tumors metastasize, as a rule, within the cranial cavity and very rarely - in internal organs.

Astrocytic tumors(gliomas) are divided into benign - astrocytoma and malignant - astroblastoma (malignant astrocytoma).

Astrocytoma– the most common benign neuroectodermal tumor that develops from astrocytes; observed at a young age, sometimes in children; is localized in all parts of the brain. The size of the tumor reaches 5–10 cm in diameter; it is not always clearly separated from the adjacent tissue; on autopsy it has a homogeneous appearance, sometimes cysts are found in the tumor; the tumor grows slowly, there are few vessels in it.

Depending on the histological structure of the tumor, three types of astrocytes are distinguished: fibrillar, protoplasmic and fibrillar-protoplasmic (mixed). Fibrillary astrocytoma is rich in glial fibers arranged in the form of parallel bundles; there are few astrocyte-type cells in the tumor. Protoplasmic astrocytoma is made up of astrocyte-like cells of various sizes and shapes with processes that intertwine to form a

dense mesh. Fibrillary-protoplasmic (mixed) astrocytoma is characterized by an even arrangement of astrocytes and glial cells with appendages; is rare.

Astroblastoma(malignant astrocytoma) differs in cellular polymorphism, possible necrosis and hemorrhage; metastasizes through the cerebrospinal fluid, grows quickly, is rare.

Oligodendroglial tumorscan be both benign - oligodendroglioma, and malignant - oligodendroglioblastoma.

Oligodendroglioma- a mature tumor, more common in women aged 30-40 years. It is localized mostly in the frontal and temporal regions, as well as in subcortical nodes. Oligodendrolioma has the appearance of a cell of homogeneous grayish-white tissue. It is made of small cells with small hyperchromic round nuclei that seem to "hang" in the light cytoplasm, the development of small cysts filled with mucous masses is possible. The tumor is highly vascularized by capillary pressure vessels. Oligodendroglioblastoma (malignant oligodendroglioma) is characterized by both tissue and cellular atypism, pathological mitoses; necroses and hemorrhages occur quite often.

Ependymal tumors and tumors of the choroid epithelium

Benign tumors of the specified origin include ependymoma and choroid papilloma, and malignant tumors include ependymoma and choroid carcinoma.

Ependymoma- a mature tumor, observed mostly in childhood and young age, is related to the ependyma of the ventricles of the brain by its origin. It has the appearance of an intra- or extraventricular nodule of gray color, quite often with foci of necrosis and cysts. Typical for this tumor are accumulations of uni- or bipolar round and oval ependymal cells that form pseudo-rosettes around vessels containing chromatin in the form of grains.

Ependymoblastoma- this is the largest tumor of the hemispheres in children, is a malignant variant of ependymoma (malignant ependymoma), differs from a benign tumor by sharply expressed cellular polymorphism, many mitoses and vascular reactions. It can resemble glioblastoma in adults, and medulloblastoma in children. The tumor grows rapidly, intensively penetrates into the adjacent tissues and. gives metastases in the subarachnoid space.

Choroid papilloma (choroid papilloma) is a mature tumor that develops from the epithelium of the vascular plexus of the ventricles of the brain; has the appearance of a villous node in the cavity of the ventricles of the brain; consists of numerous villous structures covered with a layer of epithelial cells similar to the epithelium of a normal vascular plexus.

Choroid carcinoma(malignant choroidal papilloma) externally has the appearance of a node located in the cavity of the ventricles, the tumor is connected to the vascular plexus. Built from anaplastic cells of vascular plexus (papillary cancer); is extremely rare.

Neuronal tumors

Cells of neural origin include ganglioneuroma (gangliocytoma), ganglioneuroblastoma (malignant gangliocytoma) and neuroblastoma. Ganglioneuroma (gangliocytoma) is a rare benign tumor that is localized in the region of the floor of the III ventricle; less often - in the cerebral hemispheres. The tumor is made of mature ganglion cells separated by bundles of glial stroma into areas of different sizes.

Ganglioneuroblastoma - malignant analogue of ganglioneuroma (malignant gangliocytoma) - an extremely rare tumor of the central nervous system. It is distinguished by sharply expressed cellular polymorphism; similar to malignant glioma.

Neuroblastoma – a rare highly malignant brain tumor; occurs in children. Built from large polygonal cells with a vesicular nucleus, numerous mitoses; cells form syncytial structures; there are many thin-walled vessels in the tumor.

Low-differentiated and embryonal tumors

Tumors of this origin include medulloblastoma and glioblastoma. Medulloblastoma is a tumor that is built from the most immature cells - medulloblasts, therefore it is characterized by particularly pronounced immaturity and malignancy; its most frequent localization is the cerebellar worm. The tumor occurs mostly in children, more often in boys aged 2–7 years.

Glioblastoma - an immature malignant tumor, the second most common brain tumor after astrocytoma. It occurs most often in people aged 40-60 years; is localized in the white matter of the brain. It has a soft consistency; on autopsy, it is shriveled due to the presence of necrosis and hemorrhages in it. Histologically, the tumor is made up of cells of different sizes, which differ in the shape of the nuclei and the content of chromatin in them. There is a lot of glycogen in the cells, many metoses. The tumor grows rapidly and can lead the patient to death within several months. Metastases are found only within the brain.

Meningovascular tumorsdevelop from the membranes of the brain, as well as from tissue close in structure to the membranes. The most common among them are: meningioma and meningeal sarcoma

Meningioma (arachnoidendothelioma) is a benign tumor from the cells of the medulla. In cases where a meningioma is made of arachnoidendothelial cells, i.e. cells that cover the spider web, it is called arachnoidendothelioma. The tumor has the appearance of a dense nodule connected to a hard, less often soft meninges and is made of endothelium-like cells that closely adhere to each other, forming nest-like structures. Often, cells form microconcentric structures (arachnoidendotedioma), where lime can accumulate, which leads to the formation of so-called psamoma bodies. A meningioma can be made up of spindle-shaped cells that form bundles and connective tissue fibers (fibrous arachnoidendothelioma).

Meningeal sarcoma- malignant analogue of meningioma.

On histological examination, it resembles fibrosarcoma, polymorphic cell sarcoma, or diffuse membrane sarcomatosis.

Tumors of the autonomic nervous systemdevelop from ganglion cells of different maturity (sympathogonia, sympathoblasts, ganglioneurocytes) of sympathetic ganglia, as well as from cells of non-chromaffin paraganglia (glomus), genetically related to the sympathetic nervous system. This group of tumors includes: benign - ganglioneuroma, benign non-chromaffin paraganglioma (glomus tumor, chemodectoma) and malignant - ganglioneuroblastoma, sympathoblastoma (sympathogonioma) and malignant non-chromaffin paraganglioma (hemodectoma). Some of the described tumors were previously discussed in other sections.

Benign non-chromaffin paraganglioma(hemodectoma) is morphologically similar to tumors of the ARUD system (apudomas), capable of synthesizing serotonin and, less often, ACTH. The tumor can reach significant sizes, especially. retroperitoneal In histological examination, the alveolar or trabecular structure is characteristic; it has a large number of sinusoidal vessels.

*Malignant non-chromaffin paraganglioma*is rare; is characterized by cellular polymorphism, infiltrating growth and lymphohematogenic metastases. Sympathoblastoma (sympathogonioma) is an extremely malignant tumor, found mostly in young children.

Tumors of the peripheral nervous system.

This type of tumor arises, as a rule, from nerve sheaths. They include: benign - neurillemmoma (schwannoma), neurofibroma, as well as neurofibromatosis (Recklinghausen's disease) and malignant - malignant schwannoma, or neurogenic sarcoma.

Neurolemmoma (schwannoma) is made of spindle-shaped cells with rod-shaped nuclei. The cells and fibers of the tumor form bundles that form rhythmic or "palisade" structures (nuclear palisades, Veroca's bodies) with sections that consist of fibers.

Neurofibroma – a tumor associated with nerve sheaths. Histologically, it consists of elements of connective tissue, nerve cells and fibers.

Neurofibromatosis(Recklinghausen's disease) is a systemic disease characterized by the development of multiple neurofibromas, which are often associated with various developmental defects. There are peripheral and central forms of neurofibromatosis.

Malignant neurilemmoma (neurogenous sarcoma) is a rare tumor characterized by pronounced cellular atypism and polymorphism, the presence of multinucleated symplasts and "palisade" structures.

2.3.List questions to check basic knowledge on the subject of the lesson.

1. Classification and nomenclature of tumors of the nervous system of various origins.

2. Morphology of mature and immature tumors of glial origin (astoglia, oligodendroglia, ependyma).

3. Morphology of mature and immature tumors of neural origin.

4. Morphology of meningovascular tumors.

5. Morphology of tumors of peripheral nerves and ganglia.

6. Definition and classification of tumors of melanocytic origin.

7. Nevi: definition, classification, morphological characteristics.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 Test tasks

1. In the region of the auditory nerve, a young woman was found to have a tumor in the form of a node up to 3 cm in diameter, soft-elastic, pink-white in color, homogeneous. Microscopically, the tumor is a bunch of cells with oval or curved nuclei. Fibro-cellular bundles tend to form rhythmic structures in the form of palisade structures, which are formed by parallel rows of correctly oriented cells located in the form of a palisade, between which there is a cell-free homogeneous zone. Such structures were called "Verokai's bodies". Your diagnosis?:

AND Neurinoma (schwannoma, neurilemoma)

BMalignant neurilema

Ganglioneuroma

Neuroblastoma

Ganglioneuroblastoma

2. In a young man, a tumor in the form of several nodes (multicentric growth) of a soft-elastic consistency, white in section with large foci of hemorrhages, mucinization, necrosis, and the presence of cystic cavities was found in the chest cavity. Microscopically - sharply expressed polymorphism. Cells with oval and elongated nuclei are characteristic, which are arranged randomly or form bundles in the form of palisades. There are multinucleated symplasts, individual xanthoma cells. Your diagnosis?:

AND Malignant neuroma

BNeurinoma (schwannoma, neurilemoma)

Ganglioneuroma

Neuroblastoma

Ganglioneuroblastoma

3. A 46-year-old man had a dark spot on his scalp that did not cause discomfort. Recently, the spot began to increase in size, pain appeared, the color became brown, black-brown. During histological examination of the removed tissue, spindle-shaped

and polymorphic cells are revealed, in the cytoplasm of which there is a brown-black pigment. Your diagnosis:
AND Melanoma
BHematoma
Hemangioma
Basalioma
ECarcinoid
4. In a 22-year-old man, a tumor was found in the adrenal glands, which has the appearance of a node, in places with infiltrative growth, soft consistency, yellowish color, with large areas of necrosis and hemorrhages. In addition, metastases were found in the liver (Pepper) and in the orbit of the eye (Hutchinson). Microscopically, the tumor consists of two types of cells. Some are small, like sympathogonia, round, with a narrow rim of cytoplasm and a small oval nucleus (bare nucleus), in which chromatin is arranged in the form of grains. Others, like sympathoblasts, are large, with a light nucleus and a large amount of cytoplasm. Tumor cells tend to form true and false rosettes in the form of a corolla of cells, in the center of which a delicate fibrillar substance is found. Your diagnosis?:
AND Neuroblastoma
BMalignant neuroma
Ganglioneuroma
DNeurinoma (schwannoma, neurilemoma)
Ganglioneuroblastoma
5. In a 7-year-old child, a tumor was found on the neck in the form of a soft-elastic nodule, clearly cleansed from the surrounding tissues with the presence of pronounced secondary changes in the form of ecchymosis, hemorrhages, and necrosis. There are small cysts in the tumor tissues. The microscopic picture is polymorphic. The tumor consists of immature neurocytes of various degrees of differentiation, ranging from sympathogonia and sympathoblasts to highly differentiated ganglion cells. Many active mitoses. Your diagnosis?:
AND Neuroblastoma
BMalignant neuroma
Ganglioneuroma
DNeurinoma (givannoma, neurymoma)
Ganglioneuroblastoma
6. Three brown spots measuring 0.5 to 1 cm in diameter were found on the skin of the upper limbs of a young woman. They have the appearance of growths of the form of warts, slightly protruding above the surface of the skin. Upon microscopic examination, cells with a large amount of melanin are located in the dermis in the form of separate clusters, and giant multinucleated cells are also found. Your diagnosis?:
AND Intradermal nevus
BMelanoma
C Mixed nevus
D Hemangioma

EInvolutive nevus (fibrous papule of the nose)
7. A 21-year-old patient had a tumor of the frontal lobe of the right hemisphere of the brain with a diameter of 5 cm, which was indistinctly separated from the surrounding tissue, removed. On the section, it has a uniform appearance, histologically, it consists of star-shaped cells, the numerous processes of which form dense plexuses. What kind of tumor did the patient have?:
AND Astrocytoma
Ependymoma
Choroid papilloma
Oligodendroglioma
Ganglioneuroma
8. During a neurosurgical operation, the patient had a neoplasm removed - an extra ventricular node with cysts and foci of necrosis. A histological examination revealed a cluster of unipolar and bipolar cells located near vessels and cavities. Your diagnosis?:
AND Ependymoblastoma
Ganglioneuroma
CHorioid papilloma
Choroid carcinoma
Glioblastoma
9. A 24-year-old patient did not regain consciousness after an appendectomy for acute appendicitis (pathological conclusion - phlegmonous appendicitis). He was on artificial ventilation for 1 month, his condition worsened and he died in a coma. During the examination of the brain in the subcortical zone of the left parietal lobe, a one-chamber cavity with a diameter of 4x5x6 cm was found, without liquid. The cyst wall is 1-2 mm, gray-white in color, without clear boundaries, microscopically: it consists of parallel bundles of glial fibers, between which there are cells of different sizes with processes that resemble astrocytes. Your diagnosis?:
AND Astrocytoma
BGlial scar
Astroblastoma
Oligodendroglioma
Glioblastoma
10. A 39-year-old woman had a pink-gray nodular tumor removed. It was localized in the frontal region and was up to 3 cm in diameter. Microscopically, it is represented by small monomorphic cells with round nuclei located in light cytoplasm. Places of lime deposition are observed. Your diagnosis?:
AND Oligodendroglioma
BAstroblastoma
Astrocytoma
EPendymoma
EMeningioma
11. A 75-year-old patient turned to a surgeon with complaints of a brownish leg ulcer that did not heal for a long time. During biopsy examination: diffuse growth of

polymorphic atypical cells, in the cytoplasm of which there is a brown pigment. Perls' reaction is negative. Many pathological mitoses and centers of tissue necrosis. What is the most likely diagnosis?
AND Melanoma
BLocal hemosiderosis
Intradermal nevus
D Trophic ulcer
Skin cancer
12. A 46-year-old man had a dark spot on his skin that erupted and did not cause concern. Over time, the spot began to increase in size, pain appeared, the color became black-brown; the nodule began to be palpated. Histological examination of the removed tissue revealed spindle-shaped and polymorphic cells with numerous mitoses, the cytoplasm of which contained a brown pigment. What tumor is it about?
AND Melanoma
Bbasalioma
Hemangioma
Nevus
E-
13. Histological examination of the removed eyeball revealed a black tumor measuring 1x0.4 cm thick in the choroid. Microscopically: large polymorphic cells grouped in an alveolar structure. Numerous pathological mitoses were detected, and the cytoplasm of many of them contained a yellow-brown pigment. Your diagnosis:
ANDMelanoma
In Neurinoma
C Angiosarcoma
D Neuroblastoma
E Ganglioneuroblastoma
14. A young woman was diagnosed with a tumor in the form of a blue-black soft area on her face. Microscopically, the tumor is of pronounced polymorphism, the tumor consists of spindle-shaped or pleomorphic, distorted cells. In the cytoplasm of many cells, the pigment is yellowish-brown. Many mitoses. Your diagnosis:
ANDJuvenile nevus
B Malignant neuroma
C Melanoma
D Neuroblastoma
E Ganglioneuroblastoma
15. In the case of histological examination of malignant melanoma, the prognosis is an unfavorable course of the disease associated with the manifestation of:
A Character of cellular morphology of tumors
B Propensity of the tumor to horizontal growth
C Accumulation of pigment in tumor cells
D Tendency of the tumor to vertical growth
E Disintegration of the tumor

16. Intraoperatively, a 4.5x5x3.5 cm dark brown tumor was found in the liver of a 17-year-old patient. Your previous diagnosis.
ANDMelanoma metastases
B Hemangiopericytoma
C Cavernous hemangioma
D Capillary hemangioma
E Lymphangioma

3.2. Algorithm of description of macropreparation and micropreparation

2. Description of macropreparation:
    1. Specify the name of the organ or ego part;
    2. Specify the dimensions of the body (length, widthother, thickness);
    3. Specify the surface of the organ, the condition of the capsule, overlap;
    4. Specify the consistency of the organ;
    5. The type and structure of the organ at autopsy;
    6. Indicate the presence of a pathological formation (if any);
    7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.

"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Final lesson. (Subsections Immunopathological processes. Regeneration, processes of adaptation and compensation. Tumors). Practical experience.".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association

3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 16

Topic:. Nomenclature and morphological features of tumors originating from melanin-producing tissue.

Goal: To study and know the nomenclature and morphological features of tumors originating from melanin-producing tissue. To know the patterns of growth and metastasis of such tumors.

Basic concepts: Mmelanoma (nodular melanoma, superficial melanoma, lentiginous melanoma, acral melanoma, depth of melanoma invasion according to Breslow and Clark); nevus (intradermal, mixed, borderline, juvenile, epithelioid cell, dysplastic)

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic: emphasize the definition or provide an explanation. Nevus (intradermal nevus, border nevus, mixed nevus, blue nevus, juvenile nevus), melanoma (nodular melanoma, superficial melanoma, lentiginous melanoma, acral melanoma, depth of invasion of melanoma according to Breslow and Clark).

2.2. Flow chart on the topic as a list of didactic units of the topic.

Tumors of melanin-forming tissue

develop from cells of neuroectodermal origin - melanocytes, which are contained in the basal layer of the epidermis, hair follicles, soft brain membranes, retina and cornea of the eye. Melanocytes can be the source of tumor-like formations - nevi and malignant tumors - melanoma. Nevi are found in the skin of the face, trunk and other parts of the body in the form of dark exploding formations. They can be of several types: borderline, intradermal, complex (mixed), epithelioid, or spindle cell (juvenile), blue.

Melanomas(melanoblastomas) are more common in women and occur on the skin, pigment membrane of the eye, medulla of the adrenal glands, and meninges. They grow in the form of a node or with surface distribution. Melanoma, as a rule, has the appearance of a brown (brown) spot with pink or black spots, a blue-black soft nodule or plaque. Yellow-brown melanin is often detected in the cytoplasm of cells, although pigmentless melanomas are sometimes found. Melanoma early gives hematogenous and lymphogenic metastases. The development of melanoma is often associated with increased solar insolation. Sometimes melanomas arise at the site of pigment formations: Hutchinson's spots (Lentigo maligna), dysplastic nevus, congenital giant nevi.

2.3.List questions to check basic knowledge on the subject of the lesson.

1. Classification and nomenclature of tumors originating from melanin-forming tissue
2. Melanomas: definition, classification, morphological characteristics, features of metastasis, determination of the depth of invasion according to Clark and Breslow.
3. Nevi: definition, classification, morphological characteristics of different types of nevi.

3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 Test tasks

1. In the region of the auditory nerve, a young woman was found to have a tumor in the form of a node up to 3 cm in diameter, soft-elastic, pink-white in color, homogeneous. Microscopically, the tumor is a bunch of cells with oval or curved nuclei. Fibro-cellular bundles tend to form rhythmic structures in the form of palisade structures, which are formed by parallel rows of correctly oriented cells located in the form of a palisade, between which there is a cell-free homogeneous zone. Such structures were called "Verokai's bodies". Your diagnosis?:

AND Neurinoma (schwannoma, neurilemoma)
BMalignant neurilema
Ganglioneuroma
Neuroblastoma
Ganglioneuroblastoma

2. In a young man, a tumor in the form of several nodes (multicentric growth) of a soft-elastic consistency, white in section with large foci of hemorrhages, mucinization, necrosis, and the presence of cystic cavities was found in the chest cavity. Microscopically - sharply expressed polymorphism. Cells with oval and elongated nuclei are characteristic, which are arranged randomly or form bundles in the form of palisades. There are multinucleated symplasts, individual xanthoma cells. Your diagnosis?:

AND Malignant neuroma
BNeurinoma (schwannoma, neurilemoma)

Ganglioneuroma
Neuroblastoma
Ganglioneuroblastoma
3. A 46-year-old man had a dark spot on his scalp that did not cause discomfort. Recently, the spot began to increase in size, pain appeared, the color became brown, black-brown. During histological examination of the removed tissue, spindle-shaped and polymorphic cells are revealed, in the cytoplasm of which there is a brown-black pigment. Your diagnosis:
AND Melanoma
BHematoma
Hemangioma
Basalioma
ECarcinoid
4. In a 22-year-old man, a tumor was found in the adrenal glands, which has the appearance of a node, in places with infiltrative growth, soft consistency, yellowish color, with large areas of necrosis and hemorrhages. In addition, metastases were found in the liver (Pepper) and in the orbit of the eye (Hutchinson). Microscopically, the tumor consists of two types of cells. Some are small, like sympathogonia, round, with a narrow rim of cytoplasm and a small oval nucleus (bare nucleus), in which chromatin is arranged in the form of grains. Others, like sympathoblasts, are large, with a light nucleus and a large amount of cytoplasm. Tumor cells tend to form true and false rosettes in the form of a corolla of cells, in the center of which a delicate fibrillar substance is found. Your diagnosis?:
AND Neuroblastoma
BMalignant neuroma
Ganglioneuroma
DNeurinoma (schwannoma, neurilemoma)
Ganglioneuroblastoma
5. In a 7-year-old child, a tumor was found on the neck in the form of a soft-elastic nodule, clearly cleansed from the surrounding tissues with the presence of pronounced secondary changes in the form of ecchymosis, hemorrhages, and necrosis. There are small cysts in the tumor tissues. The microscopic picture is polymorphic. The tumor consists of immature neurocytes of various degrees of differentiation, ranging from sympathogonia and sympathoblasts to highly differentiated ganglion cells. Many active mitoses. Your diagnosis?:
AND Neuroblastoma
BMalignant neuroma
Ganglioneuroma
DNeurinoma (givannoma, neurymoma)
Ganglioneuroblastoma
6. Three brown spots measuring 0.5 to 1 cm in diameter were found on the skin of the upper limbs of a young woman. They have the appearance of growths of the form of warts, slightly protruding above the surface of the skin. Upon microscopic

examination, cells with a large amount of melanin are located in the dermis in the form of separate clusters, and giant multinucleated cells are also found. Your diagnosis?:
AND Intradermal nevus
BMelanoma
C Mixed nevus
D Hemangioma
EInvolutive nevus (fibrous papule of the nose)
7. A 21-year-old patient had a tumor of the frontal lobe of the right hemisphere of the brain with a diameter of 5 cm, which was indistinctly separated from the surrounding tissue, removed. On the section, it has a uniform appearance, histologically, it consists of star-shaped cells, the numerous processes of which form dense plexuses. What kind of tumor did the patient have?:
AND Astrocytoma
Ependymoma
Choroid papilloma
Oligodendroglioma
Ganglioneuroma
8. During a neurosurgical operation, the patient had a neoplasm removed - an extra ventricular node with cysts and foci of necrosis. A histological examination revealed a cluster of unipolar and bipolar cells located near vessels and cavities. Your diagnosis?:
AND Ependymoblastoma
Ganglioneuroma
CHorioid papilloma
Choroid carcinoma
Glioblastoma
9. A 24-year-old patient did not regain consciousness after an appendectomy for acute appendicitis (pathological conclusion - phlegmonous appendicitis). He was on artificial ventilation for 1 month, his condition worsened and he died in a coma. During the examination of the brain in the subcortical zone of the left parietal lobe, a one-chamber cavity with a diameter of 4x5x6 cm was found, without liquid. The cyst wall is 1-2 mm, gray-white in color, without clear boundaries, microscopically: it consists of parallel bundles of glial fibers, between which there are cells of different sizes with processes that resemble astrocytes. Your diagnosis?:
AND Astrocytoma
BGlial scar
Astroblastoma
Oligodendroglioma
Glioblastoma
10. A 39-year-old woman had a pink-gray nodular tumor removed. It was localized in the frontal region and was up to 3 cm in diameter. Microscopically, it is represented by small monomorphic cells with round nuclei located in light cytoplasm. Places of lime deposition are observed. Your diagnosis?:
AND Oligodendroglioma

BAstroblastoma
Astrocytoma
EPendymoma
EMeningioma
11. A 75-year-old patient turned to a surgeon with complaints of a brownish leg ulcer that did not heal for a long time. During biopsy examination: diffuse growth of polymorphic atypical cells, in the cytoplasm of which there is a brown pigment. Perls' reaction is negative. Many pathological mitoses and centers of tissue necrosis. What is the most likely diagnosis?
AND Melanoma
BLocal hemosiderosis
Intradermal nevus
D Trophic ulcer
Skin cancer
12. A 46-year-old man had a dark spot on his skin that erupted and did not cause concern. Over time, the spot began to increase in size, pain appeared, the color became black-brown; the nodule began to be palpated. Histological examination of the removed tissue revealed spindle-shaped and polymorphic cells with numerous mitoses, the cytoplasm of which contained a brown pigment. What tumor is it about?
AND Melanoma
Bbasalioma
Hemangioma
Nevus
E-
13. Histological examination of the removed eyeball revealed a black tumor measuring 1x0.4 cm thick in the choroid. Microscopically: large polymorphic cells grouped in an alveolar structure. Numerous pathological mitoses were detected, and the cytoplasm of many of them contained a yellow-brown pigment. Your diagnosis:
ANDMelanoma
In Neurinoma
C Angiosarcoma
D Neuroblastoma
E Ganglioneuroblastoma
14. A young woman was diagnosed with a tumor in the form of a blue-black soft area on her face. Microscopically, the tumor is of pronounced polymorphism, the tumor consists of spindle-shaped or pleomorphic, distorted cells. In the cytoplasm of many cells, the pigment is yellowish-brown. Many mitoses. Your diagnosis:
ANDJuvenile nevus
B Malignant neuroma
C Melanoma
D Neuroblastoma
E Ganglioneuroblastoma

15. In the case of histological examination of malignant melanoma, the prognosis is an unfavorable course of the disease associated with the manifestation of:
A Character of cellular morphology of tumors
B Propensity of the tumor to horizontal growth
C Accumulation of pigment in tumor cells
D Tendency of the tumor to vertical growth
E Disintegration of the tumor
16. Intraoperatively, a 4.5x5x3.5 cm dark brown tumor was found in the liver of a 17-year-old patient. Your previous diagnosis.
ANDMelanoma metastases
B Hemangiopericytoma
C Cavernous hemangioma
D Capillary hemangioma
E Lymphangioma

3.2. Algorithm of description of macropreparation and micropreparation

3. Description of macropreparation:
   1. Specify the name of the organ or ego part;
   2. Specify the dimensions of the body (length, widthother, thickness);
   3. Specify the surface of the organ, the condition of the capsule, overlap;
   4. Specify the consistency of the organ;
   5. The type and structure of the organ at autopsy;
   6. Indicate the presence of a pathological formation (if any);
   7. Conclude.

Description of the micropreparation:

5. Specify the name of the body;
6. Specify the color;
7. Specify what changes in cells;
8. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Final lesson. (Subsections Immunopathological processes. Regeneration, processes of adaptation and compensation. Tumors). Practical experience.".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O.

Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

10. http://moz.gov.ua- Ministry of Health of Ukraine
11. www.ama-assn.org– American Medical Association /American Medical Association
12. www.who.int- World Health Organization
13. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
14. http://bma.org.uk– British Medical Association
15. www.gmc-uk.org- General Medical Council (GMC)
16. www.bundesaerztekammer.de– German Medical Association
17. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
18. http://www.webpathology.com/- Web Pathology

Practical lesson No. 17

Topic: Final lesson. (Subsections Immunopathological processes. Regeneration, processes of adaptation and compensation. Tumors). Practical experience.

Goal: Check the quality and depth of mastering the material on the following topics:Immunopathological processes. Regeneration, processes of adaptation and compensation. Tumors". To find out the ability to apply theoretical knowledge in the diagnosis of pathological processes.

Basic concepts:listed in the relevant topics.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop.

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic: emphasize the definition or provide an explanation. Hypersensitivity (anaphylaxis, atopy, granulomatosis, cytotoxic type of hypersensitivity, immunocomplex type of hypersensitivity) Autoimmune diseases (organ-specific autoimmune diseases, organ-nonspecific autoimmune diseases) Immunodeficiency, primary immunodeficiency syndromes, secondary immunodeficiency syndromes, accidental involution of the thymus, graft rejection, graft versus host, thymomegaly (sudden infant death syndrome).

Hypertrophy, hyperplasia, false hyperplasia (hypertrophy), atrophy, cachexia, brown cardiac atrophy, brown liver atrophy, metaplasia, dysplasia, scar, granulation tissue, pathological regeneration.
Tumor; invasive growth, appositional growth, expansive growth, unicentric growth, multicentric growth, exophytic, endophytic growth; atypism, morphological atypism, tissue atypism, cellular atypism, anaplasia; metastases
Papilloma, adenoma (cystadenoma, myoma, acinar, trabecular adenoma), cancer (adenocarcinoma, signet-ring cell carcinoma, solid cancer, mucosal carcinoma, cirrhosis, medullary carcinoma, squamous cell carcinoma).
Sarcoma (fibrosarcoma, liposarcoma, malignant hibernoma, angiosarcoma, osteosarcoma, chondrosarcoma, myosarcoma (rhabdomyosarcoma, leiomyosarcoma); fibroma, desmoid, leiomyoma, rhabdomyoma, lipoma, hibernoma, chondroma, osteoma, angioma (lymphangioma, hemoglomangioma), angioma.
Nevus (intradermal nevus, border nevus, mixed nevus, blue nevus, juvenile nevus), melanoma (nodular melanoma, superficial melanoma, lentiginous melanoma, acral melanoma, depth of invasion of melanoma according to Breslow and Clark). Glioma (astrocytoma, oligodendrogioma, ependymoma), meningioma, schwannoma, ganglioma, glioblastoma. Mature teratoma, immature teratoma, malignant teratoma, hamartoma, neuroblastoma, nephroblastoma, retinoblastoma.

2.2. Flow chart on the topic as a list of didactic units of the topic. Theoretical material is provided on relevant topics.
2.3. A complete list of questions to check basic knowledge on the subject of the lesson. Provided by topics
3. Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks: provided in the listed topics.

3.2. the algorithm for describing the macropreparation and the micropreparation are provided in the listed topics

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Anemia. Thrombocytopathies. Tumors of hematopoietic and lymphoproliferative tissue".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 18

Topic: Anemia. Thrombocytopathies.

Goal: To study the definition, pathogenesis, classifications andmute, thrombocytopenia; morphological changes, complications and causes of death in these diseases.

Basic concepts:anemia (posthemorrhagic, iron-deficient, B-12, folate-deficient, hemolytic, hypoplastic, hypo-hyperchromic, acquired, congenital, acute, chronic).

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic: emphasize the definition or provide an explanation. Nosology, disease, symptom, pathomorphosis. Anemia (posthemorrhagic, iron-deficient, B-12, folate-deficient, hemolytic, hypoplastic, hypo-hyperchromic, acquired, congenital, acute, chronic).

2.2. Flow chart on the topic as a list of didactic units of the topic.

Special pathological anatomy studies the material substrate of the disease, that is, it is a subject of nosology. Nosology (from the Latin noso - disease and logos - teaching) - the study of diseases, involves knowledge of etiology,

pathogenesis, manifestations (clinical and morphological) and consequences of the disease, classification and nomenclature of diseases, variability (pathomorphosis), as well as the construction of a diagnosis, principles treatment and prevention.

A disease is understood as a violation of the vital activity of the body under the influence of a certain cause. The essence of the disease is solved ecologically (from the Greek oikos - home, dwelling), that is, in terms of disturbed normal relations of the organism with the environment. This interpretation of the disease developed in the second half of the 19th century. Prominent Russian clinician O.O. Ostroumov - considered the disease as a violation of the normal life of a person with the conditions of his existence in the environment. S.P. Botkin believed that disease is a reaction of the body to the harmful effects of the external environment.

K. Bernard defined the disease as a violation of the physiological balance of the body. The interpretation of the disease in the ecological plan allows us to put forward the following theoretical propositions, which must be taken into account when studying the disease:

Classification of diseases takes into account the following signs:

1. Etiological, which allows dividing diseases into hereditary (congenital) and acquired, and the latter into non-infectious and infectious.

2. Anatomical and topographic, i.e. localization of the main focus of damage. In this regard, diseases of organ systems (diseases of the cardiovascular system), organs (diseases of cells) and tissues (diseases of connective tissue) are distinguished.

3. The commonality of pathogenetic mechanisms, on the basis of which allergic, autoimmune and rheumatic diseases are distinguished.

4. The commonality of socially mediated influence on the organism of environmental factors, which is the basis of the origin of occupational diseases, geographical and military pathology, etc.

5. Common forms of development and course of diseases make it possible to distinguish the most acute, acute, subacute and chronic, as well as cyclic and acyclic diseases.

6. Sex and age, which are used in the selection of female, male and children's diseases, as well as diseases of the elderly. When classifying diseases, their nomenclature is followed.

Anemia Anemia is a decrease in the number of erythrocytes and hemoglobin per unit volume of blood, which is often combined with changes in their quality. It can be an independent disease or one of the symptoms of other diseases and pathological conditions. From a practical point of view, the main characteristic of anemia is a decrease in Hb content in a unit of blood volume. Therefore, the essence of anemia and its importance for the body are determined primarily by a decrease in the oxygen capacity of the blood, leading to hypoxia

of the hemic type. It is hypoxia that is associated with the main clinical symptoms and disorders of vital activity in patients. General clinical manifestations of anemia Anemic syndrome (paleness of the skin and visible mucous membranes and symptoms caused by hypoxia - rapid fatigue, weakness, dizziness). Syndromes due to the peculiarity of the pathogenesis of each individual type of anemia (for example, with iron-deficiency anemia sideropenic syndrome, with B12-folate-deficient anemia – neurological disorders and damage to the gastrointestinal tract, with hemolytic anemia – jaundice). Clinical symptoms caused by compensatory reactions aimed at compensating for hypoxia (hyperventilation, tachycardia, etc.). 5 Hematological signs of anemia are divided into quantitative and qualitative. Quantitative hematological signs of anemia: • Decrease in the number of erythrocytes per unit volume of blood (less than 4 × 1012/l in men, less than 3.5 × 1012/l in women and children). • Decrease in hemoglobin concentration (in men less than 130 g/l, in women less than 120 g/l, in children under 6 years less than 110 g/l, in children older than 6 years less than 120 g/l). • Decrease in hematocrit (less than 43% in men, less than 40% in women). • Changes in color index (norm 0.85–1). Qualitative hematological signs of anemia: • Presence of regenerative forms of erythrocytes. • The presence of degenerative changes in the cells of the erythrocyte row. • The presence of cells of pathological regeneration. Classification of anemias Anemias are classified depending on the etiology, pathogenesis, type of hematopoiesis, ability of the bone marrow to regenerate, color index, diameter of erythrocytes, clinical course, degree of severity. Pathogenetic classification of anemias: • due to blood loss (posthemorrhagic acute and chronic); • as a result of a violation of erythropoiesis (deficient in vitamin, protein, iron, hypoplastic, aplastic); • due to increased blood loss: hemolytic - hereditary (hemoglobinopathies, fermentopathy, membranopathies) and acquired (autoimmune, heteroimmune, isoimmune, transimmune). Deficiency anemias are distinguished: iron deficiency, protein deficiency, vitamin-deficient (primarily B12-deficient, folio-deficient) anemias. Iron-deficiency anemia (IDA) is an anemia caused by a lack of iron in the body as a result of an imbalance between its intake, use and loss.

B12- and foliodeficiency anemia is associated with vitamin B12 deficiencyand folic acid, as a result of which the synthesis of nucleic acids is disturbed and the erythroblastic type of hematopoiesis is replaced by megaloblastic. Reasons: 1. Exogenous (alimentary) deficiency. It can develop in young children when feeding with goat's milk or dry milk formulas. 2. Violation of absorption of vitamin B12 in the small intestine: • violation of the formation and secretion of gastromucoprotein (Kastle's intrinsic factor) in hereditary disorders, atrophy of the CO of the stomach, autoimmune damage to the parietal cells of the CO of the stomach, after resection of the stomach or removal of more than 2/3 of the stomach; • disorders of the function of the small intestine (chronic diarrhea - celiac disease, resection of large sections of the intestine); competitive use of vitamin B12 by helminths (diphyllobotriosis) and intestinal microflora. 3.

Violation of the formation of transcobalamins in the liver. 4. Violation of the deposition of vitamin B12 in the liver (hepatitis, cirrhosis). 5. Increasing the use of vitamin B12 (during pregnancy).

Hemolytic anemias Hemolytic anemias are a group of diseases characterized by a decrease in the average life expectancy of erythrocytes and a predominance of the intensity of hemolysis (destruction) of erythrocytes over their formation. Among blood diseases, hemolytic anemias make up 5%, and among anemic states - 11%. Hemolysis of erythrocytes can be intravascular and extravascular (intracellular) - in the spleen, liver or bone marrow. Intravascular hemolysis is accompanied by the release of Hb from the cells into the plasma, where it partially combines with the haptoglobin protein. Intracellular hemolysis of erythrocytes develops as a result of absorption and digestion of erythrocytes by macrophages. The main clinical syndromes of hemolytic anemias: 1. Hypoxia. It is caused by anemia and is manifested by sharp weakness, unpleasant sensations in the area of the heart, palpitations, shortness of breath. 2. Hemolytic jaundice. 3. Increased formation of gallstones, especially bilirubin stones. It is explained by a significant increase in the content of bilirubin in bile and an increase in its viscosity. 4. Hemoglobinuria. It develops during hemolysis. Hemoglobin, which is released from destroyed erythrocytes, binds to the blood plasma protein haptoglobin. 100 ml of blood plasma contains so much haptoglobin that it can bind 125 mg of Hb. If the concentration of Hb in the plasma is higher than 125 mg%, then the unbound Hb passes through the kidney filter and appears in the urine. 5. Splenomegaly. Characteristic of the intracellular mechanism of erythrocyte hemolysis. The basis of this phenomenon is an increase in the functional activity of macrophages, which causes their active proliferation. 9 Splenomegaly is often accompanied by liver enlargement (proliferation of liver macrophages). 6. Hemosiderosis - deposition of hemosiderin in macrophages. Hemosiderin is partially denatured and deproteinized ferritin, that is, a protein containing a lot of iron in non-heme form (iron content in hemosiderin is 25–30%). 7. Violation of microcirculation. They often occur with intense intravascular hemolysis and are caused by the development of CVD syndrome. 8. Fever. It develops as a result of sharp activation of the phagocytic function of macrophages, as a result of which they secrete interleukin-1.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Definition of anemia. Laboratory criteria for the diagnosis of anemia.
2. Classification of anemias. (by criteria: erythrocyte morphology, pathogenesis, color index, ability of bone marrow to regenerate).
3. Anemias due to blood loss: acute and chronic posthemorrhagic anemia.
Morphological characteristics of tissues and organs, complications, causes of death.

4. Anemias caused by a violation of the formation of erythrocytes and/or hemoglobin: iron-deficiency anemia, vitamin B12-, folate-deficiency anemia, hypoaplastic anemia, siderochrist anemia, vitamin B6 deficiency anemia, anemia due to a violation of the synthesis or use of porphyrins.
5. Anemias due to increased destruction of erythrocytes (hemolytic anemia): spherocytosis (ovalocytosis), thalassemia, sickle cell anemia, immune and non-immune (due to intravascular hemolysis) hemolytic anemia; polydeficiency anemia.
6. Thrombocytopenia, definition, general characteristics, clinical significance.
7. Thrombocytopathies, definition, general characteristics, clinical significance.
8. Coagulopathy. definition, general characteristics, clinical significance. DVZ-syndrome.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1. Test tasks

1. During the examination of a 35-year-old patient, a histological examination of the red bone marrow punctate was performed and a significant decrease in the number of megakaryocytes was found. What changes in peripheral blood will this lead to?
AND Thrombocytopenia
BLeukocytosis
Thrombocytosis
Agranulocytosis
Leukopenia
2. During the autopsy of a deceased 96-year-old man who suffered from atherosclerosis and died of massive blood loss, a ruptured aneurysm of the abdominal aorta was found. A large amount of clotted blood surrounds the aneurysm. What type of anemia develops with this pathology?:
AND Acute posthemorrhagic
B Chronic post-hemorrhagic
Pernicious
D Hemolytic
EHypoplastic
3. During the examination of a 58-year-old patient suffering from varicose veins with periodic bleeding from them, a decrease in the number of erythrocytes and hemoglobin was revealed. The skin and mucous membranes are pale. What type of anemia did the patient develop?:
AND Chronic posthemorrhagic
BAcute posthemorrhagic
Hemolytic
Pernicious
EHypoplastic
4. During the examination of the patient, pallor of the skin, yellowness of the sclera was noted. The tongue is smooth, shiny, as if polished. Microscopic examination of the stomach biopsy revealed a decrease in the glands in the fundal part of the stomach. The epithelium is atrophic, only the main cells are preserved. Macrocytes and megalocytes are detected in the blood smear. This picture is characteristic of:
AND Pernicious anemia

B Iron deficiency anemia
Hypoplastic anemia
Hemolytic anemia
Posthemorrhagic anemia
5. A blood test of a 43-year-old man who worked with radioactive isotopes at a research institute revealed anemia, leukopenia, and thrombocytopenia. In punctate bone marrow, replacement of bone marrow with fat is observed. What type of anemia is characterized by the following changes?:
AND Hypoplastic
B Hemolytic
Pernicious
Posthemorrhagic
Iron deficiency
6. A 44-year-old patient, who suffered from fibro-cavernous tuberculosis for a long time, started pulmonary bleeding with blood loss in the amount of 1 liter. What type of anemia occurs in this case?:
AND Acute posthemorrhagic
B Chronic post-hemorrhagic
Hemolytic
D Iron deficiency
Pernicious
7. A 52-year-old woman suffering from iron – cystic hypoplasia of the endometrium periodically has pathological uterine bleeding. What type of anemia can occur?:
AND Chronic posthemorrhagic
BAcute posthemorrhagic
Hemolytic
DAplastic
Pernicious
8. A 47-year-old man, who suffered from stomach ulcer disease for a long time, underwent a partial resection of the stomach. During a blood test, he was found to be anemic. Anemia is a consequence?:
AND Lack of iron
BVitamin B12 deficiency
Suppression of hematopoiesis
DIncreased breakdown of erythrocytes
Acute bleeding
9. In the deceased, after a severe large burn of the skin, the following were found: general hemosiderosis, jaundice, bone marrow of tubular bones is red, in spongy bones - juicy, pinkish-red. Loose connective tissue has multiple hematopoietic cells. For which type of anemia are these changes characteristic?:
AND A. Hemolytic anemia caused by intravascular hemolysis
BHemolytic anemia caused by extravascular hemolysis
Iron deficiency anemia
Acute posthemorrhagic anemia
Chronic posthemorrhagic anemia
10. When examining the blood of a patient who complained of increased fatigue, drowsiness, immature forms of erythropoiesis - erythroblasts, normoblasts and megaloblasts - were found. The same elements were found in the bone marrow punctate. What type of anemia is characterized by this blood pattern?:
AND Pernicious (Addison – Birmer)
B Hemolytic
Posthemorrhagic

3.2. algorithm for describing a macropreparation and a micropreparation
Description of macropreparation:
15. Specify the name of the organ or ego part;
16. Specify the dimensions of the body (length, width, thickness);
17. Specify the surface of the organ, the condition of the capsule, overlap;
18. Specify the consistency of the organ;
19. The type and structure of the organ at autopsy;
20. Indicate the presence of a pathological formation (if any);
21. Conclude.

Description of the micropreparation:
1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:
- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:
- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors,

	expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Atherosclerosis and arteriosclerosis. Coronary heart disease".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association

6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9. http://www.webpathology.com/- Web Pathology

Practical lesson No. 18

Topic: Tumors of hematopoietic and lymphoproliferative tissue.

Goal: Learn and be able to apply knowledge of the morphology of tumors of hematopoietic and lymphoproliferative tissue in diagnostics.Know the definition, pathomorphogenesis, forms, morphological signs of lymphogranulomatosis.

Basic concepts:leukemia, lymphoma, Hodgkin's disease, leukemic failure, leukemic crisis, myeloma, Burkitt's lymphoma.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic: emphasize the definition or provide an explanation. Leukemia, lymphoma, Hodgkin's disease, leukemic failure, leukemic crisis, myeloma, Burkitt's lymphoma.

2.2. Flow chart on the topic as a list of didactic units of the topic.

Diseases of the blood system make up the content of clinical hematology, the founders of which in our country are I.I. Mechnikov, S.P. Botkin, M.I. Arinkin, O.I. Kryukov, I.O. Kasirskyi. These diseases develop as a result of disturbances in the regulation of hematopoiesis and hematopoiesis, which is reflected in the composition of peripheral blood. Therefore, based on the data of the study of the composition of peripheral blood, it is possible to roughly judge the state of the hematopoietic system as a whole. Yes, we can talk about changes in red and white sprouts, as well as blood plasma, both quantitatively and qualitatively.

Changes in the red germ of the blood system may refer to a decrease in the hemoglobin content and the number of erythrocytes (anemia) or their increase (true, real polycythemia or erythremia); disorders of the shape of erythrocytes - erythrocytopathy (microspherocytosis, ovalocytosis) or hemoglobin synthesis - hemoglobinopathy or hemoglobinosis (thalassemia, sickle cell anemia),

(hemoblastosis). It is equally possible to talk about an increase in the number of platelets (thrombocytosis) or their decrease (thrombocytopenia) in peripheral blood, as well as changes in their quality (thrombocytopathy).

The most complete picture of the state of the hematopoietic system is provided by the study of bone marrow punctate (sternum) and trepanobiopsy (iliac crest), which are widely used in the hematological clinic. Diseases of the blood system are extremely diverse. Anemias, hemoblastoses (neoplastic diseases arising from hematopoietic cells), thrombocytopenia and thrombocytopathy are of greatest importance.

Changes in the white germ of the blood system concern both leukocytes and platelets. The number of leukocytes in the peripheral blood may increase.

Tumors of the blood system or hemoblastosis are divided into two groups:

leukemia - systemic tumor diseases of hematopoietic tissue;

lymphomas are regional tumor diseases of hematopoietic and/or lymphatic tissue.

Classification of hematopoietic and lymphatic tissue tumors:

I. Leukosis is a systemic tumor disease.

A. Acute leukemias: 1) undifferentiated; 2) myeloblastic; 3) lymphoblastic; 4) plasmablastic; 5) monoblastic (myelomonoblastic); 6) erythromyeloblastic; 7) megakaryoblastic.

B. Chronic leukemias.

Myelocytic origin: 1) chronic myeloid; 2) chronic erythromyelosis; 3) erythremia; 4) true polycythemia (Vaquez-Osler syndrome).

Lymphocytic origin: 1) chronic lymphocytic leukemia; 2) lymphomatosis of the skin (Sézary's disease); 3) paraproteinemic leukemias: a) myeloma disease; b) primary macroglobulinemia (Waldenstrom's disease); c) disease of heavy chains (Franklin's disease).

Monocytic origin: 1) chronic monocytic leukemia; 2) histiocytosis (histiocytosis X).

II. *Lymphomas*- regional tumor diseases.

Lymphosarcoma: lymphocytic, prolymphocytic, lymphoblastic, immunoblastic, lymphoplasmacytic; African lymphoma (Burkitt's tumor). Fungal mycosis. Sézary's disease. Reticulosarcoma. Lymphogranulomatosis (Hodgkin's disease).

Leukemias are systemic tumor diseases of hematopoietic tissue

Leukosis(leukemia) are characterized by systemic progressive growth of hematopoietic cells of tumor origin - leukemic cells. At first, tumor cells grow in hematopoietic organs (bone marrow, lymph nodes, spleen), then they are hematogenously migrated to other organs and tissues, forming leukemic (leukemic) infiltrates around blood vessels, in their walls; dystrophy and atrophy develop in the parenchymal elements and then they die. Infiltration by tumor cells can be diffuse (leukemic infiltration of the spleen, liver, kidneys,

mesentery), which causes a sharp increase in organs and tissues, or focal - with the formation of tumor nodes that grow into the capsule of organs and adjacent tissues. Quite often, tumor nodes appear against the background of diffuse leukemic infiltration, however,

The appearance of leukemic cells in the peripheral blood is quite typical for leukemias.

Incessant growth of leukemic cells in organs and tissues, their "flooding" of blood leads to anemia and hemorrhagic syndrome, severe dystrophic changes in parenchymal organs. Severe ulcerative-necrotic changes and complications of an infectious nature - sepsis - develop as a result of suppression of immunity in leukemia.

The question of the etiology of leukemias and tumors is closely related, because the tumor origin of leukemias is beyond doubt. Leukemias are polyetiological diseases, various factors are responsible for their occurrence, which can cause mutation of cells of the hematopoietic system.

Mutagens include viruses, ionizing radiation, and some chemicals.

The importance of viruses in the development of leukemia is shown in experiments on animals. In humans, it has been proven in cases of acute endemic T-lymphocytic leukemia (retrovirus HTLV-I), hairy cell leukemia (retrovirus HTLV-II) and Burkitt's lymphoma (Abstein-Barr DNA virus).

It is also known that ionizing radiation can cause the development of leukemia (radiation or radiation leukemia), and the frequency of mutations depends directly on the dose of ionizing radiation. After the atomic explosion in Hiroshima and Nagasaki, the number of patients with acute and chronic leukemia among those irradiated increased by 7.5 times.

Dibenzanthracene, benzpyrene, methylcholanthrene, i.e., blastomogenic substances, are chemicals that can cause leukemia.

The pathogenesis of leukemia is associated with the activation of cellular oncogenes (proto-oncogenes) under the influence of various etiological factors, which leads to impaired proliferation and differentiation of hematopoietic cells with subsequent malignant transformation. Increased expression of a number of proto-oncogenes has been registered in humans: ras (1st chromosome) – in various leukemias; sis (22nd chromosome) – in chronic leukemia; tus (8th chromosome) - in Burkitt's lymphoma.

The importance of hereditary factors in the development of leukemia is emphasized by the family nature of the disease. When studying the karyotypes of leukemic cells, changes in the set of their chromosomes are revealed - chromosomal aberrations. In chronic myeloma leukemia, for example, a decrease in the autosome of the 22nd pair of chromosomes of leukemic cells (Ph' - chromosome or Philadelphia chromosome) is constantly found. Ph' chromosome is also found in children with Down's disease, leukemia is 10-15 times more common among them.

Thus, the mutational theory of leukemia pathogenesis is the most likely. At the same time, the development of leukemia is subject to the rules of tumor progression. The change of monoclonality of leukemic cells to polyclonality is at the basis of the appearance of blast cells, their removal from the bone marrow and the progression of the disease - the blast crisis.

Depending on the degree of increase or decrease in the total number of leukocytes in the peripheral blood, including leukemic cells, leukemic cells are distinguished (tens and hundreds of thousands of leukocytes in 1 μl of blood); subleukemic (no more than 15,000–25,000 in 1 μl of blood), leukopenic (decrease in the number of leukocytes, but leukemic cells are detected) and leukemic (leukemic cells in the blood are absent) variants of leukemia.

Depending on the degree of differentiation (maturity) of blood tumor cells and the nature of the course (malignant or benign), leukemias are divided into acute and chronic.

Acute leukemia is characterized by the proliferation of undifferentiated or poorly differentiated blast cells ("blast" leukemias) and a malignant course; for chronic leukemia - proliferation of differentiated leukemic cells ("cytary" leukemias) and relative benign course.

Taking into account the histo(cyto)genesis of leukemic cells, histo(cyto)genetic forms of both acute and chronic leukemia are distinguished. In recent years, in connection with new ideas about hematopoiesis, the histogenetic classification of leukemias has undergone significant changes. The main feature of the new hematopoietic scheme is the selection of classes of precursor cells of various hematopoietic germs.

Based on modern ideas about hematopoiesis, the following histogenetic forms are distinguished among acute leukemias: undifferentiated, myeloblastic, lymphoblastic, monoblastic (myelomonoblastic), erythromyeloblastic and megakaryoblastic. Undifferentiated acute leukemia develops from precursor cells of the first three classes, which are devoid of morphological signs of belonging to one or another series of hematopoiesis. Other forms of acute leukemia originate from progenitor cells of class IV, that is, from blast cells.

*Chronic leukemias*depending on the number of maturing cells of hematopoiesis from which they arise, they are divided into: 1) leukemias of myelocytic origin; 2) leukemia of lymphocytic origin; 3) leukemias of monocytic origin. Chronic leukemias of myelocytic origin include: chronic myeloid leukemia, chronic erythromyelosis, erythremia, true polycythemia. Chronic leukemias of lymphocytic origin include: chronic lymphocytic leukemia, lymphomatosis of the skin (Sézary's disease) and paraproteinemic leukemias (myeloma disease; Waldenström's primary macroglobulinemia; Franklin's heavy chain disease). Leukemias of monocytic origin are monocytic (myelomonocytic) leukemia and histiocytosis (histiocytosis X).

The pathological anatomy of leukemias is peculiar and applies to both acute and chronic forms. There is a certain specificity of various types of them.

Acute leukemias. A diagnosis of acute leukemia is possible only when blast cells are found in the bone marrow (punctate from the sternum). Sometimes their number is 10–20%, but then a cluster of dozens of blasts is found in the trepanation from the iliac bone. In acute leukemia, both in the peripheral blood and in the myelogram, the so-called leukemic failure is found - a sharp increase in the number of blasts and single mature elements in the absence of transitional maturing forms.

Acute leukemias are characterized by replacement of the bone marrow with young blast elements and their infiltration of the spleen, liver, lymph nodes, kidneys, brain and its membranes, and other organs, the degree of which varies depending on the form of leukemia. The form of acute leukemia is established by cytochemical examination of blast cells. When treating patients with acute leukemia with cytostatic drugs, bone marrow aplasia and pancytopenia may develop.

Acute leukemias in children have some features. In comparison with acute leukemias in adults, they occur much more often and are characterized by a wider spread of leukemic infiltration in both hematopoietic and non-hematopoietic organs (except gonads). In children, leukemias with nodular (tumor-like) infiltrates are observed more often than in adults, especially in the region of the thymus gland; acute lymphoblastic (T-dependent) leukemia is more common; less often - myeloblastic leukemia. Congenital leukemia and chloroleukosis are special forms of acute leukemia in children.

Acute undifferentiated leukemia. This form of leukemia is characterized by infiltration of the bone marrow, spleen, lymph nodes, lymphoid formations (tonsils, group lymphatic and solitary follicles), vessel walls, kidneys and other organs by undifferentiated hematopoietic cells. Leukemic infiltration in such leukemias is monotonous; the spleen and liver are moderately enlarged. The bone marrow of flat and tubular bones is red, juicy, sometimes with a gray tint. In connection with leukemic infiltration of the mucous membrane of the oral cavity and tonsils, necrotic gingivitis occurs, tonsillitis - necrotic tonsillitis. Sometimes leukemia is joined by a secondary infection, then undifferentiated acute leukemia proceeds as a septic disease.

Leukemic infiltration of tissues and organs is often associated with hemorrhagic syndrome, the development of which can be explained not only by the destruction of blood vessel walls by leukemic cells, but also by anemia, impaired platelet formation due to the replacement of bone marrow by undifferentiated cells of hematopoiesis. Hemorrhages occur in the skin, mucous membranes, internal organs, quite often in the brain.

Patients with this form of leukemia die from brain hemorrhages, gastrointestinal bleeding, necrotic-ulcerative complications, and sepsis.

Acute myeloblastic leukemia(acute myelogenous leukemia). In this form of acute leukemia, there is infiltration of the bone marrow, liver, spleen, kidneys, less often lymph nodes and skin by tumor cells of the myeloblastic series with

cytochemical features: glycogen, sudanophilic inclusions are found in them; show a positive reaction to peroxidase, anaphtylesterase and chloroacetate esterase.

The bone marrow becomes red or grayish, sometimes it acquires a purulent shade (pyoid bone marrow). The spleen and liver due to leukemic infiltration increase, but slightly; the same changes occur in the lymph nodes. Infiltration by blast cells is quite characteristic not only of the bone marrow, spleen and liver, but also of the mucous membrane of the gastrointestinal tract, as a result of which necrosis occurs in the oral cavity, tonsils, throat, and stomach. Both diffuse and focal (tumorous) infiltrates are found in the kidneys. In 1/3 of cases, leukemic infiltration of the lungs develops ("leukemic pneumonitis"); in 1/4 cases – leukemic infiltration of the meninges (“leukemic meningitis”); pronounced hemorrhagic diathesis. Hemorrhages are observed in mucous and serous membranes, internal organs. Patients die from bleeding,

In recent years, active treatment of patients (cytostatic drugs, antibiotics) has significantly changed the picture of acute undifferentiated and myeloblastic leukemia. Multiple necrosis in the oral cavity and throat are rare, hemorrhagic diatheses have decreased. At the same time, as a result of the increase in life expectancy, such extraosseous changes as "leukemic pneumonitis" and "leukemic meningitis" are more common in patients with acute leukemia. In connection with the treatment of patients with cytostatic drugs, cases of necrotic-ulcerative changes in the gastrointestinal tract are more common.

Acute promyelocytic leukemia. This form of leukemia differs from other acute leukemias in the severity of the course, malignancy and significant hemorrhagic syndrome (thrombocytopenia, hypofibrinogenemia). Leukemic cells infiltrating organs and tissues are characterized by the following morphological features: nuclear and cellular polymorphism, accumulation of pseudopodia and glycosaminoglycan granules in the cytoplasm. Almost all patients with this form of leukemia die from brain hemorrhages or from gastrointestinal bleeding.

*Acute lymphoblastic leukemia*occurs much more often in children (80% of cases) than in adults. Leukemic infiltrates predominate in the bone marrow, lymph nodes, lymphatic apparatus of the gastrointestinal tract, spleen, kidneys, and thymus gland. Bone marrow of cancellous and tubular bones is crimson-red, juicy. The spleen is sharply enlarged, juicy, red. Lymph nodes are also significantly enlarged due to their infiltration by lymphoblastic cells; on dissection, they are white-pink, juicy. The thymus gland, which can reach gigantic sizes, has a similar appearance. Sometimes the leukemic infiltrate goes beyond the gland and spreads to the anterior mediastinum, compressing the organs of the chest cavity.

In this form of leukemia, leukemic infiltrates consist of lymphoblasts, the characteristic feature of which is the accumulation of glycogen around the nucleus. Lymphoblasts belong to the T-system of lymphopoiesis, which can explain both the rapid distribution of blasts in the T-dependent zones of the

lymph nodes and the spleen, as well as the increase in the size of the latter simultaneously with leukemic infiltration of the bone marrow. A sign of the progression of leukemia can be considered lymphoblastic infiltrates of metastatic origin outside the lymphatic tissue. Especially often, such infiltrates are found in the membranes and substance of the brain and spinal cord, which is called neuroleukosis.

Acute lymphoblastic leukemia can be treated with cytostatic drugs. In 90% of sick children, it is possible to obtain a stable long-term (5–10 years) remission. Without treatment, the course of this form, like other acute leukemias, progresses: anemia increases, hemorrhagic syndrome develops, complications of infectious origin appear.

Acute plasmablastic leukemia. This form of acute leukemia arises from precursor cells of B-lymphocytes capable of producing immunoglobulins; this ability is also preserved in tumor plasmablasts. Pathological immunoglobulins - paraproteins are formed and then secreted in them, therefore acute plasmablastic leukemia should be classified as paraproteinemic hemoblastoses. Plasmablastic leukemic infiltration is found in the bone marrow, spleen, lymph nodes, liver, skin; a significant number of plasmablasts is also found in peripheral blood.

Acute monoblastic (myelomonoblastic) leukemia is almost indistinguishable from acute myeloblastic leukemia.

*Acute erythromyeloblastic leukemia.*This is a rather rare form (1–3%) among all forms of acute leukemia, in which the growth of both erythroblasts and other nuclear cells of erythropoiesis, as well as myeloblasts, monoblasts, and undifferentiated blasts occurs in the bone marrow. Anemia, leuko- and thrombocytopenia occur as a result of inhibition of hematopoiesis; at the same time, the spleen and liver increase in size.

Acute megakaryoblastic leukemia. One of the rarest forms of acute leukemia, which is characterized by the presence in the blood and bone marrow of undifferentiated blasts as well as megakaryoblasts, distorted megakaryocytes and accumulations of platelets; the number of platelets in the blood increases to 1,000 - 1,500*109/l.

Chronic leukemias.

Chronic leukemias of myelocytic origin. Such forms of leukemia are diverse in origin and morphological changes, however, the main place among them is occupied by chronic myeloid leukemia, chronic erythromyelosis, erythremia and true polycythemia.

Chronic myeloid leukemia(chronic myelosis). This form of leukemia runs into two stages: monoclonal benign and polyclonal malignant. The first stage, which takes several years, is characterized by the progressive growth of neutrophilic leukocytes with a shift to myelocytes and myeloblasts, and an increase in the spleen. Bone marrow cells in this stage of leukemia do not differ morphologically and in their ability to phagocytosis from normal cells, however, they contain the so-called Ph-chromosome (Philadelphia), which arises as a result

of the deletion of chromosomes of the 22nd pair. In the second stage, which lasts from 3 to 6 months (terminal stage), monoclonality changes to polyclonality. As a result, blast forms appear (myeloblasts, less often erythroblasts, monoblasts and undifferentiated blast cells), the number of which increases both in the bone marrow and in the blood (blast crisis).

At the autopsy of those who died from chronic myeloid leukemia in the terminal stage, changes are found in the bone marrow, spleen, liver, lymph nodes, and blood. Bone marrow of flat bones, epiphyses and diaphyses of tubular bones is juicy, gray-red or gray-yellow purulent (pioid bone marrow). During histological examination, promyelocytes and myelocytes, as well as blast cells, are found in the bone marrow. There are cells with distorted nuclei and altered cytoplasm, phenomena of karyopyknosis and karyolysis. Reactive osteosclerosis is possible in bone tissue. Blood is gray-red; internal organs are anemic.

The spleen is sharply enlarged, sometimes occupying almost the entire abdominal cavity; its weight reaches 6–8 kg. At autopsy, it is dark red in color, sometimes with ischemic heart attacks. The tissue of the spleen is displaced by a leukemic infiltrate, mainly from cells of the myeloid series, among which blasts are visible; follicles are atrophied; sclerosis and hemosiderosis of the pulp are found. Leukemic thrombi are found in blood vessels.

The liver is significantly enlarged (its weight reaches 5–6 kg). The surface is smooth, the tissue is grey-brown on dissection. Leukemic infiltration prevails along the sinusoids, less often in the portal tracts and capsule. Fatty dystrophy in hepatocytes; sometimes hemosiderosis is possible.

Lymph nodes are significantly enlarged, soft, gray-red in color with leukemic infiltration. The same infiltration is observed in the tonsils, group and solitary lymphatic follicles, intestines, kidneys, skin, sometimes in the brain and meninges (neuroleukemia). A significant number of leukemic cells appear in the vessels, which form leukemic stasis and thrombi and infiltrate the vessel wall. Such changes in blood vessels can be the cause of heart attacks and hemorrhages. Manifestations of autoinfection are quite often found in chronic myeloid leukemia.

A group related to chronic myeloid leukemia consists of osteomyeloleukosis and myelofibrosis, in which, along with the signs of myeloid leukemia, bone marrow is replaced by bone or connective tissue. In such cases, the process is characterized by a long benign course.

Treatment of patients with cytostatic drugs changes the morphological manifestations of chronic myelogenous leukemia. Along with the suppression of leukemic infiltration centers and the development of fibrosis in their place, the rejuvenation of cellular forms, the appearance of metastatic centers and tumor growths or bone marrow aplasia and pancytopenia are noted.

Chronic erythromyelosis- a rather rare form of leukemia. This is a tumor of the red and white germ of the hematopoietic tissue, in which erythrokaryocytes, myelocytes, promyelocytes and blasts grow in the bone marrow, spleen and liver.

A significant part of these cells is also found in the peripheral blood. Severe splenomegaly. In some cases, myelofibrosis (Vagan's form of chronic erythromyelosis) joins.

Erythremia. This form of leukemia mostly occurs in the elderly and is characterized by an increase in the mass of erythrocytes in the peripheral blood, that is, a plethora. The number of platelets and granulocytes also increases, blood pressure rises, tendency to thrombosis, splenomegaly appear. All sprouts grow in the bone marrow, but mainly erythrocyte sprouts. The process is benign for a long time, but often ends with transformation into chronic myeloid leukemia with foci of leukemic infiltration in the organs.

All internal organs are full of blood with the formation of blood clots in both veins and arteries. Adipose bone marrow of tubular bones becomes red; the spleen increases sharply. There is hypertrophy of the heart, especially the left ventricle. In the spleen, kidneys, and liver in the early stage of erythremia, there are foci of extramedullary hematopoiesis with a significant number of megakaryocytes, and in the late stage, when the process transforms into myeloid leukemia, foci of leukemic infiltration appear.

Polycythemia vera(Vakez-Osler disease) is close to erythremia in many morphological features.

Chronic leukemias of lymphocytic origin. These forms of leukemia are divided into two groups: the first is chronic lymphocytic leukemia and skin lymphoma bordering on it (Sézary's disease); the second - paraproteinemic leukemias.

Chronic lymphocytic leukemia. It often occurs in middle-aged and elderly people, in some cases - in members of the same family; arises from B-lymphocytes and is characterized by a long benign course. The number of leukocytes in the blood increases sharply (up to 100*109/l), lymphocytes predominate among them. Leukemic infiltrates from tumor lymphocytes are most pronounced in the bone marrow, lymph nodes, spleen, and liver with successive enlargement of these organs. Tumor B-lymphocytes almost do not produce immunoglobulins. In this regard, with chronic lymphocytic leukemia, humoral immunity is sharply suppressed, patients often have complications of infectious origin. This form of leukemia is characterized by the development of autoimmune reactions, especially autoimmune hemolytic and thrombopenic states.

Against the background of a benign course of chronic lymphocytic leukemia, the following are possible: blast crisis, generalization of the process leading to death, but more often patients die from infectious diseases or complications of autoimmune origin.

At autopsy, morphological changes are found in the bone marrow, lymph nodes, spleen, liver, and kidneys.

The bone marrow of flat and tubular bones is red in color, however, unlike myeloid leukemia, there are yellow cells in the diaphyses of tubular bones among

the red bone marrow. During histological examination, tumor cells are found in the bone marrow. In extreme cases, the entire myeloid tissue of the bone marrow is squeezed out by leukemic lymphocytic infiltrates, and only minor islands of myeloid hematopoiesis remain.

Lymph nodes of all areas of the body are sharply enlarged and form large soft or dense packages. On dissection, they are juicy, white-pink in color. Tonsils, group and solitary lymphatic follicles of the intestine, which also represent a white-pink juicy tissue, increase. The increase in lymph nodes and formations is associated with their leukemic infiltration, which leads to a sharp violation of the structure of these organs and tissues; quite often, lymphoblasts infiltrate the capsule of the nodes, as well as the tissues adjacent to them.

The spleen reaches significant sizes, its mass increases to 1 kg. On autopsy, the tissue is red in color, fleshy in consistency; follicles are preserved or lost in the pulp. Leukemic lymphocytic infiltrates occur primarily in the follicles, which become enlarged and connect with each other. Later, lymphocytes grow in the red pulp, vessel walls, trabeculae, and capsule.

The liver is enlarged, dense; on dissection, it is light brown with small gray-white nodules on the surface. Leukemic lymphocytic infiltration occurs along the portal tracts. In hepatocytes - protein and fatty dystrophy.

The kidneys are large, dense, gray-brown. Leukemic infiltration so dramatically disrupts the structure of the kidneys that it is even impossible to distinguish its layers.

Leukemic infiltration involves many organs and tissues (myocardium, mediastinum, serous and mucous membranes). It is not only diffuse, but also focal, forming nodes of significant size.

Changes characteristic of chronic lymphocytic leukemia are supplemented by infectious complications, for example, pneumonia, as well as manifestations of hemolytic conditions - hemolytic jaundice, general hemosiderosis, and diapedesis hemorrhages.

It should be borne in mind that in addition to widespread damage to the lymph nodes, moderate enlargement of the spleen and liver in chronic lymphocytic leukemia, there are cases when only some groups of lymph nodes (mediastinum, mesentery, cervical, inguinal) are sharply enlarged. In such cases, they can squeeze nearby organs (heart, esophagus, trachea and bronchi; portal vein and its branches with the development of portal hypertension and ascites).

Lymphomatosis of the skin, or Sézary's disease. This is a peculiar form of chronic lymphocytic leukemia, which is characterized by infiltration of tumor T-lymphocytes, first of all, of the skin. The bone marrow is gradually involved in the pathological process, the number of leukocytes in the blood increases, specific cells (Sézary cells) appear; peripheral lymph nodes and spleen are enlarged.

Paraproteinemic leukemias. This group of leukemias includes tumors that develop from cells of the B-lymphocytic system (precursors of plasma cells),

with the function of which reactions of humoral immunity are connected. The main feature of paraproteinemic leukemias, which are also called malignant immunoproliferative diseases, is the ability of tumor cells to synthesize homogeneous immunoglobulins or their fragments - paraproteins (monoclonal immunoglobulins). Immunoglobulin pathology determines both the clinical and morphological characteristics of paraproteinemic leukemias, which include myeloma, primary macroglobulinemia (Waldenström), and heavy chain disease (Franklin).

*Myeloma disease*is a fairly common disease that was first described by O.O. Rusticy (1873) and Kaler (1887). With this disease, tumor cells of the lymphoplasmacytic line - myeloma cells grow both in the bone marrow and outside it. Myelomatosis of the bone marrow leads to the destruction of bones.

Depending on the type of cells that grow, plasmacytic, plasmablastic, polymorphic cell and small cell myeloma are distinguished. Polymorphic and small cell myeloma belong to poorly differentiated tumors. Myeloma cells secrete paraproteins, which are found in the blood and urine of patients, as well as in the myeloma cells themselves. In myeloma disease, different types of pathological immunoglobulins are biochemically isolated from blood serum and urine. There are several biochemical variants of myeloma (A-, D-, E-myeloma, Bence-Jones myeloma). Bence-Jones protein found in urine is a type of paraprotein produced by myeloma cells; it freely penetrates through the glomerular filter of the kidneys because it has a low molecular weight.

Myeloma is mostly of the aleukemic type, but sometimes the appearance of myeloma cells in the peripheral blood is possible.

During morphological examination, depending on the type of myeloma infiltrates that appear in the bone marrow and bones, diffuse, diffuse-nodular and multiple-nodular forms of myeloma disease are distinguished. The diffuse form of myeloma is said to occur when diffuse myeloma infiltration of the bone marrow is combined with osteoporosis. In the diffuse-nodular form, tumor nodes appear against the background of diffuse myelomatosis of the bone marrow, in the multiple-nodular form, there is no diffuse myeloma infiltration.

The growth of myeloma cells is more often observed in flat bones (skull bones, ribs) and spine, less often in tubular bones (shoulder, thigh), which is accompanied by destruction of bone tissue. In the growth centers of myeloma cells in the central channel of the osteon or in the bone beam under the endosteum, the bone substance becomes fine-grained, then thins out; osteoclasts appear in it, then the endosteum exfoliates. Gradually, the entire bone beam turns into a so-called liquid bone and completely dissolves; osteon channels become wide. "Axillary resorption" of bone develops, which explains the osteolysis and osteoporosis characteristic of myeloma disease - the formation of smooth-walled, as if stamped defects in the absence or insufficient bone formation. Bones become brittle, which can explain their frequent fractures in myeloma.

In addition to bone marrow and bones, myeloma infiltration is also observed in internal organs (spleen, liver, kidneys, lungs, lymph nodes).

Some changes in the body in myeloma disease are associated with the secretion of paraprotein by tumor cells. These include: 1) amyloidosis (AL-amyloidosis); 2) deposition of amyloid-like and crystalline substances in tissues; 3) the development of paraproteinemic edema or paraproteinosis of organs (paraproteinosis of the myocardium, lungs, paraproteinemic nephrosis), which is accompanied by their functional insufficiency. Among paraproteinemic changes, paraproteinemic nephrosis or myeloma nephropathy, which can be the cause of death in 1/3 of myeloma patients, is of great importance. At the heart of paraproteinemic nephrosis is the "clogging" of the kidneys by Bence-Jones paraprotein, which leads to sclerosis of the brain and then the cortical substance and shrinkage of the kidneys (myeloma shrunken kidneys). In some cases, paraproteinemic nephrosis is combined with kidney amyloidosis.

With myeloma disease, in connection with the accumulation of paraproteins in the blood, protein stasis in the vessels, a peculiar syndrome of increased viscosity and paraproteinemic coma develop.

In connection with the immunological defenselessness observed in patients with plasmacytoma, inflammatory changes (pneumonia, pyelonephritis) are possible, arising against the background of tissue paraproteinosis and being a manifestation of autoinfection.

Primary macroglobulinemia- a rare disease, which was first described by Waldenström in 1944. It is one of the types of chronic leukemia of lymphocytic origin, in which tumor cells produce and secrete pathological macroglobulin - IgM. In this disease, an increase in the spleen, liver, and lymph nodes is observed, which is associated with their leukemic infiltration; bone destruction is rare. A rather typical hemorrhagic syndrome, as a result of hyperproteinemia, increased blood viscosity, functional deficiency of platelets, slowing of blood flow and stasis in small vessels. Such complications as hemorrhages, paraproteinemic retinopathy, paraproteinemic coma, and possible amyloidosis occur quite often.

*Disease of heavy chains*described by Franklin in 1963. In this disease, tumor cells of the lymphoplasmacytic series produce a peculiar paraprotein corresponding to the Fc fragment of the IgG heavy chain (hence the name of the disease). In this disease, there is an increase in the lymph nodes, liver, and spleen as a result of their infiltration by tumor cells. Bones do not change, bone marrow damage is not necessary. Patients die from joining infectious diseases (sepsis) as a result of hypogammaglobulinemia.

Lymphomas- regional tumor diseases of hematopoietic and lymphatic tissue

This group of diseases includes: lymphosarcoma, mycosis fungoides, Sézary's disease, reticulosarcoma, lymphogranulomatosis (Hodgkin's disease).

By origin, lymphomas can be B-cell and T-cell; it is based on the classification of lymphomas proposed by Lucas and Collins. According to this

classification, B-cell lymphomas can be: small-cell (B), centrocytic, immunoblastic (B), plasmalymphopitar, and T-cell lymphomas - small-cell (T), from lymphocytes with twisted nuclei, immunoblastic (T), and also represented mycosis fungoides and Sézary's disease. In addition, unclassified lymphomas are also distinguished. According to this classification, both small cell and immunoblastic lymphomas can develop from either B or T cells. Only B-cells develop centrocytic and plasmalymphocytic lymphomas, and only T-cells develop lymphocyte lymphoma with twisted nuclei, mycosis fungoides, and Sézary's disease.

Lymphomas do not have any features compared to leukemias. It should be emphasized that in the conditions of modern treatment of patients with cytostatic drugs, some lymphomas (lymphosarcoma) quite often "complete" the terminal stage of leukemia. At the same time, they can "transform" into leukemia. The given data show that the division of tumors of the blood system into "diffuse" and "regional", which is necessary for determining the nosology, is quite conditional from the point of view of oncogenesis.

Each lymphoma has its own morphological features.

Lymphosarcoma- a malignant tumor that arises from cells of the lymphoid line. With this tumor, morphological changes occur in lymph nodes, mainly mediastinal and extra-abdominal, less often inguinal. Sometimes the tumor develops in the lymphatic tissue of the gastrointestinal tract, spleen and other organs. Initially, the tumor is limited to several lymph nodes; they increase sharply, are connected to each other in packages that squeeze the adjacent organs and tissues. The nodes are dense, gray-pink on autopsy, with areas of necrosis and hemorrhages. In the future, the generalization of the process occurs, that is, lymphogenic and hematogenous spread with the formation of multiple metastases both in the lymph nodes and in other organs - lungs, bones, skin. Tumor cells of the type B- or T-lymphocytes, prolymphocytes, lymphoblasts and immunoblasts grow in the lymph nodes.

Depending on this, the following histo(cyto)-logical variants of lymphoma are distinguished: lymphocytic, prolymphocytic, lymphoblastic, immunoblastic, lymphoplasmacytic, African lymphoma (Burkitt's tumor). Tumors consisting of mature lymphocytes and prolymphocytes are called lymphocytomas; from lymphoblasts and immunoblasts - lymphosarcomas.

African lymphoma or Burkitt's tumor deserve special attention among lymphosarcomas.

*Burkitt's tumor*is an endemic disease that occurs among the population of Equatorial Africa (Uganda, Nigeria, Guinea, Bissau); episodic cases are also possible in other countries. Children aged 4–8 years are more often sick; the tumor is localized in the upper or lower jaw, as well as in the ovaries; less often - in the kidneys, adrenal glands, lymph nodes. Quite often, the spread of the tumor to other organs is observed. The tumor consists of small lymphocyte-like cells, among which there are large macrophages with bright cytoplasm, which gives the

impression of a peculiar appearance of the "starry sky". The occurrence of African lymphoma is associated with a herpes-like virus that was found in the lymph nodes of patients. Virus-like inclusions are found in tumor lymphoblasts.

Fungal mycosis- a relatively benign T-cell lymphoma of the skin, which belongs to the so-called lymphomatoses of the skin. Multiple tumor nodules consist of proliferating large cells with a significant number of mitoses. Plasma cells, histiocytes, eosinophils, and fibroblasts are also found in the tumor infiltrate. Tumor nodes are soft, protrude above the surface of the skin, resemble the shape of a mushroom, and are easily covered with ulcers. Such nodes are found not only in the skin, but also in the mucous membranes, muscles, and internal organs. Previously, tumor development was associated with fungal mycelium invasion, hence the erroneous name of the disease.

Sézary's disease– T-lymphocytic lymphoma of the skin with leukemia; refers to skin lymphomas. Damage to the bone marrow, the appearance of tumor cells in the blood, which is observed in Sézary's disease, served as the basis for its attribution in some cases to chronic lymphocytic leukemia.

Lymphocytic infiltration of the skin ends with the formation of tumor nodes on the face, back, and legs. Atypical mononuclear cells with sickle-shaped nuclei - Sézary cells - are found in tumor infiltrates of the skin, bone marrow, and blood. Sometimes slight tumor infiltration of lymph nodes, spleen, kidneys, and liver is possible.

Reticulosarcoma- a malignant tumor consisting of reticular cells and histiocytes. The main histological distinction between reticulosarcoma and lymphosarcoma is the production by tumor cells of reticular fibers that wrap around reticulosarcoma cells.

Lymphogranulomatosis(Hodgkin's disease) is a chronic relapsing, rarely acute disease, in which the growth of the tumor occurs mainly in the lymph nodes.

Isolated and diffuse lymphogranulomatosis are distinguished by morphological features: In isolated (local) lymphogranulomatosis, pathological changes occur in one group of lymph nodes. More often it is cervical, mediastinal or extra-abdominal; less often - inguinal, which increase in size and grow together into packages. At first, they are soft, juicy, gray or gray-pink, on autopsy with an erased structure pattern. Later, the nodes become dense, dry, with centers of necrosis and sclerosis. The primary localization of the tumor is possible not only in the lymph nodes, but also in the spleen, liver, stomach, lungs, and skin. With widespread (generalized) lymphogranulomatosis, growth of tumor tissue is found not only in the centers of primary localization, but also far beyond them; at the same time, first of all, the spleen increases. At autopsy, the pulp is red with multiple white-yellow centers of necrosis and sclerosis; it acquires a mottled "porphyry" appearance ("porphyry spleen"). Some researchers explain the development of generalized lymphogranulomatosis by tumor metastasis from the primary tumor node.

During microscopic examination, proliferation of lymphocytes, histiocytes, and reticular cells, including giant cells, eosinophils, plasma cells, and neutrophilic leukocytes, are found both in the centers of primary localization of the tumor (lymph nodes) and in metastatic screenings. Proliferating polymorphic cellular elements form nodules that are subject to caseous necrosis and sclerosis. The most characteristic feature of lymphogranulomatosis is the proliferation of atypical cells, which include: 1) small Hodgkin cells (analogous to lymphoblasts); 2) mononuclear giant cells or large Hodgkin cells; 3) multinucleated cells of Reed–Berezovsky–Sternberg, which quite often acquire gigantic sizes. The origin of the latter cells is possibly lymphocytic, although their macrophage nature cannot be ruled out.

Lymphogranulomatous cells undergo a certain evolution, which reflects the progression of the tumor, while the cellular composition of the cells changes. Using biotic research (lymph nodes) it is possible to compare histological and clinical features of lymphogranulomatosis. Such comparisons formed the basis of modern clinical and morphological classifications of lymphogranulomatosis.

Clinical and morphological classification. There are four variants (stages) of the disease: 1) variants with a predominance of lymphoid tissue (lymphohistiocytic); 2) nodular (nodular) sclerosis; 3) mixed-cell variant; 4) variant with suppression of lymphoid tissue.

The variant with a predominance of lymphoid tissue is a manifestation of the early phase of the disease and its localized forms, which corresponds to the I-1I stages of the process. Microscopic examination reveals only the proliferation of mature lymphocytes and partially histiocytes, which erases the pattern of the lymph node. In cases of progression of the disease, the histiocytic variant turns into a mixed-cell variant.

Nodular (nodular) sclerosis is characteristic for a relatively benign course of the disease; and the process initially develops in the mediastinum. During microscopic examination, the growth of connective tissue is found, which surrounds cellular accumulations, among which Reed-Berezovsky-Sternberg cells are found, and on the periphery - lymphocytes and other cells.

The mixed-cell variant reflects the spread of the pathological process and corresponds to the I-III stages of the disease. Microscopically, characteristic signs are found: proliferation of lymphoid elements of various degrees of maturity, giant cells of Hodgkin and Reed–Berezovsky–Sternberg; accumulation of lymphocytes, eosinophils, plasma cells, neutrophilic leukocytes; centers of necrosis and fibrosis.

The variant with suppression (displacement) of lymphoid tissue occurs with an unfavorable course of the disease and reflects the generalization of lymphogranulomatosis. At the same time, in some cases, diffuse growths of connective tissue are observed, among the fibers of which there are single atypical cells; in others, lymphoid tissue is displaced by atypical cells, among

which Hodgkin cells and Reed–Berezovskyi–Sternberg giant cells predominate; sclerosis does not develop.

The variant with displacement of lymphoid tissue by extremely atypical cells was called Hodgkin's sarcoma. Thus, the spread of lymphogranulomatosis is morphologically reflected by a sequential change of its three variants: with the predominance of lymphoid tissue, mixed-cellular and with suppression of lymphoid tissue. Such clinical and anatomical options can be considered as stages of lymphogranulomatosis.

2.3. List of questions to check basic knowledge on the subject of the lesson.
1. Concept of hematopoietic tissue, functions, structure.
2. Hemoblastosis, classification. Syndromes that develop with hemoblastosis (tumor intoxication, leukemic proliferation syndrome, hemorrhagic syndrome, anemic syndrome, infectious lesions)
3. General properties of hemoblastoses (a. Systemic damage associated with early metastatic spread of tumor cells in hematopoietic organs; b. Suppression of normal hematopoiesis, and primarily the unit that is the source of tumor growth;
with. Rapid spread of leukemic cells; d. Clonal origin of hemoblastosis, that is, leukemic cells are descendants of one mutated cell (one type of cell);
e. Tumor progression, which consists in qualitative changes in the behavior and morphology of tumor cells, which leads to the development of polyclonality and the appearance of the most autonomous tumor clones.)
4. Causes of the development of hemoblastosis, factors contributing to their development.
5. Acute leukemia. Classification. Specifics. Morphological characteristics.
6. Chronic leukemia. Classification. Specifics. Morphological characteristics.
7. Lymphomas. Classification. Morphological characteristics of non-Hodgkin's lymphomas.
8. Lymphogranulomatosis. Morphological characteristics.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1. Test tasks

1. During the examination of a 35-year-old patient, a histological examination of the red bone marrow punctate was performed and a significant decrease in the number of megakaryocytes was found. What changes in peripheral blood will this lead to?
AND Thrombocytopenia
BLeukocytosis
Thrombocytosis
Agranulocytosis
Leukopenia

2. During the autopsy of a deceased 96-year-old man who suffered from atherosclerosis and died of massive blood loss, a ruptured aneurysm of the abdominal aorta was found. A large amount of clotted blood surrounds the aneurysm. What type of anemia develops with this pathology?:
AND Acute posthemorrhagic
B Chronic post-hemorrhagic
Pernicious
D Hemolytic
EHypoplastic
3. During the examination of a 58-year-old patient suffering from varicose veins with periodic bleeding from them, a decrease in the number of erythrocytes and hemoglobin was revealed. The skin and mucous membranes are pale. What type of anemia did the patient develop?:
AND Chronic posthemorrhagic
BAcute posthemorrhagic
Hemolytic
Pernicious
EHypoplastic
4. During the examination of the patient, pallor of the skin, yellowness of the sclera was noted. The tongue is smooth, shiny, as if polished. Microscopic examination of the stomach biopsy revealed a decrease in the glands in the fundal part of the stomach. The epithelium is atrophic, only the main cells are preserved. Macrocytes and megalocytes are detected in the blood smear. This picture is characteristic of:
AND Pernicious anemia
B Iron deficiency anemia
Hypoplastic anemia
Hemolytic anemia
Posthemorrhagic anemia
5. A blood test of a 43-year-old man who worked with radioactive isotopes at a research institute revealed anemia, leukopenia, and thrombocytopenia. In punctate bone marrow, replacement of bone marrow with fat is observed. What type of anemia is characterized by the following changes?:
AND Hypoplastic
B Hemolytic
Pernicious
Posthemorrhagic
Iron deficiency
6. A 44-year-old patient, who suffered from fibro-cavernous tuberculosis for a long time, started pulmonary bleeding with blood loss in the amount of 1 liter. What type of anemia occurs in this case?:
AND Acute posthemorrhagic
B Chronic post-hemorrhagic
Hemolytic

D Iron deficiency
Pernicious
7. During the examination of a 16-year-old boy, enlarged submandibular and cervical lymph nodes were found. A biopsy was performed. Microscopically, the following were found in the lymph nodes: the typical structure was erased, the cell population was heterogeneous, there were large cells with a multilobed nucleus, multiple large uninucleate cells, eosinophilic and neutrophilic leukocytes, lymphocytes, and areas of necrosis and foci of sclerosis were also found. What is the most likely diagnosis?
AL Imphogranulomatosis
B GHyperplasia of the lymph node
C Granulomatous limpadenitis
D Hncommon lymphadenitis
E Nehodzhklymphoma
8. During the autopsy of a 35-year-old woman, along with the enlargement of many lymph nodes, an enlarged spleen weighing 600.0 was found; on cross-section, it is heterogeneous, dark red in color, dense, with areas of grayish-yellow necrosis, up to 1 cm in diameter (porphyry spleen). What disease can you think of?
AND Lymphogranulomatosis
B Chronic lymphoid leukemia
Chronic myeloid leukemia
Cancer metastases
Lymphosarcoma
9. When examining a biopsy of enlarged cervical lymph nodes of a young woman, proliferation of lymphocytes, reticular cells, histiocytes, large and small Hodgkin cells, Berezovsky-Sternberg multinucleated cells, numerous eosinophils, single foci of case lymph node necrosis were revealed. What is the most likely diagnosis?
AND Lymphogranulomatosis
B Tuberculosis
Acute leukemia
D Lymphosarcoma
Metastasis of lung cancer
10. When examining a biopsy of an enlarged cervical lymph node, a rough pattern was found, its tissue was represented by a large number of proliferating lymphocytes with an admixture of single Berezovsky-Sternberg cells. These changes indicate such a disease:
AND Lymphogranulomatosis with predominance of lymphoid tissue
B Lymphogranulomatosis with depletion of lymphoid tissue
C Mixed cellular variant of lymphogranulomatosis
D Lymphosarcoma
E Nodular-sclerotic variant of lymphogranulomatosis
11. When examining a biopsy of an enlarged cervical lymph node, the pattern was blurred, its tissue was represented by a large number of proliferating lymphocytes with

an admixture of single Berezovsky-Sternberg cells. These changes indicate such a disease:
AND Lymphogranulomatosis with predominance of lymphoid tissue
B Lymphogranulomatosis with depletion of lymphoid tissue
C Mixed cellular variant of lymphogranulomatosis
D Lymphosarcoma
ENodular-sclerotic variant of lymphogranulomatosis
12. During the histological examination of an enlarged cervical lymph node, it was noted that the general pattern of the node is unclear due to the growth of atypical histiocytic cells, with the presence of Berezovsky-Sternberg giant cells, areas of necrosis, sclerosis, and hyalinosis. What disease is characterized by morphological changes in the lymph node?
AND Lymphogranulomatosis
B Acute myeloid leukemia
Chronic myelogenous leukemia
D Fungal mycosis
Tuberculosis

3.2. algorithm for describing a macropreparation and a micropreparation
Description of macropreparation:
22. Specify the name of the organ or ego part;
23. Specify the dimensions of the body (length, width, thickness);
24. Specify the surface of the organ, the condition of the capsule, overlap;
25. Specify the consistency of the organ;
26. The type and structure of the organ at autopsy;
27. Indicate the presence of a pathological formation (if any);
28. Conclude.

Description of the micropreparation:
1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:
- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:
- methods: assessment of the correctness of the performance of practical skills
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Atherosclerosis and arteriosclerosis. Coronary heart disease".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

10. http://moz.gov.ua- Ministry of Health of Ukraine
11. www.ama-assn.org– American Medical Association /American Medical Association
12. www.who.int- World Health Organization
13. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
14. http://bma.org.uk– British Medical Association
15. www.gmc-uk.org- General Medical Council (GMC)
16. www.bundesaerztekammer.de– German Medical Association
17. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
18. http://www.webpathology.com/- Web Pathology

Practical lesson No. 20

Topic:Atherosclerosis and arteriosclerosis. Coronary heart disease.

Goal: To learn to determine the etiology, pathogenesis, morphology of coronary disease. Distinguish clinical and morphological forms of coronary disease, as well as their complications.

Basic concepts:atherosclerosis, coronary heart disease, etiology and pathogenesis, clinical and morphological forms,pathological anatomy of individual stages of atherosclerosis and coronary disease,consequences and complications, interpret these morphological changes.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Atherosclerosis(atherosclerosis, Greek aterr – porridge + sclerosis – compaction) is a stromal-vascular lipoprotein dystrophy of arteries of mainly elastic and elastic-muscular types.

Macroscopic manifestations of atherosclerosis

9. grease spots and bands,
10. fibrotic plaques,
11. ulcers of fibrous plaques,
12. calcification.

Microscopic signs of atherosclerosis

13. stages are distinguished histologically:
14. dolipid,
15. lipoidosis,
16. liposclerosis,
17. atheromatosis and
18. atherocalcinosis.

Clinical and morphological forms of atherosclerosis

8. atherosclerosis of the aorta,
9. atherosclerosis of the coronary vessels of the heart,
10. atherosclerosis of cerebral arteries,
11. atherosclerosis of renal arteries,
12. atherosclerosis of mesenteric arteries,
13. atherosclerosis of the arteries of the lower extremities and
14. atherosclerosis of the arteries of the small circulatory circle

CORONARY HEART DISEASE- a group of diseases of the heart muscle, the basis of which is a violation of the function of the myocardium, which is caused by a discrepancy between its oxygen supply and its needs in the case of absolute or relative insufficiency of coronary blood supply.

According to the recommendations of the VI National Congress of Cardiologists of Ukraine (2000), clinical forms of coronary heart disease include:

14. sudden coronary death,
15. angina pectoris
16. acute myocardial infarction,
17. cardiosclerosis,
18. painless form.

Causes of death in myocardial infarction

19. The immediate causes of death in the early period of a heart attack are:
20. cardiogenic shock,
21. ventricular fibrillation,
22. asystole,
23. acute heart failure.
24. Over time, rupture of the heart and thromboembolism of cerebral vessels take first place.

With chronic ischemic heart disease, death occurs from heart failure, thromboembolic complications, and rupture of the aneurysm wall.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Definition of atherosclerosis, etiology.
2. Pathogenesis of atherosclerosis.
3. Microscopic manifestations of atherosclerosis, stages, morphological characteristics.
4. Macroscopic manifestations of atherosclerosis, stages, morphological characteristics.
5. Clinico-morphological forms of atherosclerosis.
6. Morphological changes in atherosclerosis of cerebral vessels, complications, outcomes.
7. Morphological changes in atherosclerosis of the aorta, complications, causes of death.
8. Morphological changes in atherosclerosis of mesenteric vessels, complications, causes of death.
9. Morphological changes in atherosclerosis of kidney vessels, complications, causes of death.
10. Morphological changes in atherosclerosis of leg vessels, complications, causes of death.
11. Morphological changes in atherosclerosis of the coronary arteries of the heart, complications, causes of death.
12. Definition of ischemic heart disease, etiology.
13. Pathogenesis of ischemic heart disease.
14. Clinical and morphological forms of coronary artery disease.
15. Morphological (macro-microscopic) changes in gosroic coronary artery disease.
16. Complications and outcomes of acute coronary artery disease.
17. Morphological changes (macro-microscopic) in chronic coronary artery disease.
18. Complications and outcomes in chronic coronary artery disease.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

In the deceased with atherosclerosis of the cerebral vessels, an autopsy revealed a focus in the left hemisphere of the brain, which was represented by a flaccid, structureless grayish-yellowish tissue with indistinct edges. What pathological process is it about?

Correct answer Ischemic stroke

BMultifocal tumor growth with cystic transformation

Countless foci of fresh and old hemorrhages in the brain
Focal encephalitis
Senile encephalopathy
During a histological examination of the lungs of a person who died from heart failure, foci of inflammation were found with alveoli filled with pale pink liquid, in places with the presence of thin pink threads, which form a small looped mesh with a small number of lymphocytes. What is the nature of the exudate in the lungs?
Correct answer Serous-fibrinous
B Hemorrhagic
Serous
Purulent
EFibrinous
During the autopsy of a 70-year-old man who suffered from atherosclerosis for a long time and died of cardiovascular insufficiency, dense, oval-shaped fibrous plaques with lime deposits in the form of dense, brittle plates were observed in the abdominal department of the aorta under macroscopic examination. Specify the stage of atherosclerosis morphogenesis:
Correct answer Atherocalcinosis
Liposclerosis
Atheromatosis
D Ulceration
Lipoidosis
During the microscopic examination of the coronary artery in the 53-year-old deceased, a narrowing of the lumen of the vessel due to a fibrous plaque with an admixture of lipids was found. The most benign form of atherosclerosis:
Correct answer Liposclerosis
Lipoidosis
Dolipidna
Atheromatosis
E Ulceration
During the autopsy of a 70-year-old man who died of heart failure, deformed, narrowed coronary arteries were found. On cross-section, the inner surface of the arteries is bumpy, the wall is whitish, brittle, stony density. What is the stage of atherosclerosis?
Correct answer Atherocalcinosis
Liposclerosis
Atheromatosis
Lipoidosis
E Ulceration
In a 75-year-old patient who suffered from widespread atherosclerosis and died of chronic heart failure, the autopsy revealed narrowed and deformed coronary arteries, a bumpy intima on a section of white color, stony. Name the stage of atherosclerosis morphogenesis:

Correct answer Atherocalcinosis
Lipoidosis
Liposclerosis
D Dolipid stage
Stage of atheromatosis
At the autopsy of the deceased, who suffered from hypertensive disease for a long time, the pathologist found that the kidneys were sharply reduced in size, dense, their surface was uniformly fine-grained, and on cross-section, the parenchyma, especially the cortical substance, was uniformly rounded. He came to the conclusion that it is:
Correct answer Arteriosclerotic shrunken kidney
B Atherosclerotic shrunken kidney
CPyelonephritic shriveled kidney
DAmyloid shrunken kidney
E-
A 73-year-old man, who had been suffering from essential hypertension for 22 years, died of chronic kidney failure. At the autopsy of the deceased's body: the kidneys are halved in size, dense, with a fine-grained surface, with a significantly thinned cortical substance. What kidney pathology was detected?
Correct answer Arteriosclerotic nephrocirrhosis
B Chronic glomerulonephritis
Atherosclerotic nephrocirrhosis
Pyelonephritic nephrocirrhosis
Amyloidosis shriveled kidneys

A microscopic examination of the coronary artery of a 53-year-old deceased revealed a narrowing of the vessel lumen due to a fibrous plaque with an admixture of lipids. The most likely form of atherosclerosis:
Correct answer Liposclerosis
B Ulcer
C Dolipidna
Lipoidosis
EAtheromatosis
On the autopsy of the dead man, 73 years old, who suffered from coronary heart disease with heart failure for a long time, the following were found: "muscat" liver, purple induration of the lungs, cyanotic induration of the kidneys and spleen. Indicate which of the types of circulatory disorders is most likely?
Correct answer Chronic general venous anemia
BChronic anemia
CAcute general venous congestion
Arterial hyperemia
E Acute anemia
A 44-year-old man who developed a myocardial infarction died of left ventricular failure. An autopsy revealed: pulmonary edema, small hemorrhages in the serous and

mucous membranes. Microscopically: dystrophic and necrobiotic changes in the epithelium of the proximal tubules of the kidneys, centrilobular hemorrhages and foci of necrosis in the liver. Which of the types of circulatory disorders is most likely?
Correct answer Acute general venous congestion
Arterial hyperemia
Chronic general venous congestion
DAcute anemia
EChronic anemia
A 45-year-old man who died of sudden cardiac arrest was found to have a symmetrical type of obesity of the III degree, a rupture of the wall of the right ventricle with a hemopericardium, excess fat deposits under the epicardium. Microscopically: adipose tissue from the epicardium spreads to the myocardium with atrophy of muscle fibers. What is the most likely diagnosis?
Correct answer Acute myocardial infarction
BIschemic heart disease
Hypertensive disease
Heart obesity
Fatty dystrophy of the myocardium
At the autopsy of a 70-year-old man who died of cardiovascular insufficiency, chronic venous perfusion of the organs, hypertrophy of the left ventricle of the heart with small-focal cardiosclerosis, voluminous yellowish-whitish plaques in the intima of the aorta with pulp-like masses in the center, which sink into the thickness, were found walls What pathological process is most likely in the aorta?
Correct answer Atheromatosis
Liposclerosis
Calcinosis
Arteriosclerosis
Lipoidosis
At the autopsy of the corpse of a 60-year-old man in the myocardium of the front wall of the left ventricle of the heart, an irregularly shaped dense focus of gray color 5 x 4 cm with clear edges of the fibrous structure was found. What is the most likely diagnosis?
Correct answer Postinfarction myocardiosclerosis
BCardiomyopathy
Diffuse small focal myocardiosclerosis
Heart attack
EMyocarditis
A 65-year-old patient suffering from atherosclerosis was hospitalized in the surgical department due to spilled purulent peritonitis. Thrombosis of the mesenteric arteries was diagnosed during the operation. What is the most likely cause of peritonitis?
Correct answer Ischemic heart attack
BHemorrhagic heart attack
Compression ischemia

Angiospastic ischemia
Stasis
At the autopsy of the corpse of a 60-year-old man, a gray, irregularly shaped dense focus of 5 x 4 cm with clear edges and a fibrous structure was found in the myocardium of the front wall of the left ventricle of the heart. What is the most likely diagnosis?
Correct answer Postinfarction myocardiosclerosis
BCardiomyopathy
Diffuse small focal cardiosclerosis
Heart attack
EMyocarditis
During the autopsy of the corpse of a 67-year-old woman with coronary heart disease, a large, irregularly shaped white focus of dense consistency was found in the front wall of the left ventricle. What type of chronic coronary heart disease is characterized by these changes?
Correct answer Post-infarction (focal) cardiosclerosis
BAcute myocardial infarction
Central myocardial dystrophy
D Chronic aneurysm of the heart
ESmall focal cardiosclerosis
When examining the amputated lower limb of a patient suffering from atherosclerosis, it was found that the tissues of the foot are dry, dense, black, and the borders with normal tissues are clear. What complication of atherosclerosis developed in the patient.
Correct answer Dry gangrene of the foot
BStenotic atherosclerosis
Thrombosis of the deep veins of the lower leg
DMesenteric form of atherosclerosis
Calcinosis of the aorta

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

8. Specify the name of the organ or ego part;
9. Specify the dimensions of the body (length, width, thickness);
10. Specify the surface of the organ, the condition of the capsule, overlap;
11. Specify the consistency of the organ;
12. The type and structure of the organ at autopsy;
13. Indicate the presence of a pathological formation (if any);
14. Conclude.

Description of the micropreparation:

1. Specify the name of the body;

2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

4. methods: assessment of the correctness of the performance of practical skills
5. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical

knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.
The topic of the next lesson is emphasized: "Hypertension and arteriolosclerosis. Hypertensive disease and symptomatic arterial hypertension. Cerebrovascular disease.".
Suggested topics for essays:
1. Atherosclerosis. Morphological characteristics.
2. Ischemic heart disease. Morphological characteristics.
5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

19. http://moz.gov.ua- Ministry of Health of Ukraine
20. www.ama-assn.org– American Medical Association /American Medical Association
21. www.who.int- World Health Organization
22. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
23. http://bma.org.uk– British Medical Association
24. www.gmc-uk.org- General Medical Council (GMC)
25. www.bundesaerztekammer.de– German Medical Association
26. http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
27. http://www.webpathology.com/- Web Pathology

Practical lesson No. 21

Topic: Hypertension and arteriolosclerosis. Hypertensive disease and symptomatic arterial hypertension.

Goal: learn to determine the etiology, pathogenesis, morphology of hypertensive disease. Distinguish clinical and morphological forms of hypertension, as well as their complications.

Basic concepts:hypertension, arteriosclerosis, hypertension, cerebrovascular disease,etiology, pathogenesis, clinical and morphological stages andpathological anatomy of individual stages of hypertensive disease,consequences and complications, interpret these morphological changes.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Hypertensive disease isdisease (primary arterial hypertension) is a chronic disease, the basis of which is a long-term and persistent increase in blood pressure.

3 stages of hypertension:

I - preclinical (transient) or functional;

II - stage of morphological changes in small arteries and arterioles;

III - stage of secondary morphological changes in internal organs (due to ischemia or due to hemorrhage).

Clinical and anatomical forms of hypertension: cerebral, cardiac, renal. Causes of death in hypertension:

Such complications as:

- brain strokes,
- myocardial infarction,
- uremia (chronic renal failure with primary shrunken kidney),
- acute renal failure in the malignant course of hypertensive disease.

Theories of the pathogenesis of hypertension.

Hypertension is a syndrome of increased blood pressure. Hypertension can be vasoconstrictor (with spasm or hyalinosis of vascular walls) and hypervolemic (with the connection of sodium aldosterone mechanism and an increase in the volume of circulating blood - BCC).

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Definitionsymptomatic arterial hypertension, etiology.
2. Definitionhypertensive disease, etiology, its manifestations.
3. Pathogenesis of hypertension.
4. Clinical and morphological forms of hypertension. Morphological characteristics of the malignant form of hypertension.
5. Clinical and morphological stages of benign hypertension.
6. Microscopic changes in blood vessels in hypertensive disease.
7. Macroscopic changes in internal organs in hypertensive disease.
8. Complications and outcomes of hypertension.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

A 68-year-old woman who suffered from hypertension for a long time (blood pressure up to 220/110 mm Hg) was admitted to the clinic with complaints of a sharp headache and movement disorders in the left limbs. After 5 hours she died. At autopsy, in the right hemisphere of the brain, there is a cavity with uneven edges, filled with dark red, elastic blood clots. What circulatory disorder developed in the brain?

Correct answer Hematoma

B Hemorrhagic infiltration

CPetechiae

D Local venous congestion

Thrombosis

The patient suffered from hypertension for many years in the past. Death came from acute kidney failure. Name the characteristic changes in the kidneys found at autopsy?

Correct answer Arteriosclerotic nephrosclerosis

Arteriolonecrosis

C Renal infarction

D. Malignant hypertension

Athero-arteriosclerotic nephrosclerosis

Name the changes characteristic of the kidneys, found at the autopsy of a 45-year-old man who suffered from severe hypertension (BP 240/130 mm Hg) and died in the presence of chronic renal failure:

Correct answer The kidneys are small, dense, the surface is fine-grained

BLarge white kidneys

CLarge variegated kidney

DLarge sebaceous kidney

ECystic kidney
A 30-year-old man turned to a doctor with complaints of heart pain, palpitations, and shortness of breath upon slight physical exertion. During the last 5 years, a significant increase in blood pressure was noted. An adenoma of the adrenal cortex was revealed during the examination. Your diagnosis?
Correct answer Symptomatic hypertension
BHypertensive disease 1st stage
Hypertensive disease 2nd stage
Eccentric hypertrophy of the myocardium
Arteriosclerotic nephrosclerosis
During the autopsy of the corpse of a 72-year-old man who suffered from hypertensive disease and died due to increasing symptoms of autointoxication due to uremia, kidneys were found measuring 6.5x2.5x2 cm, weighing 70.0 g. They were dense, their surface was fine-grained, the parenchyma, especially the cortical substance , uniformly thinned. Under microscopic examination, most nephrons are replaced by connective tissue. What is the name of the state of auto-intoxication in this case?
Correct answer Chronic renal failure (azotemic uremia)
BHypertensive crisis
Acute renal failure
Benign hypertension
Malignant hypertension
The 68-year-old patient suffered from hypertension for a long time and died during another sharp rise in blood pressure. At the autopsy, a massive hematoma in the subcortical nuclei on the right, numerous small hemorrhages and a "rusty" cyst in the occipital region were found in the brain. Your diagnosis?
Correct answer The 3rd stage of hypertensive disease during a crisis
BIschemic cerebral infarction
C1st stage of hypertensive disease, hypertensive crisis
Diapedic hemorrhages
EHemorrhagic cerebral infarction
At the autopsy, the deceased, who suffered from hypertension for a long time, had a transmural myocardial infarction in the past and died of chronic cardiovascular failure. It was established: the kidneys are small, dense, the heart is hypertrophied, the cavities are enlarged. Your diagnosis?
Correct answer Hypertensive disease of the 3rd stage
BAtherosclerosis, cardiac form
Hypertensive disease, pre-clinical stage
Hypertensive disease of the 2nd stage
ESymptomatic hypertension
The autopsy of the deceased from heart failure revealed an enlarged heart weighing 550 g, fibrinous pericarditis, as well as shrunken dense kidneys weighing 50 g with a fine-grained surface and pronounced hyalinosis of arterioles and glomeruli. Name the main disease:

Correct answer Hypertensive disease
Atherosclerosis
Rheumatism
Pericarditis
Cardiomyopathy
A microscopic examination of the tissues of the organs of the deceased, who suffered from hypertension, revealed widespread hyalinosis of the arterioles of the kidneys, brain, intestines, elastofibrosis and atherosclerosis in the elastic and muscular arteries. For which stage of hypertensive disease are these changes characteristic?
Correct answer Hypertensive disease of the 2nd century.
BHypertensive disease 1 st.
Preclinical
Hypertensive disease of the 3rd century.
Hypertensive disease with secondary changes
A 55-year-old man died of a massive brain hemorrhage that occurred as a result of hypertension. The autopsy revealed typical pathomorphological signs of arterial hypertension. Damage to which vessels is a typical sign of hypertension?
Correct answer Arteriole
Artery of muscular type
Artery of elastic type
Artery of the elastic-muscular type
Large veins
An autopsy of a man who died of cardiac decompensation revealed a sharply enlarged heart weighing 960 g ("bull's heart"), with 90 ml of straw-colored liquid in the pericardial cavity. The cavities of the heart are sharply enlarged, the myocardium is flaccid, the thickness of the wall of the left ventricle is 2.3 cm. The kidneys are reduced in size, weighing 70 g each, and their surface is fine-grained. During the microscopic examination of internal organs, hyalinosis of arterioles and hypertrophy of the muscular layer of larger arteries were revealed. Name the main disease:
Correct answer Hypertensive disease
Ischemic heart disease
Cardiomyopathy
Atherosclerosis
Rheumatism
A 52-year-old patient with a persistent increase in blood pressure up to 200/110 mm Hg. died of chronic cardiovascular insufficiency. At autopsy: the weight of the heart was 600 g, the thickness of the wall of the left ventricle was 2.2 cm, the heart cavity was enlarged. Histologically pronounced hyalinosis and sclerosis of myocardial arterioles. For which disease are the described changes characteristic?
Correct answer Hypertensive disease
Atherosclerosis of heart arteries
CDilated cardiomyopathy
Hypertrophic cardiomyopathy

Endomyocardial fibrosis

.

A 67-year-old patient suffered from hypertension for 20 years. He died of chronic kidney failure. What did the kidneys look like when the body was dissected?

Correct answer Small, dense, fine-grained surface

BLarge variegated

Big red ones

DBig whites

Large with multiple thin-walled cysts

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

6. methods: assessment of the correctness of the performance of practical skills
7. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Ratin	**Evaluation criteria**

g	
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Systemic connective tissue diseases with autoimmunization: rheumatism, systemic lupus erythematosus, rheumatoid arthritis, systemic scleroderma, dermatomyositis, Bekhterev's disease.".

Suggested topics for essays:

1. Hypertensive disease. Morphological characteristics.
2. Cerebrovascular disease. Morphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 22

Topic: Cerebrovascular disease. Alzheimer's disease. Multiple sclerosis. Amyotrophic lateral sclerosis. Postresuscitation encephalopathy. Diseases of the peripheral nervous system.

Goal: To study pathomorphological signs, morphological classification, morphological characteristics of the most important diseases of the nervous system, which are associated with impaired cerebral blood circulation.

Basic concepts:cerebrovascular disease,etiology, pathogenesis, clinical and morphological stages andpathological anatomy of certain CNS diseases,consequences and complications, interpret these morphological changes.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

*Cerebrovascular disease*in the form of cerebral apoplexy, or stroke (insultus — stroke), is a sudden cerebrovascular blood circulation disorder. It is the cause of death in about 10% of people who die from all known diseases. Among people who survive a stroke, about 15% lose their ability to work. The incidence increases with age, and almost 80% of patients are older than 65 years. Moreover, at least 84% of people with cerebrovascular disease suffer from brain infarctions (in 53% of them, infarcts occur as a result of thrombosis and in 31% - as a result of embolism). The last 16% of patients have various forms of hemorrhages (in 10% of them, spontaneous hemorrhage develops, and in 6%, hemorrhage occurs due to the rupture of a vascular aneurysm). A distinction is made between a temporary (transient) attack of ischemia and a complete, as a rule, acute violation of cerebral circulation. A transient ischemic attack is a completely reversible neurological disorder that lasts from a few minutes to (very rarely) 1 day, during which no structural damage to the brain occurs. In case of an acute violation of cerebral blood circulation, on the contrary, there is a pronounced damage to the brain tissue. There are many risk factors for the development of acute cerebrovascular accident. Atherosclerotic plaques in cerebral arteries and hypertension (in particular, hypertension) play a dominant role. Among other factors, it is worth mentioning: disorders of plasma lipid content, diabetes, atherosclerosis of the coronary arteries of the heart, heart failure, as well as atrial fibrillation. A long-term smoking habit, obesity, certain dietary features, and alcoholism can also be of great etiological significance. Factors that attract cerebral hemorrhage are hypertension,
Brain infarction (ischemic stroke). As inin other organs, the damage appears as a result of a local delay or a significant decrease in the blood supply of the brain substance and is a zone of necrosis, either small in size and clearly limited, or the organ occupies most of it. A heart attack can occur in any part of the brain, but the most common area of damage is the pool of the middle cerebral artery. Only a part of the specified zone or its entire territory may be affected here. Often, a few days before an ischemic stroke, dizziness, headache, general weakness, numbness in an arm or leg are noted. The onset develops acutely: the limbs on one side of the body cease to function, speech disorders appear. With the development of right-sided hemiplegia (paralysis of the muscles of one half of the body), such violations occur. have a stable character. Consciousness is usually preserved.
Let's take a look at the three most common causes of cerebral infarction: thromboembolism in the vessels of the brain, thrombosis and stenosing atherosclerosis of the cerebral arteries, lesions of the cerebral arterial network of a local inflammatory or systemic nature. The sources of cerebral thromboembolism can be wall thrombi in infectious endocarditis (vegetation), myocardial infarction, severe arrhythmia, non-

bacterial thromboendocarditis in persons with cachexia and severe chronic diseases. Brain damage due to embolism can also complicate open heart or coronary artery bypass surgery. Another source of thromboembolism can be an atherosclerotic plaque covered with ulcers in the aorta or neck arteries.

As for thrombosis and atherosclerotic plaques, it's closealong with the cerebral arteries, they often affect the internal carotid and vertebral arteries. Atherosclerotic plaques in cerebral arteries tend to associate with plaques in many other vessels, including the arteries of the extremities. Stenosis caused by these plaques does not necessarily lead to a cerebral infarction, because at normal blood pressure, the caliber of the artery must decrease by 90% before a decrease in blood supply occurs.

However, in many cases, cerebral infarction is the result of a combination of systemic circulatory failure and atherosclerotic stenosis of the neck and/or brain arteries. It can also develop as a result of occlusion (clogging) of intracranial or cervical arteries. The most common site of thrombotic occlusion is the middle cerebral artery. Atherosclerotic narrowing or occlusions can be found in any part of the carotid and vertebral arteries. The most common area of damage is the place where the internal carotid artery begins. However, when the specified section of the artery is blocked, a heart attack will develop only if the blood circulation through the collateral pathways is insufficient. In some patients, the thrombus spreads along the internal carotid artery into the middle and anterior cerebral arteries. In this case, a large part of the cerebral hemisphere is exposed to a heart attack.

In addition to all of the above, lesions of an inflammatory nature or a systemic nature play a certain role in the pathogenesis of cerebral infarctions. We are talking about periarteritis nodosa, systemic lupus erythematosus and giant cell arteritis. Cerebrovascular accidents can complicate a number of diseases and conditions of completely different etiology: polycythemia vera and sickle cell anemia, pregnancy and the postpartum period, and the use of some oral contraceptives.

*Brain infarction*can be red (hemorrhagic) and white (pale, ischemic). With a pronounced hemorrhagic component, the lesion resembles a hematoma, but the difference is the preservation of the general architecture of the affected tissue. White heart attack (white or gray softening of the brain) is difficult to determine macroscopically if the process is less than 1 day old. At later times, the dead tissue has a loose consistency, looks swollen, and can stick to the blade of a knife. Internal and external hydrocephalus often develop. The boundary between gray and white matter in the area of such a lesion is usually absent. Under the microscope, ischemic necrosis of neurons, pallor of myelin staining, and sometimes accumulation of leukocytes around the dead vessels are determined. If the damage is extensive, swelling of the dead tissue and swelling of the surrounding brain matter can lead to an increase in intracranial pressure. Within a few days, the tissue in the infarct zone becomes even more moist and susceptible to decay. At this stage, under the microscope, you can see many macrophages ("granular balls") filled with fat granules and other inclusions, which are products of the breakdown of myelin and other components of brain tissue. Enlarged astrocytes and proliferation of capillaries are determined around the infarct zone.

During the following weeks, the dead tissue (or rather, the products of its decay) is rejected and gliosis develops. Ultimately, at the site of the infarct, the tissue shrinks along the borders of the infarct and a cyst is formed. Sometimes such a cyst is crossed by small blood vessels and glial fibers. If the heart attack was red (hemorrhagic), then many macrophages absorb hemosiderin, the masses of which, lying inside and outside the cells, give the forming walls of the cyst a brown color. Shrinkage of the tissue along the borders of the infarction is usually accompanied by expansion of the lateral ventricle on the side of the lesion. In addition to the shrinkage of the brain tissue and the formation of a cyst, the consequences of a brain infarction include the Wallerian degeneration of those nerve fibers that were destroyed in the area of damage. If the infarct touches the internal capsule, progressive degeneration and shrinkage of the tissue of the corresponding pyramidal pathway in the brain stem and spinal cord are noted. In addition to the shrinkage of the brain tissue and the formation of a cyst, the consequences of a brain infarction include the Wallerian degeneration of those nerve fibers that were destroyed in the area of damage. If the infarct touches the internal capsule, progressive degeneration and shrinkage of the tissue of the corresponding pyramidal pathway in the brain stem and spinal cord are noted. In addition to the shrinkage of the brain tissue and the formation of a cyst, the consequences of a brain infarction include the Wallerian degeneration of those nerve fibers that were destroyed in the area of damage. If the infarct touches the internal capsule, progressive degeneration and shrinkage of the tissue of the corresponding pyramidal pathway in the brain stem and spinal cord are noted.

Selective necrosis of neurons (dyscirculatory or ischemic encephalopathy). Neurons need a constant and adequate supply of oxygen and glucose.Oxygen supply largely depends on the function of the lungs and the level of cerebral circulation. The latter, in turn, is related to the perfusion pressure, the value of which is the difference between the parameters of systemic (arterial) pressure and venous cerebral pressure. Blood circulation in the brain is controlled by self-regulating mechanisms that maintain the relative constancy of incoming blood volumes, despite changes in perfusion pressure. In other words, blood circulation remains within normal limits even if systemic blood pressure drops to 50 mmHg. But with systemic pressure below the specified value, the degree of blood supply to the brain drops very quickly. Cerebral blood circulation decreases during cardiac arrest or an attack of hypotension. In the first case, as a rule, diffuse damage to the brain is noted, in the second - focal damage.

D a m a g e brain during cardiac arrest. Many patients with severe diffuse cerebral lesions resulting from cardiac arrest die within a few days. Brain damage is usually limited to selective neuronal necrosis (a necrotic process affecting only neurons), while most patients do not have an overt infarction. In people who survive within 12 hours after a cardiac arrest, widespread and pronounced necrosis of neurons is determined under a microscope. Due to the selective sensitivity of groups of neurons to hypoxia, necrosis is most pronounced in the hippocampus, the third, fifth and sixth layers of the cerebral cortex (in particular, in the furrows of the posterior halves of both hemispheres), some basal nuclei of pear-shaped neurocytes of the cerebellum (Purkinje

cells). After a few days, the dead neurons disappear and an intense reaction is observed on the part of astrocytes, microglia and capillaries. Similar changes occur with carbon monoxide poisoning, severe forms of epilepsy, and hypoglycemia.

H y p o t e n s i v e brain damage They mainly affect the border zones between the arterial basins of the cerebrum and cerebellum. In the parietal-occipital regions, where the basins of the front, middle and back cerebral arteries meet, heart attacks show a tendency to larger volumes of damage. It is possible to involve the basal nuclei in the area of such a lesion, in particular the head of the caudate nucleus (adjacent to the lateral ventricle and separated by the internal capsule from the lenticular nucleus and the thalamus) and the upper third of the shell (putamen) of the lenticular nucleus. The hippocampus, despite its extreme sensitivity to ischemia during cardiac arrest, remains intact. Hypotensive lesions of the brain occur mainly with a sudden drop in blood pressure, after which the pressure quickly returns to normal. Due to a sharp drop in blood increased pressure, the self-regulation of cerebral blood circulation is disturbed. Areas most removed from the main arterial trunks experience the greatest insufficiency of blood supply. Numerous examples of brain lesions are known, which develop in connection with major surgical operations under general anesthesia, as well as with myocardial infarction or severe hemorrhages.

Spontaneous intracranial hemorrhage. The most common optionsisintracerebral hemorrhage in hypertension and subarachnoid hemorrhage in arterial aneurysm rupture.

I n t r a c e r e b r a l hemorrhage (hemorrhagic stroke, cerebral apoplexy). A huge number of intracranial hematomas develop in old age in people suffering from hypertension due to the rupture of one of the numerous microaneurysms. At this time, it is considered established that such small aneurysms are formed in the vascular network of the brain in most people with hypertension. The most frequent localizations of hypertensive intracerebral hemorrhages are the zones of the basal nuclei and the internal capsule, then - the bridge of the brain (Varoli's bridge) and the cerebellum. Usually, the hematoma quickly increases in volume, leads to a sudden increase in intracranial pressure, rapid deformation of the brain and the formation of internal hernias. Masses of spilled blood can break into the ventricular system or the subarachnoid space.

*Clinically*they note a sudden onset, loss of consciousness, not uncommon development of a comatose state. A little later, meningeal symptoms may be detected: stiffness (increased tone, tension) of the muscles of the back of the head, Kernig's symptom (impossibility of passive extension of the leg bent at the hip and knee joint). When blood breaks into the ventricles of the brain, the patient's condition worsens. Narrowing of the pupils (miosis) is also a characteristic feature of a hemorrhagic stroke. Patients with a large brain hemorrhage rarely survive 1-2 days.

*In pathological examination*external appearanceCerebral hematoma varies and depends on the age of the process. A recent hemorrhage looks like a cluster of dark red blood clots. If its volume is not so large as to lead to quick death, then after about 1 week the peripheral zone of the hematoma acquires a brownish color. Under the microscope,

hemorrhage is represented by masses of spilled blood, which can hardly cause (or rather, not have time to cause) a reactive response of glia. Over time, proliferative changes in capillaries and astrocytes appear around the hemorrhage zone, in addition to gliosis and hemosiderin, the masses of which are determined outside cells and inside macrophages. If the patient continues to live, gliosis turns into a tender capsule. Eventually, hemolyzed blood products are broken down and completely removed by macrophages,

Another quite frequent cause of spontaneous intracerebral hemorrhage is the rupture of a vascular malformation. We are talking about varicose veins, arteriovenous aneurysms, etc., the sizes of which can vary from small capillary angiomas to massive formations built from large and thick-walled vascular channels. Many malformations do not prevent the long life of the patient, but some of them end with subarachnoid hemorrhage.

S u b a r a c h n o i d h e m o r r h a g e . About 65% of patients with spontaneous non-traumatic subarachnoid hemorrhages have at the basis of these intracranial catastrophes the rupture of a saccular aneurysm of one of the main cerebral arteries. About 5% of observations of subarachnoid hemorrhages are due to cases of rupture of vascular malformations, another 5% - to blood diseases, as well as the spread of intracranial or intraventricular hematoma into the subarachnoid space. In approximately 25% of cases, the cause cannot be identified, despite full cerebral angiography and thorough postmortem examinations.

Subarachnoid hemorrhage develops acutely. Disturbances and a twilight state are noted, there is a short-term loss of consciousness. 50% of patients develop vomiting, bradycardia, stiffness of the neck muscles, bilateral Kernig symptom. In the future, a rise in body temperature, moderate leukocytosis, and blood in the cerebrospinal fluid are observed. If the patient continues to live, then after a few days xanthomatosis (yellow colors) of the cerebrospinal fluid is determined, and after about 3 weeks the cerebrospinal fluid becomes colorless. Approximately 40% of patients with subarachnoid hemorrhages die, especially when blood breaks from the substance of the brain into the lateral or IV ventricles.

S e c o n d a r y brain damage. It will be about intracranial hemorrhages. These are frequent complications of head injuries, widespread causes of sudden deterioration of the condition and death of patients who were conscious immediately after the injury. Intracranial hematomas are especially common in people with skull fractures. They can be extradural, subdural and intracerebral. Let's dwell on each of these options.

Extradural (epidural) hematoma. Such a hematoma is formed as a result of hemorrhage from meningeal blood vessels, as a rule, from the middle meningeal artery. As the hematoma develops, the dura mater peels off with masses of blood from the adjacent bones of the skull. At the same time, there is a progressive compression of the brain tissue by the spilled blood. In young children, extradural hematoma can occur even without skull fractures. In the initial stages, the disease can proceed relatively easily. For several hours, the patient has a period of clear consciousness. Then headache and drowsiness develop. As the volume of the hematoma increases, the intracranial

pressure increases, the patient falls into a comatose state and may die if the mass of blood from the hematoma is not evacuated. Extradural hematomas are sometimes found in the frontal, parietal areas or the posterior cranial fossa.

Subdural hematoma.This hematoma is formed, as a rule, as a result of the rupture of venous bridges flowing into the upper sagittal sinus, or with severe superficial contusions. Diffuse distribution of blood masses in the subdural space is noted. In the case of rapid onset of death after an injury, an acute subdural hematoma is often found at the post-mortem examination. It can be large and be a voluminous intracranial lesion. There are also cells in the form of a thin strip of blood. But even in the latter case, intracranial pressure often increases, which is due to swelling of the underlying brain tissue. In some patients with an acute subdural hematoma, a period of clear consciousness is noted, similar to what happens with extradural hemorrhage. Chronic subdural hematoma is fixed at the stage when when it exists for several weeks or months after a normal brain injury. The hematoma gradually undergoes organization and is surrounded by a fibrous capsule. Because chronic subdural hematoma is quite common in old people who already have some brain atrophy, and because this hematoma spreads slowly, it can recur with small hemorrhages, it can reach large volumes while remaining asymptomatic for some time. In the absence of treatment, death is associated with secondary brain tissue damage due to high intracranial pressure. Chronic subdural hematoma is often a bilateral lesion. Because chronic subdural hematoma is quite common in old people who already have some brain atrophy, and because this hematoma spreads slowly, it can recur with small hemorrhages, it can reach large volumes while remaining asymptomatic for some time. In the absence of treatment, death is associated with secondary brain tissue damage due to high intracranial pressure. Chronic subdural hematoma is often a bilateral lesion. Because chronic subdural hematoma is quite common in old people who already have some brain atrophy, and because this hematoma spreads slowly, it can recur with small hemorrhages, it can reach large volumes while remaining asymptomatic for some time. In the absence of treatment, death is associated with secondary brain tissue damage due to high intracranial pressure. Chronic subdural hematoma is often a bilateral lesion. associated with secondary brain tissue damage due to high intracranial pressure. Chronic subdural hematoma is often a bilateral lesion. associated with secondary brain tissue damage due to high intracranial pressure. Chronic subdural hematoma is often a bilateral lesion.

Intracerebral (parenchymal) hematoma.It is conditionedcontusions and occurs mainly in the frontal and temporal lobes. The name "open lobe" is used for superficial contusions to denote an intracerebral hematoma that continues into a subdural hematoma. Small and deeply located intracerebral hematomas, which often appear in the form of hematomas of the basal nuclei (caudate and lenticular subcortical nuclei of the base of the cerebral hemispheres), are more common in people with diffuse damage to axons.

O t h e r s brain damage Secondary injuries that develop during a craniocerebral injury are often accompanied by an increase in intracranial pressure, deformation and

herniated protrusions of the brain tissue. Swelling of the brain often contributes to the increase in pressure. Some swelling is always noted in the areas of contusions, significant swelling of the brain tissue can occur in connection with a subdural hematoma. In addition, ischemic brain damage is found in 90% of people who die from craniocerebral injuries. Their pathogenesis is unclear; assume a connection with acute attack-like disorders of blood supply to the brain tissue, which may be a consequence of the injury itself, as well as shifts and deformations of the brain with increased intracranial pressure.

In some people, ischemic damage develops in connection with a delay in cardiac activity and breathing, as well as in epilepsy. Approximately 10% of people who have suffered a serious head injury are at risk of developing epilepsy (a chronic disease of a cerebral nature that manifests itself in repeated convulsive or other seizures and personality, accompanied by various changes). With open craniocerebral injuries, the incidence of epilepsy reaches 45%. As a rule, the disease develops within the 1st week after the injury (early epilepsy), less often it manifests itself after 2-3 months (late epilepsy). The presence of pressed fractures of the bones of the skull and intracranial hematomas are considered to be predisposing factors for the occurrence of late-onset epilepsy. In this variant, convulsive seizures are repeated more often than in early epilepsy.

Among other secondary lesions of the brain, it is worth mentioning post-traumatic amnesia or infectious complications, which are associated with an open trauma of the skull vault or a fracture of the base of the skull. These rather rare complications usually manifest as meningitis. The latter does not necessarily occur in the early post-traumatic period, since infectious agents can gradually penetrate through a small traumatic fistula that passes from the subarachnoid space into one of the main air cavities (sinuses) at the base of the skull. An even rarer infectious complication is intracranial abscess (traumatic brain abscess).

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Definition of cerebrovascular disease. Complications and causes of death.
2. Ischemic cerebral infarction, etiology, morphological characteristics, consequences.
3. Epidural hematoma, etiology, morphological characteristics, consequences.
4. Subdural hematoma, etiology, morphological characteristics, consequences.
5. Intracerebral hematoma, etiology, morphological characteristics, consequences.
6. Alzheimer's disease, etiology, morphological characteristics, consequences.
7. Multiple sclerosis, etiology, morphological characteristics, consequences.
8. Amyotrophic lateral sclerosis, etiology, morphological characteristics, consequences.
9. Postresuscitation encephalopathy, etiology, morphological characteristics, consequences.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues

and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

A 68-year-old woman who suffered from hypertension for a long time (blood pressure up to 220/110 mm Hg) was admitted to the clinic with complaints of a sharp headache and movement disorders in the left limbs. After 5 hours she died. At autopsy, in the right hemisphere of the brain, there is a cavity with uneven edges, filled with dark red, elastic blood clots. What circulatory disorder developed in the brain?
Correct answer Hematoma
B Hemorrhagic infiltration
CPetechiae
D Local venous congestion
Thrombosis
The patient suffered from hypertension for many years in the past. Death came from acute kidney failure. Name the characteristic changes in the kidneys found at autopsy?
Correct answer Arteriosclerotic nephrosclerosis
Arteriolonecrosis
C Renal infarction
D. Malignant hypertension
Athero-arteriosclerotic nephrosclerosis
Name the changes characteristic of the kidneys, found at the autopsy of a 45-year-old man who suffered from severe hypertension (BP 240/130 mm Hg) and died in the presence of chronic renal failure:
Correct answer The kidneys are small, dense, the surface is fine-grained
BLarge white kidneys
CLarge variegated kidney
DLarge sebaceous kidney
ECystic kidney
A 30-year-old man turned to a doctor with complaints of heart pain, palpitations, and shortness of breath upon slight physical exertion. During the last 5 years, a significant increase in blood pressure was noted. An adenoma of the adrenal cortex was revealed during the examination. Your diagnosis?
Correct answer Symptomatic hypertension
BHypertensive disease 1st stage
Hypertensive disease 2nd stage
Eccentric hypertrophy of the myocardium
Arteriosclerotic nephrosclerosis
During the autopsy of the corpse of a 72-year-old man who suffered from hypertensive disease and died due to increasing symptoms of autointoxication due to uremia, kidneys were found measuring 6.5x2.5x2 cm, weighing 70.0 g. They were dense, their surface was fine-grained, the parenchyma, especially the cortical substance , uniformly

thinned. Under microscopic examination, most nephrons are replaced by connective tissue. What is the name of the state of auto-intoxication in this case?
Correct answer Chronic renal failure (azotemic uremia)
BHypertensive crisis
Acute renal failure
Benign hypertension
Malignant hypertension
The 68-year-old patient suffered from hypertension for a long time and died during another sharp rise in blood pressure. At the autopsy, a massive hematoma in the subcortical nuclei on the right, numerous small hemorrhages and a "rusty" cyst in the occipital region were found in the brain. Your diagnosis?
Correct answer The 3rd stage of hypertensive disease during a crisis
BIschemic cerebral infarction
C1st stage of hypertensive disease, hypertensive crisis
Diapedic hemorrhages
EHemorrhagic cerebral infarction
At the autopsy, the deceased, who suffered from hypertension for a long time, had a transmural myocardial infarction in the past and died of chronic cardiovascular failure. It was established: the kidneys are small, dense, the heart is hypertrophied, the cavities are enlarged. Your diagnosis?
Correct answer Hypertensive disease of the 3rd stage
BAtherosclerosis, cardiac form
Hypertensive disease, pre-clinical stage
Hypertensive disease of the 2nd stage
ESymptomatic hypertension
The autopsy of the deceased from heart failure revealed an enlarged heart weighing 550 g, fibrinous pericarditis, as well as shrunken dense kidneys weighing 50 g with a fine-grained surface and pronounced hyalinosis of arterioles and glomeruli. Name the main disease:
Correct answer Hypertensive disease
Atherosclerosis
Rheumatism
Pericarditis
Cardiomyopathy
A microscopic examination of the tissues of the organs of the deceased, who suffered from hypertension, revealed widespread hyalinosis of the arterioles of the kidneys, brain, intestines, elastofibrosis and atherosclerosis in the elastic and muscular arteries. For which stage of hypertensive disease are these changes characteristic?
Correct answer Hypertensive disease of the 2nd century.
BHypertensive disease 1 st.
Preclinical
Hypertensive disease of the 3rd century.
Hypertensive disease with secondary changes

A 55-year-old man died of a massive brain hemorrhage that occurred as a result of hypertension. The autopsy revealed typical pathomorphological signs of arterial hypertension. Damage to which vessels is a typical sign of hypertension?
Correct answer Arteriole
Artery of muscular type
Artery of elastic type
Artery of the elastic-muscular type
Large veins
An autopsy of a man who died of cardiac decompensation revealed a sharply enlarged heart weighing 960 g ("bull's heart"), with 90 ml of straw-colored liquid in the pericardial cavity. The cavities of the heart are sharply enlarged, the myocardium is flaccid, the thickness of the wall of the left ventricle is 2.3 cm. The kidneys are reduced in size, weighing 70 g each, and their surface is fine-grained. During the microscopic examination of internal organs, hyalinosis of arterioles and hypertrophy of the muscular layer of larger arteries were revealed. Name the main disease:
Correct answer Hypertensive disease
Ischemic heart disease
Cardiomyopathy
Atherosclerosis
Rheumatism
A 52-year-old patient with a persistent increase in blood pressure up to 200/110 mm Hg. died of chronic cardiovascular insufficiency. At autopsy: the weight of the heart was 600 g, the thickness of the wall of the left ventricle was 2.2 cm, the heart cavity was enlarged. Histologically pronounced hyalinosis and sclerosis of myocardial arterioles. For which disease are the described changes characteristic?
Correct answer Hypertensive disease
Atherosclerosis of heart arteries
CDilated cardiomyopathy
Hypertrophic cardiomyopathy
Endomyocardial fibrosis
.
A 67-year-old patient suffered from hypertension for 20 years. He died of chronic kidney failure. What did the kidneys look like when the body was dissected?
Correct answer Small, dense, fine-grained surface
BLarge variegated
Big red ones
DBig whites
Large with multiple thin-walled cysts

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;

2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

8. methods: assessment of the correctness of the performance of practical skills
9. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem,

	tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Systemic connective tissue diseases with autoimmunization: rheumatism, systemic lupus erythematosus, rheumatoid arthritis, systemic scleroderma, dermatomyositis, Bekhterev's disease.".

Suggested topics for essays:

1. Alzheimer's disease. Morphological characteristics.
2. Cerebrovascular disease. Morphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association

6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 23

Topic: Systemic connective tissue diseases with autoimmunization: rheumatism, systemic lupus erythematosus, rheumatoid arthritis, systemic scleroderma, dermatomyositis, Bekhterev's disease.

Goal: learn to determine etiology, pathogenesis, morphologysystemic diseases of connective tissue with autoimmunization.

Basic concepts:general characteristics of rheumatic (collagenous) diseases, diseases included in the rheumatic group, etiology, pathogenesis, pathological anatomy, clinical and anatomical forms and complications of rheumatism, the main changes in systemic lupus erythematosus, rheumatoid arthritis, systemic scleroderma, dermatomyositis, Bekhterev's disease.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Rheumatic diseases (systemic diseases of connective tissue, mesenchyme) - a group of diseases characterized by progressive systemic disorganization of connective tissue, which occurs in 4 phases:
1) mucoid swelling;
2) fibrinoid swelling;
3) productive inflammation (granulomatosis);
4) sclerosis of connective tissue;
as well as hypergammaglobulinemia, plasmatization of lymphoid tissue and bone marrow. Rheumatic diseases include 14 nosological units.

rheumatism - against the background of systemic connective tissue pathology, the heart, musculoskeletal system, and central nervous system suffer the most.
dermatomyositis - skin (dermis) and muscles (striated and smooth).
Nodular periarteritis is the adventitial sheath of arterioles and veins, manifested by dystrophy and sclerosis in internal organs, up to heart attacks, and hemorrhages.
Systemic scleroderma - damage to blood vessels (vasculitis) of the skin, muscles, joints, kidneys, heart ("scleroderma kidney" and heart).
Systemic lupus erythematosus (SLE) affects blood vessels, skin, joints, as well as visceral organs (kidneys, liver, myocardium).
Rheumatoid arthritis - joints (joint capsule, synovial membrane, articular cartilage), stroma of visceral organs.
Sjogren's syndrome - skin lesions, xerophthalmia, xerostomia.
Bekhterev-Marie-Strümpel's disease (Ankylosing spondylitis) is a predominant lesion of the articular-ligamentous apparatus of the spine, as well as the heart and lungs.
Wegener's granulomatosis - angiitis of the kidneys, lungs and upper respiratory tract.
Scheme of the typical development of rheumatism (with the selection of three periods according to Nesterov):
streptococcal infection (more often angina) and the I period of sensitization of the body to streptococcal infection, i.e. the latent period, which is 2-4 weeks, when the production of anti-streptococcal antibodies and the formation of the immune complex AG-streptococcus + AT + C is carried out.
// period - hyperergic reaction: due to damage by immune complexes, as well as the reaction of cross-reactivity of antibodies from AG-connective tissue, prostheses break down glycoprotein complexes of connective tissue, supporting long-term autoimmune inflammation with granulomatosis (like HRT. Clinically, the second period is manifested by a primary rheumatic attack.
/// period - the period of recurrence of the process. The morphologic substrate of rheumatism is systemic progressive disorganization of connective tissue (ST) and a specific proliferative cellular reaction, especially in endocarditis and microcirculatory vessels.
A) Specific proliferative cellular reaction, ST disorganization.
1) Mucoid swelling - surface reversible disorganization of ST with the release of acidic glycosaminoglycans (k-hag), which cause metachromasia, and also increase vascular-tissue permeability, causing tissue infiltration by plasma P-proteins. The result is a transition to fibrinoid swelling and necrosis.
2) Fibrinoid swelling and necrosis - irreversible disorganization of the ST with the formation of a complex fibrinoid complex, resulting in fibrinoid necrosis.
3) Cellular reactions (granulomatosis). The cycle of granuloma development lasts up to 6 months.
Phase I - accumulation of macrophages in the focus of damage;
Phase II - "Flowering" ("mature") granuloma - macrophages are fan-shaped around fibrinoid masses;

III phase - "withered" granuloma - the focus decreases due to the lysis of fibrinoid masses by macrophages, and macrophages begin to form into fibroblasts;
IV phase - "scarring" granuloma - complete resorption of fibrinoid by macrophages, completion of transformation of macrophages into fibroblasts, scarring of the organ.
4) Sclerosis (secondary sclerosis) - the result of the development of a granuloma. At all stages of its development, Ashoff-Talalaiv granuloma is surrounded by lymphocytes and histiocytes that activate fibroblasts by releasing lymphokines.
B) There are also nonspecific cellular reactions in the form of lymphohistiocytic infiltration of the interstitial tissue of internal organs or vasculitis in MCR.
3. Clinical and anatomical forms of rheumatism.
• Cardiovascular (rheumatic endo-, myo- and pancarditis);
• Polyarthritic (rheumatic polyarthritis);
• Cerebral (small chorea) - movement disorders due to damage to brain vessels;
• Nodous (nodular rheumatism) - granulomas in the subcutaneous tissue, aponeuroses, tendons, fascia and muscles.
Complications and consequences of rheumatic diseases.
The results of rheumatic endocarditis - thickening and deformation of valve leaflets and chordae leads to insufficiency of the atrioventricular valve; stenosis of the fibrous ring and fusion of the leaflets - to stenosis of the atrioventricular opening, i.e., heart defects are formed. Rheumatic myocarditis ends with cardiosclerosis and a decrease in the contractile function of the myocardium. Warty endocarditis can be complicated by thromboembolism with heart attacks of the kidneys, spleen, brain, retina, and gangrene of the limbs.
Rheumatic polyserositis (including pericarditis) can lead to obliteration of the pleura and pericardium.
Thanatogenesis: death occurs from thromboembolism, or more often from decompensation of a heart defect.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Give a general description of rheumatic diseases.
2. Definition of rheumatism, etiology.
3. Pathogenesis of rheumatic disease.
4. Microscopic characteristics of rheumatic granuloma.
5. Clinical and morphological forms of rheumatic disease.
6. Types of endocarditis. Morphological characteristics.
7. Types of myocarditis. Morphological characteristics.
8. Complications and outcomes of rheumatic disease.
9. Definition of SLE (systemic lupus erythematosus).
10. Morphological characteristics of changes in organs and tissues in SLE.
11. Complications and causes of death of microwave oven.
12. Definition of rheumatoid arthritis.
13. Morphological characteristics. Complications and causes of death.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

In a 68-year-old man who died of chronic heart failure, the autopsy revealed deformed, thickened leaflets of the mitral valve, which had fused together, and small (1-2 mm) blood clots were detected along the edge of the valve closure. Which endocarditis caused the development of chronic heart failure?
The correct answer is Reverse-warty
B Diffuse
CSharp warty
DFibroplastic
EPolypous-ulcerative
The 50-year-old patient had rheumatic heart disease for many years. During the exacerbation of the disease, hemiplegia developed and death occurred. Histologically, severe sclerosis, focal cellular infiltrates, and small thrombotic stratifications were found in the mitral valve. For which form of endocarditis are the detected changes characteristic?
Correct answer Turn-warty
BSharp warty
Diffuse
DFibroplastic
E Ulcerative-polypous
A young woman has dermatitis in the form of a "red butterfly" on her face. Microscopic examination of the skin biopsy revealed changes represented by proliferative-destructive vasculitis, edema of the papillary layer of the dermis, focal, mainly perivascular, lymphohistiocytic infiltration. Make a diagnosis:
Correct answer Systemic lupus erythematosus
Rheumatism
Scleroderma
Rheumatoid arthritis
Dermatomyositis
At the autopsy of the deceased, morphological manifestations were revealed: stenosis of the left atrioventricular opening, insufficiency of the mitral valve. Histologically, in the myocardium there is focal cardiosclerosis, the presence of flowering Ashof-Tallalaev granulomas. Which of the following diagnoses is most likely?
Correct answer Rheumatism
B Dermatomyositis

CScleroderma
DNodular periarteritis
Systemic lupus erythematosus.
At the autopsy of a 25-year-old patient who died of uremia: the kidneys are enlarged, variegated, with foci of hemorrhages. Pathohistologically, hematoxylin bodies, capillary membranes of glomeruli in the form of wire loops, hyaline thrombi and foci of fibrinoid necrosis are detected, "onion" sclerosis in the vessels of the spleen. What is the most likely diagnosis?
Correct answer Systemic lupus erythematosus
BSystemic scleroderma
Rheumatic arthritis
DNodular periarteritis.
Rheumatoid arthritis
A 50-year-old patient had rheumatic heart disease for many years. As the disease worsened, hemiplegia developed and death ensued. Histologically, severe sclerosis, focal cellular infiltrates, and fibrinoid deposits were found in the mitral valve. What form of endocarditis are characteristic of the detected changes?
Correct answer Reverse-Warty
B Diffuse
CSharp warty
DFibroplastic
E Ulcerative polyposis
The autopsy of the dead man revealed a widespread thromboembolic infarction of the left hemisphere of the brain, as well as a large septic spleen, immune complex glomerulonephritis, ulcers in the leaflets of the aortic valve with polyp-like thrombi and colonies of staphylococci. What disease led to cerebral thromboembolism?
Correct answer Septic bacterial endocarditis
BRheumatic thromboendocarditis
Acute rheumatic valvulitis
Septicemia
ESeptikopyemia
An enlarged spleen was found at the autopsy of a 40-year-old woman suffering from rheumatoid arthritis. In section, its tissue is brownish-red with enlarged follicles that look like translucent grayish-white grains. Indicate which of the listed pathological processes is the most likely?
Correct answer Sago spleen
BHyalinosis of the spleen
Porphyry spleen
DGlazed spleen
ESebaceous spleen
A 54-year-old woman had significant deformation of the joints of her fingers and feet. Histologically: peri-articular connective tissue – mucous swelling, areas of fibrinous necrosis, accumulation of macrophages, areas of sclerosis; in the synovial membrane -

swollen villi with signs of mucous and fibrinous swelling, in the synovial cavity there are single "rice bodies". Diagnose the disease:
Correct answer Rheumatoid arthritis
Rheumatism
Hematogenous tuberculosis
Gout
E Bechterew's disease
At the autopsy of the deceased, morphological manifestations of stenosis of the left atrioventricular opening, insufficiency of the mitral valve were found. Histologically, in the myocardium there is focal cardiosclerosis, the presence of flowering Ashof-Tallalaev granulomas. Which of the following diagnoses is most likely?
Correct answer Rheumatism
B Dermatomyositis
Systemic lupus erythematosus.
DNodular periarteritis
Scleroderma
At the autopsy of the body of a 40-year-old woman who died of uremia, the following were found: enlarged variegated kidneys, in the kidneys - thickening of the capillary membranes of the glomeruli in the form of "wire loops", foci of fibrinous necrosis of their walls and "hyaline" thrombi in the lumens, in the nuclei - "hematoxylin body"; Libman-Sachs endocarditis in the heart. What kidney damage is most likely?
Correct answer Lupus nephritis
BSclerosed kidney
CTerminal glomerulonephritis
Rheumatic glomerulonephritis
Cholera glomerulonephritis
In a 7-year-old child, dense, painless nodules 1-2 mm in size appeared on the skin of the extensor surfaces of the elbow and knee joints. In the nodule biopsy, there is a large focus of fibrinous necrosis of connective tissue with lymphocytes and macrophages on the periphery. In what disease are such nodules observed?
Correct answer Rheumatism
BSystemic lupus erythematosus.
CScleroderma
Rheumatoid arthritis
ENodular periarteritis
Microscopic examination of the myocardium of a girl who died of diphtheria due to heart failure revealed: fatty dystrophy and multiple foci of cardiomyocyte necrosis, minor focal cellular infiltrates in the interstitial tissue. What kind of myocarditis are we talking about?
Correct answer Interstitial myocarditis
B Granulomatous myocarditis
C Diffuse exudative myocarditis
Focal exudative myocarditis

E Alterative myocarditis

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

10. methods: assessment of the correctness of the performance of practical skills
11. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the

	discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Final lesson. Practical skills".

Suggested topics for essays:

1. Sisthmic scleroderma. Morphological characteristics.
2.Dermatomyositis.Morphological characteristics.
3. Bekhterev's disease.Morphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine

2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 24

Topic: Final lesson.(section Diseases of the blood system and cardiovascular system. Nervous diseases). Practical experience.

Goal:to test the ability to describe micro and macro drugs with a grant of the conclusion of the pathological process according to the topics of the final control.

Basic concepts:see above according to the topics of the final control.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

see above according to the topics of the final control.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Describe the micropreparation, draw a conclusion.
2. Describe the macropreparation, draw a conclusion.
3. Definition of this pathological process.

4. Complications and causes of death of this pathological process.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

see above according to the topics of the final control.

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

12. methods: assessment of the correctness of the performance of practical skills
13. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Respiratory diseases.".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 25

Topic: Respiratory diseases.

Goal: learn to determine etiology, pathogenesis, morphologyrespiratory diseases.

Basic concepts: Acute bronchitis: pathological anatomy. Acute bronchiolitis (primary, follicular, obliterating): pathological anatomy. Complication. Acute inflammatory lung diseases. General characteristics, modern classification of pneumonia. Clinical and morphological features, stages of development, complications, consequences of acute inflammatory lung diseases. Chronic non-specific lung diseases. Definition, classification, morphogenesis. Chronic obstructive bronchitis, chronic obstructive emphysema, bronchiectasis and bronchiectatic disease, morphological characteristics, complications, consequences. Bronchial asthma, diffuse chronic lesions, morphological characteristics, complications, consequences. Tumors of bronchi and lungs.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present,

announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Acute bronchitis - acute inflammation of the bronchi - can be an independent disease or a manifestation of a number of diseases, in particular pneumonia, chronic glomerulonephritis with renal failure (uraemic bronchitis).
We talk about chronic bronchitis if clinical symptoms of the disease (cough and sputum production) are observed for at least three months within two years.
Acute bronchitis, as a rule, is more severe in children. Clinically, it is manifested by cough, dyspnea and tachypnea.
Pathological anatomy. With acute bronchitis, the mucous membrane of the bronchi becomes full of blood and swollen, small hemorrhages and ulceration are possible. In most cases, there is a lot of mucus in the lumen of the bronchi. Various forms of catarrhal inflammation develop in the bronchial mucosa with the accumulation of serous, mucous, purulent, mixed exudate. Fibrinous or fibrinous-hemorrhagic inflammation often occurs in the bronchi; destruction of the bronchus wall is possible, sometimes covered with ulcers on the mucous membrane of the latter (in this case, they speak of destructive ulcerative bronchitis).
Acute bronchitis can be productive, which leads to thickening of the wall due to its infiltration by lymphocytes, macrophages, plasma cells, proliferation of the epithelium. In the proximal parts of the bronchi, only the mucous membrane (endobronchitis) or the mucous membrane and the muscle layer (endomesobronchitis) is mainly damaged. In the distal parts of the bronchi, all layers of the bronchial wall are involved in the process (panbronchitis and panbronchiolitis), while the transition of inflammation to the peribronchial tissue (peribronchitis) is possible.
complications of acute bronchitis are often associated with a violation of the drainage function of the bronchi, which contributes to the aspiration of infected mucus into the distal parts of the bronchial tree and the development of lung tissue inflammation (bronchopneumonia). With panbronchitis and panbronchiolitis, the transition of inflammation is possible not only to the peribronchial tissue, but also to the interstitial tissue of the lungs (peribronchial interstitial pneumonia).
Acute pneumonia can be classified according to several features. Acute pneumonia is divided into:
primary;
secondary
Primary acute pneumonia includes pneumonia as an independent disease and as a manifestation of another disease that has nosological specificity (for example,

influenza, plague pneumonia). Secondary acute pneumonia is often a complication of many diseases.
Three main types of pneumonia are distinguished according to the topographic-anatomical feature (localization):
parenchymatous pneumonia;
interstitial pneumonia;
bronchopneumonia
According to the prevalence of inflammation:
miliary pneumonia, or alveolitis;
acinous;
partial, draining partial;
segmental polysegmental;
partial pneumonia.
According to the nature of the inflammatory process, pneumonia is:
serous (serous-leukocyte, serous-desquamative, serous-hemorrhagic);
purulent;
fibrinous;
hemorrhagic
Acute pneumonia is classified into pneumonia that develops in a normal (non-immunosuppressive) organism and pneumonia that develops in an immunosuppressive organism.
Croupous pneumonia is an acute infectious-allergic disease in which one or more parts of the lungs are affected (partial, Lobar pneumonia), fibrinous exudate appears in the alveoli (fibrinous, or croupous, pneumonia), and fibrinous overlays (pleuropneumonia) appear on the pleura. croup pneumonia is an acute infectious-allergic disease in which one or more parts of the lungs are affected (partial, lobar pneumonia), fibrinous exudate appears in the alveoli (fibrinous, or croupous, pneumonia), and fibrinous overlays (pleuropneumonia) appear on the pleura.
Morphogenesis, pathological anatomy. Partial pneumonia is a classic example of acute inflammation and consists of four stages:
Tide stage. The first stage lasts 24 hours and is characterized by inflammation of alveoli with protein-rich exudate and venous congestion in the lungs. The lungs become dense, heavy, swollen and red.
The stage of red burning. In the second stage, which lasts several days, there is a massive accumulation of polymorphonuclear leukocytes with a small number of lymphocytes and macrophages in the lumen of the alveoli, fibrin threads fall out between the cells. Also, the exudate contains a large number of erythrocytes. Often, the pleura above the lesion is covered with fibrinous exudate. The lungs become red, dense and airless, resembling the liver in consistency.
The stage of gray burning. This stage can also last several days and is characterized by the accumulation of fibrin and the destruction of white and red blood cells in the exudate. The lungs become gray-brown and dense on the section.

The decision stage. The fourth stage begins at the 8-10th week of the disease and is characterized by resorption of exudate, enzymatic breakdown of inflammatory detritus and restoration of the integrity of the alveolar walls. Fibrinous exudate under the influence of proteolytic enzymes of neutrophils and macrophages undergoes dilution and resorption. The lungs are cleaned of fibrin and microorganisms: exudate along the lymphatic drainage of the lungs with sputum. Fibrinous overlays on the pleura dissolve. The stage of "resolution" sometimes stretches for several days after the clinically fever-free course of the disease.
Complication. Pulmonary and extrapulmonary complications of croup pneumonia are distinguished. Pulmonary complications develop in connection with a violation of the fibrinolytic function of neutrophils. When this function is insufficient, the masses of fibrin in the alveoli undergo organization, that is, they grow into granulation tissue, which, maturing, turns into a mature fibrous connective tissue. This process of organization is called carnification (from the Latin Sarpeo - meat). The lung turns into airless dense fleshy tissue. With excessive activity of neutrophils, the development of lung abscess and gangrene is possible. Attachment of pus to fibrinous pleurisy leads to pleural empyema.
Extra-pulmonary complications: observed in the generalization of the infection. With lymphogenic generalization, purulent mediastinitis and pericarditis occur, with hematogenous - peritonitis, metastatic abscesses in the brain, purulent meningitis, acute ulcerative or polyposis-ulcerative endocarditis, more often of the right heart, purulent arthritis, etc. Modern means of treating croupous pneumonia have dramatically changed its clinical and morphological picture, which allows us to talk about the induced pathomorphosis of this disease. Under the influence of antibiotics and chemopreparations, croup pneumonia takes an abortive course, the number of cases of both pulmonary and extrapulmonary complications decreases.
Death with croupous pneumonia occurs from heart failure (especially often in old age, as well as chronic alcoholism) or from complications (brain abscess, meningitis, etc.).
Chronic non-specific lung diseases (CKD) include:
chronic bronchitis;
emphysema of the lungs;
bronchial asthma;
bronchiectasis;
chronic pneumonia;
interstitial lung diseases; - pneumofibrosis (pneumocirrhosis).
Bronchitogenic, pneumonogenic and pneumonitogenic are distinguished among the mechanisms of development of these diseases.
At the heart of the bronchitogenic mechanism of chronic obstructive pulmonary disease is a violation of the drainage function of the bronchi and bronchial conduction. Diseases that are united by this mechanism, or chronic obstructive lung diseases, are represented by chronic bronchitis, bronchiectasis (bronchiectatic disease), bronchial asthma and emphysema of the lungs (especially chronic diffuse obstructive). Chronic (diffuse) obstructive diseases are characterized by reversible or irreversible violations

of the structure of the bronchi or bronchioles, which lead to impaired lung ventilation. With a significant lesion of the bronchi, there is a decrease in the functional indicators of the lungs, namely:
decrease in the vital capacity of the lungs (LV);
decrease in the ratio of the maximum volume of inhalation and exhalation to the LV;
- reduction of the maximum speed of forced exhalation.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Acute bronchitis: pathological anatomy. Acute bronchiolitis (primary, follicular, obliterating): pathological anatomy. Complication.
2. Acute inflammatory lung diseases. General characteristics, modern classification of pneumonia.
3. Clinical and morphological features, stages of development, complications, consequences of acute inflammatory lung diseases.
4. Chronic non-specific lung diseases. Definition, classification, morphogenesis.
5. Chronic obstructive bronchitis, chronic obstructive emphysema, bronchiectasis and bronchiectasis, morphological characteristics, complications, consequences.
6. Bronchial asthma, diffuse chronic lesions, morphological characteristics, complications, consequences.
7. Tumors of bronchi and lungs.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

During the macroscopic examination of the lung tissue, areas of increased airiness with the presence of small bubbles were found, and histologically - thinning and rupture of alveolar membranes with the formation of large cavities of various shapes. What disease was detected in the lungs?
Correct answerEmf pulmonary edema
B Bronchiectatic disease
Cavernous tuberculosis
Chronic bronchitis
EFibrosing alveolitis

At autopsy, the upper part of the right lung is enlarged, gray in color, airless, cloudy liquid flows from the cut surface, there are many fibrinous films on the pleura. Microscopically, exudate with the presence of neutrophils, desquamative alveolocytes and fibrin threads is detected in the alveoli. The bronchus wall is intact. What is the most likely diagnosis?

Correct answer Croupous pneumonia
Interstitial pneumonia
Lung abscess
Focal pneumonia
Influenza pneumonia
Examination of the bronchobiopsy showed atrophy of the mucous membrane, cystic transformation of the glands, focal metaplasia of the covering prismatic epithelium into a multi-layered flat one, an increase in the number of goblet cells, in places in the wall of the bronchus and especially in the mucous membrane, there was a pronounced cellular inflammatory infiltration and proliferation of bronchial tissue, which erupted into the in the form of a polyp. What is the most likely diagnosis?
Correct answer Chronic bronchitis
BAcute pneumonia
Acute bronchitis
Bronchopneumonia
Interstitial pneumonia
The patient has a high temperature, shortness of breath, pain in the right side of the chest. Pleural puncture yielded 700 ml of thick yellow-green fluid. What pathological process developed in the pleural cavity?
Correct answer Pleural empyema
Bronchopneumonia
Serous pleurisy
D Hemorrhagic pleurisy
E Carcinomatosis of the pleura
As a result of a histological examination of a biopsy from the wall of a bronchus of a patient with chronic bronchitis, the growth of granulation tissue protruding above the surface of the mucous membrane and a diffuse inflammatory infiltrate were found in the mucous layer. Diagnose a type of bronchitis?
Correct answer Chronic polyposis bronchitis
B Chronic serous-purulent bronchitis
C Chronic serous bronchitis
D Chronic purulent bronchitis
E Chronic deforming bronchitis
A 67-year-old patient had a severe form of influenza with a fatal outcome. At section: changes in the lungs were similar to changes in "large variegated lungs". Microscopic examination revealed: acute fullness of blood vessels, hemorrhages, swelling of lung tissue, in the lumen of the bronchi and alveoli exudate, which contains mainly erythrocytes. What character of lung inflammation do these morphological signs indicate?
Correct answer Hemorrhagic bronchopneumonia
Purulent bronchopneumonia
Fibrinous pneumonia.
Desquamative bronchopneumonia

E Catarrhal bronchopneumonia
At the autopsy of a 47-year-old man who died from pulmonary heart failure, a 4x4 cm cavity was found in the left lung, filled with pus, the walls were uneven, represented by lung tissue. Most likely it is:
Correct answer Acute abscess
Bechinococcosis
CFibrosing alveolitis
D Chronic abscess
E Cavernous tuberculosis
Microscopic examination of a bronchobiopsy revealed a tumor that is made up of clustered atypical cells of a multi-layered flat epithelium, with characteristic "pearls" in places. Your diagnosis?
Correct answer Squamous cell carcinoma with keratinization
BSquamous cell carcinoma without keratinization
C Solid cancer
D Skirr
E Mucosal cancer
In a bronchus biopsy of a patient who abuses smoking, chronic inflammation and transformation of a single-layered ciliated epithelium into a multi-layered flat epithelium was found in the thickened mucous membrane. Which of the processes is most likely?
Correct answer Metaplasia
BHypertrophy of the epithelium
Squamous cell cancer
Epithelial hyperplasia
ELeukoplakia
At the autopsy, it was found that the entire right lung was enlarged, dense, fibrin was layered on the pleura, and the tissue was gray in cross section, from which a cloudy liquid was flowing. For which lung disease is this picture characteristic?
Correct answer Croupous pneumonia
BGangrene of the lungs
CFibrosing alveolitis
Focal pneumonia
Interstitial pneumonia
During the autopsy of the corpse of a man with a malignant tumor of the stomach, who died of cancer intoxication, in the back-lower parts of the lungs, dense gray-red, irregularly shaped foci were found, which protrude above the cut surface. Microscopically: in the lumen of the walls of small bronchi and alveoli, an exudate with many neutrophils was found. What disease do the changes in the lungs of the deceased indicate?
Correct answer Acute purulent bronchopneumonia
BIntermediate pneumonia
Ccropulous pneumonia

DAcute serous bronchopneumonia
Acute bronchitis
Macroscopic examination of the lung tissue revealed areas with increased air content and the presence of small bubbles, and histologically - thinning and rupture of alveolar septa with the formation of large cavities of various shapes. What disease was detected in the lungs?
Correct answer Emphysema of the lungs
BFibrosing alveolitis
Cavernous tuberculosis
Bronchoectatic disease
Chronic bronchitis
The patient died of pulmonary and heart failure. Histological examination revealed: diffuse lung damage with interstitial edema, tissue infiltration by lymphocytes, macrophages, plasma cells; pneumofibrosis, panacinar emphysema. What is the most likely diagnosis?
Correct answer Fibrosing alveolitis
B Bronchial asthma
Chronic bronchitis
D Bronchopneumonia
Atelectasis of the lungs
When examining the bronchobioptate, the following were found: atrophy of the mucous membrane, cystic transformation of the glands, focal metaplasia of the covering prismatic epithelium into a multilayered flat one, an increase in the number of goblet cells; in places - in the wall of the bronchus and especially in the mucous membrane, cellular inflammatory infiltration and growth of granulation tissue, which explodes into the lumen of the bronchus in the form of a polyp, is pronounced. What is the most likely diagnosis?
Correct answer Chronic bronchitis
BPartial pneumonia
Acute bronchitis
Bronchopneumonia
Interstitial pneumonia
On cross-section, the lung tissue has a large-celled appearance in connection with bag-like and cylindrical expansions of the bronchi; microscopically, leukocyte infiltration with a predominance of neutrophils is noted in the wall of such bronchi; elastic, muscle fibers, as well as cartilaginous plates are partially destroyed and replaced by connective tissue. Adjacent lung tissue with foci of inflammation, fields of fibrosis, vascular sclerosis, signs of emphysema. Hypertrophied right ventricle of the heart. What is the most likely diagnosis?
Correct answer Bronchiectatic disease
BEmphysema of the lungs
Interstitial pneumonia
Pneumofibrosis

Chronic bronchitis

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

14. methods: assessment of the correctness of the performance of practical skills
15. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates

	practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Diseases of esophagus, stomach and intestines.".

Suggested topics for essays:

1. Bronchial asthma. Morphological characteristics.
2.Emphysema of the lungs.Morphological characteristics.
3. Bronchiectatic disease. Morphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine

2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 26

Topic: Diseases of esophagus, stomach and intestines.

Goal: To study pathomorphological signs, morphological classification, morphological characteristics of the most important diseases of the digestive organs.

Basic concepts: Diseases of the esophagus: morphological characteristics. Chronic gastritis: clinical and morphological characteristics, complications, outcome. Ulcer disease: clinical and morphological characteristics, complications, outcome. Tumors of the esophagus and stomach. Enterocolitis: clinical and morphological characteristics, complications. Tumors of the small and large intestine, morphological characteristics. Appendicitis. Classification. Morphological characteristics of manifestations of acute and chronic appendicitis. Complication.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

DISEASES OF THE ESOPHAGUS. Among diseases of the esophagus, diverticula, inflammation (esophagitis) and tumors (cancer) are more common.
Esophageal diverticulum is a limited blind protrusion of its wall, which consists of all the membranes of the esophagus wall (true diverticulum) or only the mucosa and submucosa, which protrude through the slits of the muscular membrane (muscular diverticulum). Depending on the localization and topography, pharyngoesophageal, bifurcation, epinephric and multiple diverticula are distinguished, and depending on the features of origin - adhesion diverticula, which arise as a result of inflammatory processes in the mediastinum, and relaxation diverticula, which are based on local relaxation of the esophageal wall. Esophageal diverticulum can be complicated by an inflammatory process (diverticulitis).
The causes of diverticulum formation can be congenital (inferiority of the connective and muscular tissues of the wall of the esophagus, pharynx) and acquired (inflammation, sclerosis, narrowing scars, increased pressure in the esophagus).
Inflammatory processes in the stomach, which manifest as gastritis (from the Greek Gaster - stomach), as well as in other organs, can be acute or chronic. The pathogenesis of chronic gastritis is complex. Until recently, it was believed that in one form of chronic gastritis - type A, autoimmune reactions are observed, and in another form (non-immune gastritis type B), inflammation occurs as a result of long-term exposure to various non-specific irritants, such as exogenous (for example, hot drinks or spicy spices) or endogenous (for example, bile reflux). It has now been proven that type B chronic gastritis is a response to a bacterial infection. Gastritis, which develops as a result of bile reflux, is excluded from this group.
CHRONIC GASTRITIS
Today, the following forms of chronic gastritis are distinguished:
autoimmune chronic gastritis;
Helicobacter - associated chronic gastritis;
chemical (reflux) gastritis;
other forms of gastritis.
Types of chronic gastritis

Etiology	pathogenetic	histological changes	Accompanying clinical
autoimmune	Antibodies against parietal cells and receptors for external factor Castle. Sensitized T-	Atrophy of the glands in the body of the stomach. intestinal metaplasia	Pernicious anemia
bacterial infection (H. rori)	cytotoxins Mucolytic enzymes Synthesis of ammonium ions by	Active chronic inflammation Multifocal atrophy, more in the antral region.	ulcers Stomach cancer

chemical damage NSAIDs Bile reflux Alcohol	direct damage Damage to the mucous layer Degranulation of mast cells	Pituitary epithelium hyperplasia edema vasodilatation	A small number of inflammatory cells erosion stomach ulcers

Ulcer disease of the stomach and duodenum

ulcers are violations of the integrity of the epithelial cover and underlying tissues of the digestive tract as a result of their damage by acid and pepsin. Ulcers are divided into acute and chronic.

acute ulcers

The reason for the development of acute ulcers can be:

1. Severe course of acute gastritis.

The deep spread of erosion in acute gastritis occurs mostly with the use of non-steroidal anti-inflammatory drugs (NSAIDs) or alcohol, with treatment with corticosteroids, which leads to the appearance of deep ulcers.

2. Strong stress. Acute ulcers can occur as a result of the action of various factors that lead to stress, for example, with widespread burns, brain injuries. In this case, ulcers are formed as a result of ischemia of the mucous membrane, which leads to a decrease in its resistance to acid.

3. Expressed increase in acidity. Increased acidity, for example, in patients with gastrin-secreting tumors (Zollinger-Ellison syndrome), leads to the formation of multiple ulcers in the antral part of the stomach, duodenum, and even the small intestine.

chronic ulcers

Chronic peptic ulcers are most often formed at the junction of different types of mucous membranes. So, for example, in the stomach, ulcers are observed at the point of transition of the body into the antrum, in the duodenum - in the proximal area at the border with the pylorus, in the esophagus - in the multilayered epithelium in front of the esophageal-gastric junction, postoperative ulcers are localized in the stoma (in conjunction). That is, ulcers appear in those places where acid and pepsin come into contact with an unprotected mucous membrane.

APPENDICITIS

Appendicitis is a primary inflammation of the appendix with a peculiar clinical syndrome. Therefore, not every inflammation of the appendix in clinical and anatomical terms should be considered as appendicitis (for example, when the inflammatory process spreads from nearby organs, when it is affected by tuberculosis, etc.).

There are two clinical and anatomical forms of appendicitis: acute and chronic.

acute appendicitis is the most frequent cause of emergency operations in surgery. It occurs in all age groups, but mostly in teenagers.

Most often, the causes of acute appendicitis are obstruction of the lumen of the appendix with fecal matter or an enlarged submucosa as a result of lymphoid

hyperplasia, as well as when the appendix is bent. At the same time, increased reproduction of microorganisms such as Escherichia coli, Streptococcus faecalis and anaerobic bacteria occurs in the distal segment. These bacteria then penetrate the mucosa and other membranes of the appendix, causing acute inflammation.
Pathomorphological changes. It is accepted to distinguish the following main morphological forms of acute appendicitis:
simple;
superficial;
destructive (which, in turn, is divided into phlegmonous, apostematous, phlegmonous-ulcerative, gangrenous).
Complication. Local spread of the inflammatory process can lead to involvement of periappendicular tissues, which is manifested by the development of "appendicular infiltrate" or abscess. As a result of perforation, peritonitis may develop, distant abscesses may form, most often in the rectal-bladder and subphrenic spaces. It is very rare to observe the spread of inflammation along the veins, which leads to the development of thrombophlebitis of the portal vein with the formation of multiple pylephlebitic liver abscesses.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Diseases of the esophagus: morphological characteristics.
2. Chronic gastritis: clinical and morphological characteristics, complications, causes of death.
3. Ulcer disease: clinical and morphological characteristics, complications, causes of death.
4. Tumors of the esophagus and stomach.
5. Enterocolitis: clinical and morphological characteristics, complications.
6. Tumors of the small and large intestine, morphological characteristics.
7. Appendicitis. Classification. Morphological characteristics of manifestations of acute and chronic appendicitis. Complication.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

The patient has peptic ulcer of the stomach, complicated by bleeding, and after endoscopy, liquid the color of coffee grounds was found in the stomach. What pigment caused this color of stomach contents?
The correct answer is hydrochloric acid hematin

B Hemosiderin
C Gemin
D Ferritin
E Porphyrins
In a patient with vomiting with blood, an ulcer penetrating into the muscular layer of the stomach was found during the operation on the stomach. The edges of the ulcer are dense, at the bottom - a bleeding blood vessel. During a cytobiopsy, scar tissue was found at the edges and at the bottom of the ulcer. What type of ulcer does the patient have?
Correct answer Chronic ulcer with bleeding
B Acute bleeding ulcer
C Malignant ulcer
DRupture of a stomach ulcer
E Penetrate into the ulcer
In a 33-year-old woman, an autopsy revealed thickening of the stomach wall in the pyloric section (layers of the wall are visible on the section) with the growth of dense whitish tissue in the submucosal base and small strands in the muscle layer. The relief of the mucous membrane is preserved, the folds are rigid and motionless. What form of macroscopic tumor is likely in this case?
Correct Answer Infiltrate
B Sosochkova
C Ulcer
D Cyst
E Infiltrative-ulcerative form
A 42-year-old patient complains of pain in the epigastric area, vomiting; vomiting masses of the color of "coffee grounds", melanorrhea. There is a history of peptic ulcer disease. In the blood: Er- 2.8 * 1012. Specify the most likely complication that developed in the patient:
The correct answer is Bleeding
B Pylorystenosis
C Cancer
D Perforation
E Penetration
A 45-year-old woman suffers from peptic ulcer disease. Suddenly I felt bad: sharp pain in the stomach, vomiting of "coffee grounds", cold sticky sweat, after a while, melanorrhea. What complication of stomach ulcer developed?
Correct Answer Bleeding from an ulcer
B Perforation of the ulcer
C Malignant change of the ulcer
D Ulcer penetration
E Stenosis of the stomach wall

A 40-year-old man had been suffering from peptic ulcer disease for a long time. Hellicobacter-associated chronic gastritis was diagnosed during endoscopy. Which of the following is a type of gastritis?
The correct answer is Type B
B Type A
C Type C
D Eosinophilic
E Granulomatous
A biopsy of the gastric mucosa revealed a decrease in the number of glands, growth of connective tissue. Preserved glands were placed in groups, their ducts were dilated. The mucosa is infiltrated with lymphocytes, plasma cells, and single neutrophils. Goblet cells and Paneth cells are observed in the gastric glands. Your diagnosis:
The correct answer is Menetrier's disease
B Chronic endogastritis
C Chronic gastrotrophy
D Squamous cell carcinoma of the stomach
E Adenosquamous gastric cancer
A 40-year-old man had been suffering from peptic ulcer disease for a long time. During endoscopy with biopsy, chronic gastritis type C was diagnosed. The cause of this gastritis is damage to the mucous membrane of the stomach as a result of exposure to:
Correct answer Bile acids
In Medicines
C Alcohol
D Pancreatic juice
E Spicy food
A 37-year-old woman suffered from pain in the right iliac fossa, nausea, vomiting. She was operated on for ulcerative and phlegmonous appendicitis. After 3 days, hectic fever appeared, pain in the right hypochondrium, pronounced ochroderma and visible mucous membranes, an increase in the level of direct and indirect bilirubin was detected in the blood. What complication of acute destructive appendicitis developed in this case?
Correct answer Pilephlebitic abscesses
B Abscesses of the subdiaphragmatic space
C Pelvic abscess
D General fibrinous-purulent peritonitis
E Viral hepatitis
During the autopsy of the corpse of a 28-year-old woman who suffered from acute appendicitis, multiple liver abscesses were discovered, which led to death. What morphological changes in the wall of the appendix and surrounding tissues led to the development of fatal complications?
Correct answer Thrombophlebitis of the appendix
B Perforation of the wall of the appendix
C Empyema of the appendix

D Thromboarteritis of the appendix
E Self-amputation of the appendix

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

16. methods: assessment of the correctness of the performance of practical skills
17. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the

	discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Diseases of the liver, pancreas, gall bladder.".

Suggested topics for essays:

1. Tumors of the stomach. Morphological characteristics.
3. Intestinal tumors. Morphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine

2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 27

Topic: Diseases of the liver, biliary system and pancreas.

Goal: To study pathomorphological signs, morphological classification, morphological characteristics of the most important diseases of organs: liver, biliary system and pancreas.

Basic concepts: Hepatosis. Classification. Morphological characteristics of manifestations of acute and chronic appendicitis. Complication. Hepatitis. Classification. Morphological characteristics of manifestations of acute and chronic hepatitis. Complication. Pancreatitis. Classification. Morphological characteristics of manifestations of acute and chronic pancreatitis. Complication.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Hepatosis is a liver disease characterized by dystrophy and necrosis of hepatocytes; it can be both hereditary and acquired.
A fairly significant group of hereditary hepatoses consists of the so-called metabolic diseases of the liver. They arise in connection with a violation of the metabolism of proteins and amino acids (cystinosis and aminoaciduria, or Debré - de Toni - Fanconi syndrome); fats (hereditary lipidosis), carbohydrates (glycogenosis), pigments (hereditary pigmentary hepatosis), minerals (hemochromatosis, hepatocerebral dystrophy, or Wilson-Konovalov disease). Most hereditary hepatoses are accumulation diseases and end in liver cirrhosis.
Hepatitis is a disease of the liver, the basis of which is its inflammation, which is manifested both by dystrophic and necrobiotic changes in the parenchyma, and by inflammatory infiltration of the stroma. By origin, hepatitis can be primary, occurring as an independent disease, and secondary - as a manifestation of another disease. Depending on the course, acute and chronic hepatitis are distinguished.
Cirrhosis of the liver is a chronic disease in which liver failure progressively increases due to cicatricial wrinkling and structural remodeling of the liver. The term "cirrhosis of the liver" (from the Greek Kirrhos - red) was proposed by R. Laenek (1819), referring to the peculiarities of the morphological changes of the liver (dense bumpy red liver).
Complication. Complications of liver cirrhosis include hepatic coma, bleeding from dilated veins of the esophagus or stomach, ascites-peritonitis, portal vein thrombosis, and the development of cancer. Most of these complications cause the death of patients.
Pancreatitis - inflammation of the pancreas - depending on the course is acute or chronic.

Acute pancreatitis develops when the outflow of pancreatic secretions is disturbed (ductal dyskinesia), the penetration of bile into the excretory ducts of the gland (biliopancreatic reflux), alcohol poisoning, dietary disorders (overeating), etc. Morphological manifestations of changes in the gland are swelling, the appearance of white-yellow areas of necrosis (fat necrosis), hemorrhages, suppuration, false cysts, sequestrations. With the predominance of hemorrhagic changes that become diffuse, we are talking about hemorrhagic pancreatitis; purulent inflammation - about acute purulent pancreatitis; necrotic changes - pancreatic necrosis.

Chronic pancreatitis can be a consequence of acute relapses. Its causes are also infectious diseases and intoxications, metabolic disorders, poor nutrition, diseases of the liver, gall bladder, stomach and duodenum. In chronic pancreatitis, in contrast to acute, not destructively inflammatory, but sclerotic and atrophic processes combined with the regeneration of acinous cells and the formation of regenerative adenomas prevailed. Sclerotic changes lead to a violation of the patency of the ducts and the formation of cysts. Cicatricial deformation of the gland is associated with its calcification; at the same time, iron decreases, acquires cartilage density. With chronic pancreatitis, manifestations of diabetes mellitus are possible. Death of patients with acute pancreatitis occurs from shock or peritonitis.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Liver diseases: morphological characteristics.
2. Hepatosis: morphological characteristics.
3. Liver diseases: morphological characteristics.
4. Acute hepatitis: clinical and morphological characteristics, complications, causes of death.
5. Chronic hepatitis: clinical and morphological characteristics, complications, causes of death.
6. Liver cirrhosis, forms, complications, causes of death.
7. Acute pacreatitis: clinical and morphological characteristics, complications.
8. Chronic pancreatitis: morphological characteristics, complications.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

The patient was diagnosed with ascites, a twice-enlarged spleen, and varicose veins of the esophagus and rectum. Micronodular cirrhosis was detected in the histological examination of a liver biopsy. What pathological process is complicated by liver cirrhosis?

Correct answerPortal hypertension syndrome
B Pulmonary heart
C Hepato-lanolin syndrome
D Heart failure
E Hepatocellular failure

Hydropic dystrophy of hepatocytes, "wax-like hepatocytes", acidophilic Rada bodies, and macrophage-lymphocyte infiltration in the portal tracts were histologically detected in the liver of the deceased, who received numerous injections of drugs during his life. The most likely etiology of the disease:

Correct answerToxic
B Bacterial
C Viral
D Parasitic
E Fungal

Macroscopically, the liver is enlarged, dense, grayish-yellow in color, the cut surface is sebaceous. What pathological process underlies these changes?

Correct answerAmyloidosis
B Mucoid edema

C Hemachromatosis
D Hyalinosis
E Fatty dystrophy
In a 45-year-old woman who died of chronic alcohol intoxication, an autopsy revealed a sharply enlarged liver of a mushy consistency and a yellowish color.
Microscopically: the cytoplasm of hepatocytes when stained with hematoxylin and eosin contains optically empty vacuoles of various sizes. What type of dystrophy?
Correct answerParenchymatous fat
B Carbohydrate parenchymatous
C Hyaline droplet
D Mesenchymal fat
E Hydropic
A 59-year-old man with parenchymal jaundice and portal hypertension. Histological examination of the liver biopsy revealed: the typical structural architectonics of classic lobules is disturbed, part of the hepatocytes has signs of fatty dystrophy, the formation of porto-portal connective tissue membranes with the formation of pseudolobules, with the presence of periportal lymph. macrophage infiltrates. Diagnose liver disease?
Correct answerCirrhosis
B Viral hepatitis
C Toxic clothing
D Alcoholic hepatitis
E Chronic hepatotoxicity
Liver biopsy of a patient with liver-cell failure revealed vacuolar, balloon degeneration of hepatocytes, necrosis of individual cells, Councilman bodies, infiltration of stroma of portal and lobular particles mainly by lymphocytes and macrophages with a small number of polymorphonuclear leukocytes. What is the most likely diagnosis?
Correct answerAcute viral hepatitis
B Chronic persistent hepatitis
C Autoimmune hepatitis
D Chronic active hepatitis
E Alcoholic hepatitis
A puncture biopsy of the liver revealed degeneration of hepatocytes with necrosis and sclerosis with a violation of the typical lobular structure with the formation of false lobules and regenerative nodes. Choose the most likely diagnosis:
Correct answerCirrhosis
B Chronic hepatitis
C Chronic hepatotoxicity
D Acute hepatitis
E Progressive massive liver necrosis
A 38-year-old patient with severe jaundice has small hemorrhages on the skin, general weakness, and loss of appetite. A liver biopsy was performed. Histological examination revealed widespread degeneration, necrosis of hepatocytes, and the presence of Councilman's bodies. A significant infiltration of lymphocytes, individual

multinucleated hepatocytes was found on the periphery of the lobules. What is the most likely diagnosis?
Correct answerAcute viral hepatitis
B Acute alcoholic hepatitis
C Toxic degeneration of the liver
D Chronic hepatitis
E Miliary cirrhosis
During the autopsy of the corpse of a man who abused alcohol for a long time, it was found: a liver of small size, dense, nodular shape. Microscopically: small pseudoparticles separated by narrow layers of connective tissue with lympho-macrophagic infiltration; hepatocytes in a state of globular fatty dystrophy. What is the most likely diagnosis?
Correct answerAlcoholic cirrhosis
B Alcoholic chronic persistent hepatitis
C Steatosis
D Chronic active alcoholic hepatitis
E Toxic degeneration of the liver
The patient was admitted with complaints of diarrheal disorders, melena, and hemorrhoidal bleeding. During the examination, an increase in the abdomen, expansion of the network of venous vessels of the anterior abdominal wall was revealed. What pathology has such symptoms?
Correct answerPortal hypertension
B Ulcer disease
C Intestinal autointoxication
D Enteritis
E Toxic degeneration of the liver

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

18. methods: assessment of the correctness of the performance of practical skills
19. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson: "Kidney diseases" is emphasized.

Suggested topics for essays:
1. Tumors of the pancreas.Morphological characteristics.
2. Liver tumors. Morphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 28

Topic: Kidney diseases.

Goal: To study pathomorphological signs, morphological classification, morphological characteristics of the most important kidney diseases.

Basic concepts: Kidney diseases: Classification. Glomerulopathy. Tublopathy. Interstitial kidney diseases. Glomerulonephritis, classification, morphological

characteristics. Nephrotic syndrome, morphological characteristics, complications. Amyloidosis, morphological characteristics. Tubolopathies: clinical and morphological characteristics, complications, causes of death. Acute and chronic renal failure: clinical and morphological characteristics, complications, causes of death. Pyelonephritis, classification, clinical and morphological characteristics, complications. Urinary stone disease, morphological characteristics, complications. Nephrosclerosis, morphological characteristics, complications, outcome. Kidney tumors.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Glomerulonephritis is a disease of an infectious-allergic or unknown nature, the basis of which is bilateral diffuse, rarely focal, non-purulent inflammation of the glomerular apparatus (glomerulus) with characteristic renal and extrarenal symptoms. Renal symptoms: oliguria, proteinuria, hematuria, cylindruria. Extrarenal symptoms: arterial hypertension, left ventricular hypertrophy, dysproteinemia, edema, hyperazotemia, uremia.

Clinical forms of glomerulonephritis: hematuric, nephrotic (nephrotic syndrome), hypertensive, mixed.

The classification of glomerulonephritis takes into account: 1) belonging to the nosology (primary - an independent disease, secondary - a manifestation of another disease); 2) nature of established etiology - bacteria, viruses, protozoa and undetermined etiology; 3) pathogenesis (immunologically conditioned and immunologically unconditioned); 4) course (acute, subacute, chronic); 5) morphology (topography, nature and spread of the inflammatory process).

a complication of both acute and subacute glomerulonephritis is acute renal failure. Chronic glomerulonephritis is characterized by chronic renal failure with manifestations of azotemic uremia. Cardiovascular failure and brain hemorrhage are also possible, which causes death.

The outcome of acute glomerulonephritis is mostly favorable - recovery of the patient; subacute and chronic - quite often chronic kidney failure develops.

nephrotic syndrome

Nephrotic syndrome is characterized by: high proteinuria, lipoproteinemia, hypoproteinemia, hyperlipidemia (hypercholesterolemia) and edema.
Classification. Primary (idiopathic) nephrotic syndrome is distinguished as an independent disease, and secondary - as a manifestation of kidney disease (glomerulonephritis, amyloidosis, etc.). The primary nephrotic syndrome includes: lipoid nephrosis (nephropathy with minor changes), membranous nephropathy (membranous glomerulonephritis) and focal membranous sclerosis (hyalinosis). Lipoid nephrosis occurs both in children and adults.
amyloidosis of the kidneys is one of the manifestations of general amyloidosis with bright clinical, morphological and nosological specificity (nephropathic amyloidosis).
complication:
-infectious diseases (pneumonia, mumps) associated with a decrease in body resistance and metabolic disorders;
- heart failure, hemorrhages, heart attacks;
- thrombosis of the venous system of the kidneys;
-acute renal failure (the cause is excessive accumulation of protein breakdown products in the blood, reduction of renal blood circulation, vascular disorders and intercurrent diseases).
death occurs as a result of chronic renal failure and uremia in the last stage of the disease, acute renal failure, infectious diseases.
Acute renal failure
Acute renal failure is a syndrome that is characterized by necrosis of the tubule epithelium and deep disorders of blood and lymph circulation. Acute renal failure is equated with necrotic nephrosis (necronephrosis). Complication: Segmental (total) necrosis of the cortical substance of the kidneys.
result: In the case of hemodialysis treatment, acute renal failure usually ends with recovery. In some cases, death from uremia in the shock or oligouric stage is possible. After a few years, scarring of the kidneys can develop, then patients die from chronic kidney failure.
Interstitial nephritis is the inflammation of mainly the interstitial tissue of the kidneys, with successive involvement of the entire nephron in the pathological process. Among the diseases of this group, tubulo-interstitial nephritis and pyelonephritis are the most important.
Classification. There is a distinction between primary (an independent disease) and secondary (often with systemic lupus erythematosus, rheumatoid arthritis and Goodpascher's syndrome, kidney rejection reactions) nephritis. The course is acute and chronic tubulo-interstitial nephritis.
Pyelonephritis is an infectious disease in which the pelvis, its calyces, and the substance of the kidneys are involved in the pathological process, with predominant damage to the interstitial tissue of the kidney. Depending on the spread of the pathological process, it can be unilateral or bilateral. There is a distinction between acute and chronic pyelonephritis, which mostly takes on a recurrent course in the form of acute attacks.

complications: in acute pyelonephritis, the progress of the purulent process leads to the fusion of abscesses and the formation of a carbuncle of the kidney, the connection of purulent cavities from the bowl (pyonephrosis), the transition of the process to the fibrous capsule (perinephritis) and renal tissue (paranephritis). Sometimes necrosis of the papillae of the pyramids (papillonecrosis) develops as a result of the toxic effect of bacteria in conditions of urinary stasis. Sometimes pyelonephritis becomes a source of sepsis. When the purulent process is limited, chronic kidney abscesses may develop. With chronic pyelonephritis, especially one-sided, nephrogenic arterial hypertension and arteriolosclerosis may occur in the second (unchanged) kidney. Bilateral pyelonephritic shrinkage of the kidneys ends in chronic renal failure.
result. During the treatment of patients with acute pyelonephritis, recovery occurs. Severe complications - pyonecrosis, sepsis, papillonecrosis - can be the cause of death of patients. Chronic pyelonephritis ends with azotemic uremia. In the case of the development of nephrogenic arterial hypertension, the causes of death are the same as in hypertensive disease (stroke, myocardial infarction, etc.).
Urinary stone disease (nephrolithiasis) is a disease with a chronic course, in which stones of different nature, structure and chemical composition are formed in the calyces, bowls of the kidneys, and ureters of one or both kidneys.
nephrosclerosis - compaction and deformation (shrinking) of the kidneys as a result of the development of connective tissue in them.
Pathological anatomy is the pathological anatomy of extrarenal excretory systems (skin, mucous membranes). At the autopsy of a patient with uremia, the smell of urine is felt, the reaction with xanthhydrol allows to detect urea in all organs, especially in the lungs, stomach, and spleen. Volatile ammonia compounds with concentrated hydrochloric acid form ammonium chloride vapors in the form of clouds. The skin is gray-earthy due to the accumulation of urochrome. Sometimes, especially on the face, it seems to be powdered. Hemorrhages and rashes often appear in the skin as manifestations of hemorrhagic diathesis. Uremia, laryngitis, tracheitis, gastroenteritis and pneumonia are observed, which are fibro-necrotic or fibro-hemorrhagic; characteristic uremic pulmonary edema. Fatty dystrophy in the liver. Serous, serous-fibrinous, fibrinous pericarditis, myocarditis, uremia are often found. The development of uremic pleurisy and peritonitis is possible. The brain is pale, swollen, with areas of softening and hemorrhage. The spleen is enlarged, resembling a septic one. Uremia develops not only with chronic, but also with acute renal failure. It is also observed in eclampsia and chlorohydropenia. Chronic renal failure can last for many years with hemodialysis. At the same time, the patient is in the stage of chronic suburemia. At the same time, pathological anatomical changes acquire a different character. Metabolic injuries (myocardial necrosis), productive inflammation (valvular pericarditis, obliteration of cavities around the heart sac) dominate; bone changes (osteoporosis, osteosclerosis), sometimes general amyloidosis, changes in the endocrine system (adaptive hypertrophy of the parathyroid glands). The brain is pale, swollen, with areas of softening and hemorrhage. The spleen is enlarged, resembling a septic one. Uremia develops not only with chronic, but also with acute renal failure. It is also observed in

eclampsia and chlorohydropenia. Chronic renal failure can last for many years with hemodialysis. At the same time, the patient is in the stage of chronic suburemia. At the same time, pathological anatomical changes acquire a different character. Metabolic injuries (myocardial necrosis), productive inflammation (valvular pericarditis, obliteration of cavities around the heart sac) dominate; bone changes (osteoporosis, osteosclerosis), sometimes general amyloidosis, changes in the endocrine system (adaptive hypertrophy of the parathyroid glands). The brain is pale, swollen, with areas of softening and hemorrhage. The spleen is enlarged, resembling a septic one. Uremia develops not only with chronic, but also with acute renal failure. It is also observed in eclampsia and chlorohydropenia. Chronic renal failure can last for many years with hemodialysis. At the same time, the patient is in the stage of chronic suburemia. At the same time, pathological anatomical changes acquire a different character. Metabolic injuries (myocardial necrosis), productive inflammation (valvular pericarditis, obliteration of cavities around the heart sac) dominate; bone changes (osteoporosis, osteosclerosis), sometimes general amyloidosis, changes in the endocrine system (adaptive hypertrophy of the parathyroid glands). Uremia develops not only with chronic, but also with acute renal failure. It is also observed in eclampsia and chlorohydropenia. Chronic renal failure can last for many years with hemodialysis. At the same time, the patient is in the stage of chronic suburemia. At the same time, pathological anatomical changes acquire a different character. Metabolic injuries (myocardial necrosis), productive inflammation (valvular pericarditis, obliteration of cavities around the heart sac) dominate; bone changes (osteoporosis, osteosclerosis), sometimes general amyloidosis, changes in the endocrine system (adaptive hypertrophy of the parathyroid glands). Uremia develops not only with chronic, but also with acute renal failure. It is also observed in eclampsia and chlorohydropenia. Chronic renal failure can last for many years with hemodialysis. At the same time, the patient is in the stage of chronic suburemia. At the same time, pathological anatomical changes acquire a different character. Metabolic injuries (myocardial necrosis), productive inflammation (valvular pericarditis, obliteration of cavities around the heart sac) dominate; bone changes (osteoporosis, osteosclerosis), sometimes general amyloidosis, changes in the endocrine system (adaptive hypertrophy of the parathyroid glands). At the same time, the patient is in the stage of chronic suburemia. At the same time, pathological anatomical changes acquire a different character. Metabolic injuries (myocardial necrosis), productive inflammation (valvular pericarditis, obliteration of cavities around the heart sac) dominate; bone changes (osteoporosis, osteosclerosis), sometimes general amyloidosis, changes in the endocrine system (adaptive hypertrophy of the parathyroid glands). At the same time, the patient is in the stage of chronic suburemia. At the same time, pathological anatomical changes acquire a different character. Metabolic injuries (myocardial necrosis), productive inflammation (valvular pericarditis, obliteration of cavities around the heart sac) dominate; bone changes (osteoporosis, osteosclerosis), sometimes general amyloidosis, changes in the endocrine system (adaptive hypertrophy of the parathyroid glands).

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Definition of glomerulopathies, general morphological characteristics.
2. Definition of tubolopathies, general morphological characteristics.
3. Definition of glomerulonephritis, classification.
4. Acute glomerulonephritis, morphological characteristics, complications, causes of death.
5. Subacute glomerulonephritis, morphological characteristics, complications, causes of death.
6. Chronic glomerulonephritis, morphological characteristics, complications, causes of death.
7. Renal amyloidosis, morphological characteristics, complications, outcome.
8. Nephrotic syndrome, morphological characteristics.
9. Acute renal failure, morphological characteristics, complications, causes of death.
10. Chronic kidney failure, morphological characteristics, complications, causes of death.
11. Pyelonephritis, morphological characteristics, complications, outcome.
12. Urolithiasis, morphological characteristics, complications.
13. Nephrosclerosis, morphological characteristics, complications, causes of death.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

The patient, who has been suffering from kidney disease for many years, died due to uremia. On autopsy: the kidneys are sharply reduced in size, dense, fine-grained, light gray in color. What are the kidneys with such changes called?

Correct answer Secondary wrinkled

BPrimarily wrinkled

CStroke

DSalni

E-

The kidney biopsy revealed: interstitial tissue infiltrated by leukocytes, miliary abscesses, tubules in a state of dystrophy, filled with desquamated epithelium and leukocytes. What disease can you think of?

Correct answer Pyelonephritis

B Glomerulonephritis

CPielit

DNecrotic nephrosis

Nephrolithiasis

The autopsy of a 52-year-old woman who had been suffering from chronic glomerulonephritis for a long time revealed: significantly reduced, dense, fine-grained

kidneys, fibrinous inflammation of the serous and mucous membranes, dystrophic changes in parenchymal organs, cerebral edema. What complications are caused by the described changes in the serous membranes and internal organs?
Correct answer Uremia
Anemia
Sepsis
DDVZ-syndrome
Thrombocytopenia
An autopsy of the deceased patient revealed an adenoma of the prostate gland and large kidneys with sharply enlarged balls and calyces filled with a clear liquid. Name the process in the kidneys.
Correct answer Hydronephrosis
Amyloidosis
Pyelonephritis
D Tuberculosis
E Glomerulonephritis
A 62-year-old man had a kidney removed, in which a macroscopic examination revealed a tumor in the form of a nodule with a diameter of up to 8 cm. The tissue of the tumor on the section is variegated, with multiple hemorrhages, necrosis. Histologically: the tumor consists of light cells that form alveolar and papillary structures, moderately expressed invasive growth. Pathological mitoses and hyperchromic nuclei are found in many tumor cells. Diagnose the detected kidney tumor.
Correct answer Clear cell cancer
Adenocarcinoma
Acidophilic adenoma with malignancy
DNephroblastoma
Clear cell adenoma
At the autopsy of a 25-year-old patient who died of uremia, the kidneys were enlarged, variegated, with foci of hemorrhages. Pathohistologically, hematoxylin bodies, capillary membranes of glomeruli in the form of wire loops, hyaline thrombi and foci of fibrinous necrosis were detected, in the vessels of the spleen - "onion" sclerosis. What is the most likely diagnosis?
Correct answer Systemic lupus erythematosus
BSystemic scleroderma
Rheumatic arthritis
DNodular periarteritis.
Rheumatoid arthritis
An autopsy revealed a significant increase in the volume of the right kidney. A stone was found on the incision. The lumen of the renal pelvis is stretched by urine. The kidney parenchyma is sharply thinned. What is the most accurate diagnosis?
Correct answer Hydronephrosis
Amyloidosis

CHydrourethronephrosis
Pyeloectasia
EKidney cyst
At the autopsy of the deceased, who suffered from cystitis and dyskinesia of the ureters, morphological signs of uremia were found. The kidney is unevenly scarred and wrinkled. There are small urate stones and sand in the lumen of the balls. Histologically, a "thyroid kidney" and foci of interstitial inflammation were detected. Which of the following diagnoses is most likely?
Correct answer Chronic pyelonephritis
BAmloid shrunken kidney
Atherosclerotic shrunken kidney
Acute pyelonephritis
EPrimary shriveled kidney
A patient with fibrous-cavernous tuberculosis died with increasing symptoms of renal failure. At the autopsy - the smell of urine, hypertrophy of the left ventricle, fibrinous pericarditis, fibrinous and hemorrhagic enterocolitis. The kidneys are slightly reduced in size, very dense, with multiple involvements. Histologically, on preparations stained with congo-rot, pink masses in glomeruli or vessel walls, death and atrophy of most nephrons, nephrosclerosis. Describe the kidney in this pathology:
Correct answer Amyloid shrunken kidneys
BSecondary shrunken kidneys
CPyelonephritic shriveled kidneys
D Atherosclerotic shrunken kidneys
EPrimarily shrunken kidneys
In a patient with chronic cystitis, foci of multi-layered flat non-keratinized epithelium were found in the biopsy of the mucous membrane of the urinary bladder together with the transitional epithelium. What process underlies the described changes in the epithelium?
Correct answer Metaplasia
BHyperkeratosis
Hyperplasia
Dystrophies
Dysplasia
At the autopsy, it was found that the kidneys were enlarged, the surface was large and bumpy due to the presence of multiple cavities with smooth walls, filled with a transparent liquid. What disease are we talking about?
Correct answer Polycystic disease
Pyelonephritis
Necrotic nephrosis
D Glomerulonephritis
Heart attack
A 49-year-old woman suffered from chronic glomerulonephritis for a long time, which caused her death. At the autopsy, it was established that the kidneys have dimensions

of 7x3x2.5 cm, weight 65 g, dense, fine-grained. Microscopically: fibrinous inflammation of serous and mucous membranes, dystrophic changes in parenchymal organs, cerebral edema. What complication led to the specified changes in the serous membranes and internal organs?
Correct answer Uremia
B Thrombocytopenia
Sepsis
Anemia
EDVZ-Syndrome
A 19-year-old man suffered from bronchiectasis since early childhood. Died of kidney failure. At autopsy, in addition to multiple bronchiectatic cavities filled with purulent exudate, enlarged kidneys of a dense consistency were found, the cortical layer was thickened, white in color, and dense. Kidney pyramids are anemic, clear. Name the process that developed in the kidneys?
Correct answer Secondary amyloidosis
B Chronic pyelonephritis
Secondary nephrosclerosis
DCongenital cystic kidney
E Glomerulonephritis

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

20. methods: assessment of the correctness of the performance of practical skills
21. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Hypothalamic-pituitary disorders. Adrenal gland pathology. Pathology of the thyroid gland. Pathology of the endocrine apparatus of the pancreas.".

Suggested topics for essays:

1. Kidney tumors. Morphological characteristics.
2. Polycystic kidney disease.Morphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 29

Topic: Hypothalamic-pituitary disorders. Adrenal gland pathology. Pathology of the thyroid gland. Pathology of the endocrine apparatus of the pancreas.

Goal: learn to determine the etiology, pathogenesis, morphology of endocrine gland diseases, as well as their complications.

Basic concepts: Hypothalamic-pituitary disorders, pathology of the adrenal glands, pathology of the thyroid gland, pathology of the endocrine apparatus of the pancreas. Classification,morphological characteristics, complications, causes of death.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Adrenal glands

In the cortex of the adrenal glands, mineralocorticosteroids (aldosterone), glucocorticoids and sex hormones are formed, the secretion of which is controlled, respectively, by adrenocorticotropic and gonadotropic hormones of the anterior lobe of the pituitary gland. Strengthening of the tropical effects of the pituitary gland or the development of a hormonally active tumor of the adrenal cortex leads to their hyperfunction, and the weakening of these effects or the destruction of the adrenal cortex leads to hypofunction. The secretion of hormones of the medulla of the adrenal glands (adrenaline, norepinephrine) is stimulated by the sympathetic nervous system. Its hypofunction is well compensated by chromaffin tissue, hyperfunction is associated with a tumor (pheochromocytoma) (see Tumors of endocrine glands).

Addison's disease (named after the English doctor T. Addison, who described this disease in 1849), ilibronzo disease. The disease is caused by bilateral damage mainly to the cortical substance of the adrenal glands and shutdown (acorticism) or decrease (hypoadrenocorticism) of its hormone production. The most common causes of bronze disease are tumor metastases in both adrenal glands, their autoimmune damage (primary Addison's disease), amyloidosis (epinephric amyloidosis), hemorrhages, necrosis due to vascular thrombosis, tuberculosis.

Death in Addison's disease occurs from acute adrenal insufficiency, cachexia (suprarenal cachexia), or cardiovascular failure.

Goiter (Struma) is a pathological enlargement of the thyroid gland.

Classification of goiter takes into account, on the one hand, morphological features, on the other - epidemiology, causes, functional and clinical features.

Based on morphological features, diffuse, nodular and diffuse-nodular (mixed) goiters are distinguished by their appearance, colloidal and parenchymatous by histological structure.

Diffuse toxic goiter (Bazedov's disease, Graves' disease) is the most striking manifestation of the hyperthyroidism syndrome, therefore it is also called therotoxic goiter. The reason for its development is autoimmunization: autoantibodies stimulate cell receptors of thyrocytes. This makes it possible to attribute diffuse toxic goiter to "antibody receptor diseases".

Morphological features of diffuse toxic goiter are revealed only by microscopic examination. These include the transformation of the prismatic epithelium of the follicles into a cylindrical one; proliferation of the epithelium with the formation of papillae branched inside the follicles; vacuolization and change in tinctorial properties of the colloid (poorly perceives dyes) due to its dilution and iodine depletion; lymphoplasmacytic infiltration of the stroma, formation of lymphatic follicles with germinal centers.

A number of visceral manifestations are found in Basedov's disease. In the heart, the myocardium of which is hypertrophied (especially the left ventricle), in connection with thyrotoxicosis, serous edema and lymphoid infiltration of the interstitial tissue, as well as intracellular swelling of muscle fibers, are observed - Thyrotoxic heart. Diffuse intermediate sclerosis develops as a result. Serous edema is also observed in the liver, rarely resulting in fibrosis (thyrotoxic liver fibrosis). Dystrophic changes in nerve cells, perivascular cell infiltrates are found in the intermediate and medulla oblongata. Thymus gland enlargement, hyperplasia of lymphoid tissue, and atrophy of the adrenal cortex are often found.

Death with diffuse toxic goiter can occur from heart failure, exhaustion. Acute adrenal insufficiency may develop during goitre removal surgery.

Thyroiditis. This is a group of diseases, among which the main importance is Hashimoto's thyroiditis, or Hashimoto's disease - a real autoimmune disease.

Diabetes mellitus (sugar disease) is a disease caused by a relative or absolute lack of insulin.

Classification. The following types of diabetes are distinguished: spontaneous, secondary, gestational diabetes and latent (subclinical). Among spontaneous diabetes, type I diabetes (insulin-dependent) and type II diabetes (insulin-independent) are distinguished. Secondary diabetes is called diabetes in diseases of the pancreas (pancreatic diabetes), diseases of the endocrine system (acromegaly, Itsenko-Cushing syndrome, pheochromocytoma), complex genetic syndromes (Louis-Bar ataxia-telangiectasia, myotonic dystrophy, etc.), when using a number of medicinal funds (medical diabetes). Odiabetes of pregnant women is said to occur during pregnancy when impaired glucose tolerance begins, and so-called latent (subclinical) diabetes is caused by impaired glucose tolerance in seemingly healthy people.

Complication. Complications of diabetes are diverse. Possible development of diabetic coma. Complications due to macro- and microangiopathy (gangrene of the limb, myocardial infarction, blindness), especially diabetic nephropathy (renal failure - acute with papillonecrosis, chronic with glomerulosclerosis) often occur. Patients with diabetes easily develop infections, especially purulent ones (pyoderma, furunculosis, sepsis), often have exacerbation of tuberculosis with generalization of the process and predominance of exudative changes.

Death in diabetes occurs from complications. Diabetic coma is now rare. Most often, patients die from gangrene of a limb, myocardial infarction, uremia, complications of an infectious nature.

Pituitary disorders are associated with a tumor of the pituitary gland, its autoimmune

immune lesion, inflammation, necrosis (ischemic heart attack)
or develop as a result of damage to the hypothalamus or other departments
central nervous system Therefore, in some cases, we can talk about the brain and
hypothalamus
pituitary diseases: acromegaly, pituitary dwarfism, cerebro-
ral-pituitary cachexia, Itsenko-Cushing's disease, adiposogenic
mental dystrophy, diabetes insipidus, pituitary tumors.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Hypothalamic-pituitary disorders, etiology, morphological characteristics.
2. Goitre, classification, morphological characteristics.
3. Diffuse toxic goiter, morphological characteristics, complications, causes of death.
4. Thyroiditis, morphological characteristics, complications.
5. Diabetes mellitus, classification, etiology.
6. Diabetes, type 1, morphological characteristics.
7. Complications, the result of diabetes, type 1.
8. Diabetes, type 2, morphological characteristics.
9. Complications as a result of type 2 diabetes.
10. Addison's disease, morphological characteristics, complications, causes of death.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

A 50-year-old patient complains of thirst, drinks a lot of water, secretes a lot of urine (6-8 liters per day). Glucose in the blood is 4.8 mmol/l, there is no glucose or ketone bodies in the urine. Insufficient function of which gland can be the cause of the specified clinical changes?

Correct answer Neurohypophysis
B Thyroid gland
Adenohypophysis
D Parachute
EPancreatic

An excess of somatotropin hormone was found in the young man, the nose, lips, ears, lower jaw, hands and feet were enlarged. Your diagnosis:

Correct answer Acromegaly
BAdiposogenital dystrophy
Itsenko-Cushing's disease
D Pituitary dwarfism
E Addison's disease

The patient had a 2-fold increase in the thyroid gland. On palpation, the gland is dense, the surface is unevenly hilly. In histological examination - diffuse infiltration of the gland tissue by lymphocytes, plasma cells with the formation of follicles and increased growth of connective tissue. What disease does the patient have?
Correct answer Hashimoto's goiter
BDiffuse toxic goiter
CZob Riedel
Endemic goiter
E Sporadic gob
A histological examination of the thyroid gland removed during surgery revealed destruction and atrophy of the follicles. Diffuse lymphocytic infiltration with the formation of lymphoid follicles in the stroma. To which group of diseases does this thyroiditis belong?
Correct answer Viruses
Bacterial
Autoimmune
D Infectious and allergic
E Caused by physical factors
A 42-year-old patient had a significant increase in the size of the nose, ears, lower jaw, and feet. What disease can be suspected?
Correct answer Acromegaly
BAdiposogenital dystrophy
Gigantism
Dnanism
ECerebral cachexia
The patient has an elevated blood glucose level, and sugar is present in the urine. A kidney biopsy revealed: expansion of the mesangium with a focal accumulation of a membrane-like substance with periglomerular sclerosis of some glomeruli, hyalinosis and plasma leakage of arterioles, lymphohistiocytic infiltration of the stroma with the presence of polymorphonuclear leukocytes; glycogen infiltration of nephrocytes of a narrow segment. What is the most likely diagnosis?
Correct answer Diabetic glomerulosclerosis
BAcute glomerulonephritis
Chronic glomerulonephritis
Pyelonephritis
Subacute glomerulonephritis
.
A patient with the upper type of obesity had long-term hypertension, hyperglycemia, and glucosuria. Death came from a brain hemorrhage. Pathomorphological examination revealed a basophilic adenoma of the pituitary gland, hyperplasia of the adrenal cortex. What is the most likely diagnosis?
Correct answer Itsenko-Cushing's disease
Acromegaly

C Pituitary dwarfism
Diabetes mellitus
EAdiposogenital dystrophy
An autopsy of a 40-year-old woman who died of a cerebral hemorrhage during a hypertensive crisis revealed obesity of the upper type, hypertrichosis and hirsutism, striae on the skin of the thighs and abdomen. Basophilic adenoma is located in the anterior part of the pituitary gland. Which of the listed diagnoses is the most likely?
Correct answer Itsenko-Cushing's disease
B Cerebral obesity
Alimentary obesity
DSimmonds disease
Hypertensive disease
During the examination of the 32-year-old patient, disproportionate skeletal dimensions, an increase in the browbones, nose, lips, jawbones, and feet were noted. Which gland's function is impaired?
Correct answer Pituitary
BPancreatic
C Shield-shaped
D Adrenal glands
Epiphysis
The patient is concerned about polyuria (7 liters per day) and polydipsia. During the examination, no disorders of carbohydrate metabolism were found. Dysfunction of which endocrine gland can be the cause of these disorders?
Correct answer Neurohypophysis
BAdenohypophysis
Adrenal cortex
D The adrenal medulla
Pancreatic islets

Topic Diseases of the female and male reproductive system. Diseases of pregnancy and the postpartum period.
A histological examination of a scraping of the walls of the uterine cavity of a 45-year-old woman with ovarian and menstrual cycle disorders revealed an increase in the number of endometrial glands, some of which are convoluted, some of which are cystically enlarged. Diagnose the disease.
Correct answer Glandular - cystic hyperplasia of the endometrium
Adenocarcinoma of the endometrium
Atypical endometrial hyperplasia
Placental polyp
Endometrial glandular polyp

Microscopic examination of the cervical biopsy revealed cellular and nuclear atypia of the multilayered squamous epithelium, pathological mitoses, as well as horn pearls in the depth of the epithelial layers. Your diagnosis:
Correct answer Squamous cell carcinoma with keratinization
BTransitional cell cancer
Squamous cell carcinoma without keratinization
Glandular cancer
Anaplastic cancer.
A histological examination of a scraping of the uterine mucosa of a 54-year-old patient with a clinical diagnosis of ovarian-menstrual cycle disorder revealed the growth of atypical glandular structures consisting of cells with hyperchromic nuclei, mitotic figures, and atypia. Atypical glandular structures grow into the myometrium. Which pathological process is characterized by microscopic changes?
Correct answer Adenocarcinoma of the uterus
BAcute endometritis
Chorioepithelioma of the uterus.
Placental polyp
Glandular hyperplasia of the endometrium
In a 46-year-old woman, during a palliative operation for stomach cancer, the presence of Krukenbergian metastases in the ovaries ("Krukenbergian ovarian cancer") was established. which of the following routes of metastasis led to ovarian damage?
Correct answer Implantation
BLymphogenic orthograde
CLymphogenic retrograde
DHematogenous
Canalicular
In connection with acute pain in the iliac region, a young woman had her fallopian tube removed with a local expansion of its middle third, filled with blood. During histological examination, chorionic villi, large fields of erythrocytes with an admixture of leukocytes were found in the opening of the tube. Your diagnosis:
Correct answer Tubal pregnancy
BAcute purulent salpingitis
Bleeding in the fallopian tube
D Hemorrhagic salpingitis
Purulent salpingitis
The autopsy of a 73-year-old man revealed an enlarged, soft, elastic, slightly bumpy prostate gland, which on cross-section consists of separate nodes separated by layers of connective tissue. Microscopy revealed an increase in the number of glandular elements. The size of the particles and the number of glandular elements in them are different. What process takes place in the prostate gland?
Correct answer Glandular nodular hyperplasia
BMuscular - fibrous (stromal) nodular hyperplasia
C Mixed nodular hyperplasia

Adenocarcinoma
Undifferentiated cancer
During histological examination of the prostate gland surgically removed from a 72-year-old man who complained of difficult urination, an increase in the number of glandular and muscular elements was found. The lobular structure of the gland is disturbed. What is the most likely process in the prostate gland?
Correct answer Mixed form of prostatopathy
BMuscular-fibrous hyperplasia
Glandular hyperplasia
Prostatitis
Adenocarcinoma
The patient was 42 years old, suffered from menometrorrhagia, and a supravaginal amputation of the uterus was performed. Macroscopic examination revealed multiple intramural and submucosal dense nodes in the uterus, 1 to 5 cm in size, whitish in section, fibrous structure. Microscopically, they are represented by randomly arranged bundles of smooth muscle fibers. Your diagnosis?
Correct answer Multiple leiomyoma
B Chorion carcinoma
CFibroma
D Polyp
Endocervicosis
A 57-year-old female patient developed periodic uterine bleeding. For diagnostic purposes, the uterine cavity was scraped. In the obtained material, glandular complexes of various sizes and shapes, formed by atypical cells with hyperchromic nuclei, with numerous mitoses (including irregular ones) are observed among the blood elements. Your diagnosis:
Correct answer Cancer of the body of the uterus (adenocarcinoma)
BUterine fibromyoma
Chorioepithelioma
Glandular hyperplasia of the endometrium
Endometritis
The 23-year-old patient's condition deteriorated sharply the next day after giving birth, her body temperature rose to 39.0C, discharge from the uterus was yellow-green with an unpleasant smell. Microscopic examination of the endometrial scraping revealed continuous neutrophilic granulocytes. Your diagnosis:
Correct answer Acute endometritis
BGlandular hyperplasia of the endometrium
Cancer of the body of the uterus
D Physiological state
Sepsis

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

22. methods: assessment of the correctness of the performance of practical skills
23. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .

"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Pathomorphological changes in diseases related to nutrition." Vitamins Occupational diseases. Radiation sickness. Parathyroid osteodystrophy, osteomyelitis, fibrous dysplasia, osteopetrosis, Paget's disease, muscular dystrophies, myasthenia.

Suggested topics for essays:

1. Itsengo-Cusheng syndrome and disease. Morphological characteristics.
2. Addison's disease.Morphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization

4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 30

Topic: Pathomorphological changes in diseases related to nutrition. Vitamins Occupational diseases. Radiation sickness. Parathyroid osteodystrophy, osteomyelitis, fibrous dysplasia, osteopetrosis, Paget's disease, muscular dystrophies, myasthenia.

Goal:learn to determine etiology, pathogenesis, morphologydiseases related to nutrition, vitamin supplements, occupational diseases, radiation sickness, Paget's disease, muscular dystrophy, myasthenia.

Basic concepts: Pathomorphological changes in diseases related to nutrition, vitamin deficiency, occupational diseases, radiation diseases and Paget's disease, muscular dystrophies, myasthenia.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Diseases of the bone system

The disease of this system can be caused by:
1. ***Dystrophic:***toxic (Urov disease), alimentary (rickets),
endocrine, nephrogenic. A significant place belongs to parathyroid osteodystrophy.
2. Incendiary
3. Dysplastic: fibrous dysplasia of bones, osteopetrosis, Paget's disease.
4. Neoplastic - often develop against the background of dysplastic.

Parathyroid osteodystrophy

Parathyroid osteodystrophy (Recklinghausen's disease, generalized osteodystrophy) is a disease caused by hyperfunction of the parathyroid glands and accompanied by generalized damage to the skeleton. It occurs mainly in women aged 40-50.

Etiology. Parathyroid osteodystrophy is a consequence of primary hyperparathyroidism caused by adenoma of parathyroid glands or hyperplasia of gland cells.

*Pathogenesis.*Increased parathyroid hormone synthesis causes hypercalcemia with progressive demineralization of the entire skeleton. In the bone tissue, osteoclasts are activated, diffuse fibroosteoclasy increases - bone tissue is replaced by fibrous connective tissue. Bone deformation, osteoporosis, pathological fractures are possible. Formations resembling giant cell tumors appear in the changed cells. They are reactive structures that are built by giant cell granulomas.

Hypercalcemia leads to the development of calcareous metastases. Nephrocalcinosis often develops.

*Pathological anatomy.*Adenoma, rarely cell hyperplasia, is often found in the parathyroid glands. Morphological changes of the skeleton depend on the stage and course of the disease. In the initial stage, they are completely absent, then they find deformation of the bones, especially the limbs, spine, ribs. They become soft, easily cut with a knife.

During microscopic examination, foci of lacunar resorption, neoplasms of fibrous tissue are found in bone tissue, giant cell granulomas, accumulation of erythrocytes and hemosiderin are possible in tumor-like formations.

The death of patients occurs from cachexia or uremia due to shrinkage of the kidneys.

Osteomyelitis

Osteomyelitis is an inflammation of the bone marrow, which spreads to the spongy and compact substance of the bone and to the periosteum. According to the course, acute and chronic osteomyelitis are distinguished, according to the mechanism of infection - primary and secondary.

Primary hematogenous osteomyelitis

Acute hematogenous osteomyelitis is most common in young people. Chronic osteomyelitis is a consequence of acute.

*Etiology.*The causative agents of acute osteomyelitis are mostly purulent microbes: hemolytic staphylococcus, streptococcus, coliform bacilli, pneumococci, gonococci. It is most likely that patients with osteomyelitis have bacteremia with minor intestinal trauma, dental disease, and upper respiratory tract infection.

Pathogenesis. The purulent inflammatory process begins in the bone-marrow crevices of the metaphyses, where there is slowed blood circulation. Further, the process spreads to the bone marrow, where necrosis appears, and passes to the cortical layer of the bone, periosteum, and adjacent soft tissues.

*Pathological anatomy.*In acute hematogenous osteomyelitis, the inflammation has a phlegmonous nature. Resorption of bone tissue near the epiphyseal cartilage can end with the separation of the metaphysis from the epiphysis (epiphyolysis). Tissue infiltration by neutrophils appears around necroses; thrombi are found in the vessels of the compact plate. Abscesses often develop under the periosteum.
Chronic hematogenous osteomyelitis, as a result of acute, is accompanied by the formation of sequestrations, around which granulation tissue and a capsule are formed. From the sequestrations, fistulas go to the surface of the skin or to the body cavity. Along with the destruction of the bone in the periosteum and bone marrow canal, bone formation occurs - the bones become thick and deformed. Scars form in soft tissues.
Complications of primary hematogenous osteomyelitis: bleeding from fistulas, spontaneous fractures, formation of false joints, development of sepsis, secondary amyloidosis in chronic osteomyelitis.

Fibrous dysplasia

Fibrous dysplasia (fibrous osteodysplasia, Lichenschein-Breitsev disease) is a disease in which bone tissue is replaced by fibrous tissue, which leads to bone deformation.

*Etiology and pathogenesis.*The reasons for the development of the disease are unknown, perhaps hereditary factors are of some importance. It is believed that the tumor process is at the root of the disease. The disease begins in childhood, but can also develop in adults.
*Classification.*Depending on the spread of the pathological process, two forms of fibrous dysplasia are distinguished:
5. Monoosseous - pathological changes occur in only one bone. It can develop at any age.
6. Polyosseous - several bones are affected, mostly on one side of the body. Sometimes it is combined with melanosis of the skin. It develops in childhood.
*Pathological anatomy.*With the monoaxial form, pathological changes most often develop in the ribs, long tubular bones, shoulder blades, skull bones; with poliomyelitis - more than 50% of the bones of the skeleton, mainly on one side of the body. The damaged bone at the beginning of the disease retains its shape and size. In the future, "swelling", deformations of the bone, its lengthening or shortening appear. Femurs acquire the shape of a "shepherd's staff". On the cutting, clearly limited areas of whitish color with black-brown inclusions are determined. The bone marrow canal is expanded or filled with newly formed tissue. Upon microscopic examination, the centers of fibrous dysplasia are represented by fibrous fibrous tissue, which in some areas consists of randomly arranged bundles of mature collagen fibers and spindle-shaped cells, and in others - from thin collagen fibers and stellate cells. If fibrous dysplasia affects the bones of the face, then the dense component in the cells may be represented by cement-type tissue (cement-like formations).
*Complication*represented by pathological bone fractures, especially often in children, the femur is broken. A sarcoma may develop.

Osteopetrosis

Osteopetrosis (marble disease, congenital osteosclerosis, Albers-Schönberg disease) is a rare hereditary disease in which generalized excessive bone formation is observed, which leads to bone thickening, narrowing, and even complete disappearance of bone-marrow cavities. Osteopetrosis is characterized by a triad: increased bone density, bone fragility, and anemia.

*Etiology and pathogenesis.*Undoubted participation of hereditary factors, which are associated with a violation of the development of bone and hematopoietic tissue. The development of anemia, thrombocytopenia, the appearance of extraosseous hematopoietic centers in the liver, spleen, and lymph nodes is associated with the growing squeezing of bone marrow by the bone.

*Classification.*There are two forms of osteopetrosis:

1. Early (autosomal recessive) - appears at an early age, proceeds malignantly, often ends fatally.
2. Late (autosomal dominant) - a more benign course.

*Pathological anatomy.*The whole skeleton can be affected, but especially tubular bones, bones of the base of the skull, pelvis, spine, ribs. In the early form, the face acquires a characteristic appearance: it is wide, with widely spaced eyes, the root of the nose is depressed, and the lips are thick. With this form, hydrocephalus, increased hair growth, hemorrhagic diathesis, and multiple bone lesions are noted.

Characteristic column-shaped expansion of the lower femurs. On cuts in long bones, the medullary canal is filled with bone tissue and is often not defined. The spongy substance resembles polished marble.

The microscopic picture is peculiar: pathological ossification occurs throughout the entire bone, the bone substance is randomly accumulated in the internal parts of the bones. Osteoclasts are single, signs of bone resorption are insignificant. Bone architecture loses its functional characteristics. At the base of the cartilage, peculiar round islands of bone beams are formed.

*Complication:*bone fractures, especially femoral fractures, purulent osteomyelitis.

*Causes of death.*Patients often die in early childhood from anemia, pneumonia, sepsis.

Paget's disease

Paget's disease (deforming ostosis, deforming osteodystrophy) is a disease characterized by increased pathological remodeling of bone tissue, continuous changes in the processes of bone resorption and new formation, while the bone tissue acquires a peculiar mosaic structure. It is observed more often among men older than 40 years, progresses slowly, becomes noticeable only in old age. The lesion may involve a single bone (mono-osseous form) or several often paired or regional bones (poly-osseous form), but is never generalized.

*Etiology.*The reasons are unknown, the family nature of the disease is emphasized.

*Patho- and morphogenesis.*Bone tissue reconstruction processes are continuous, there is no connection with functional load. There are three phases of the disease:

1. Initial (osteolytic) - the processes of bone resorption with the participation of osteoclasts prevail, deep lacunae are formed in the bone tissue.
2. Active (combination of osteolysis and osteogenesis) - osteoblasts appear, lacunae are filled with newly formed bone substance. The bone beams are built from small fragments forming a characteristic mosaic.
3. Inactive - the process of osteosclerosis prevails.

*Pathological anatomy.*Long tubular bones, especially the femur and tibia, are covered, sometimes spiral-shaped, which is due to the growth of the bone during its reconstruction. A narrow medullary canal is revealed on cuttings. When the periosteum is removed, there are numerous small openings of vascular channels on the surface of the cortical layer. On cutting, the cortical layer loses its compact structure and becomes almost spongy.

When the bones of the skull are damaged, only the bones of the brain skull are involved in the pathological process. The entire bone mass has an uneven spongy structure with pockets of rarefaction and compaction.

In the spine, the process involves one or more vertebrae in different parts of it, but never affects the entire spine. The vertebrae increase in volume or, on the contrary, flatten, depending on the stage of the disease. Focal points of osteoporosis and osteosclerosis are found on bone cuts.

Microscopic examination: determine small fragments of bone structures with uneven contours, with wide, well-defined basophilic adhesion lines. The areas of the bone fragments of the mosaic are usually calcified, their structure is irregular, thin-fibrous or lamellar. A large number of osteoblasts, axillary resorption cavities are found in the deep lacunae of bone structures. Signs of a bone neoplasm are noted: expanded bone cavities are filled with delicate fibrous tissue.

*Complication:*hemodynamic disorders (related to the expansion of blood vessels in the affected bone tissue), pathological fractures (develop in the active phase), osteogenic sarcoma (in 1-10% of patients, is localized in the thigh, lower leg, pelvic bones, in the scapula).

Diseases of the joints

Joint diseases can be associated with dystrophic processes of the structural elements of the joints (arthrosis) or their inflammation (arthritis). Among arthrosis, osteoarthrosis occupies a significant place, and among arthritis - rheumatoid arthritis.

Osteoarthritis

Osteoarthritis- one of the most frequent diseases of the joints of a dystrophic nature. Elderly women suffer more often. Osteoarthritis is divided into primary (idiopathic) and secondary (in other diseases). The pathological process develops in the joints of the lower limbs - pelvic-femoral, tibio-foot.

*Etiology and pathogenesis.*Hereditary (genetically determined disturbance of metabolism in articular cartilage) and acquired (mechanical trauma) factors are important.

Classification. There are three stages of osteoarthritis:

4) Pain in the joints during exercise, narrowing of the joint space and osteophytes (radiologically) are noted.

5) Pain in the joints becomes constant, the narrowing of the joint space and the development of osteophytes are more pronounced.

6) Along with constant pain, functional insufficiency of the joints due to the development of subchondral sclerosis is noted.

*Pathological anatomy.*Macroscopic changes depend on the stage of the disease. In the early stage, the edges of the articular cartilage appear fibrous, fibrous tissue. In the second stage, patterns and humps are found on the articular surface of the cartilage, bone growths - osteophytes - are formed. In the third (late) stage, the articular cartilage disappears, depressions appear on the bones of the joints, and the joints themselves are deformed. The amount of synovial fluid decreases sharply.

Microscopic changes: in the first stage, the cartilage retains its structure, the amount of glycosaminoglycans decreases in its surface and intermediate zones. In the second stage, shallow patterns appear in the surface zone of the cartilage, on the crowns of which chondrocytes accumulate. The pathological process also develops in the subchondral part of the bone. In the third stage, the surface zone and part of the intermediate zone of cartilage die, in the deep zone the number of glycosaminoglycans is sharply reduced, and the number of chondrocytes with pyknotic nuclei is increased.

Diseases of skeletal muscles

Among skeletal muscle diseases, the most widespread are striated muscle diseases of dystrophic (myopathy) and inflammatory (myositis) origin. Progressive muscular dystrophy and myopathy in myasthenia occupy a significant place among myopathies.

Progressive muscular dystrophy

Progressive muscular dystrophy (progressive myopathy) is a variety of primary hereditary chronic diseases of striated muscles. The disease is characterized by growing, often symmetrical, muscle atrophy, accompanied by progressive muscle weakness, almost to complete immobility.

*Etiology and pathogenesis*little studied. The significance of abnormalities in structural proteins, sarcoplasmic reticulum, innervation, and enzymatic activity of muscle cells is discussed.

*Classification.*There are three main forms of progressive muscular dystrophy:

- Duchenne (early form). The recessive type of inheritance associated with the X-chromosome occurs mainly in children aged 3-5 years. First, the muscles of the pelvic girdle, thighs and lower legs are affected, then the shoulder girdle and trunk.
- Erba (youth form). Autosomal dominant type of inheritance, develops during puberty. Changes develop first in the muscles of the chest and shoulder girdle, sometimes in the face (smooth forehead, insufficient closing of the eyes, thick lips).

- Leyden Autosomal recessive type of inheritance, begins in childhood or during puberty. It begins in the muscles of the pelvic girdle and hips, gradually covering the muscles of the trunk and limbs.

*Pathological anatomy.*Muscles are atrophic, thin, depleted of myoglobin, resemble fish meat at autopsy.

Upon microscopic examination, muscle fibers are different in size: along with atrophic ones, there are sharply enlarged (thickened) ones. Pronounced dystrophic changes of muscle fibers, their necrosis and phagocytosis. Adipose tissue accumulates between damaged muscle fibers.

Ultrastructural changes in muscle fibers in Duchenne muscular dystrophy: at the beginning of the disease, expansion of the sarcoplasmic reticulum, foci of destruction of myofibrils, and movement of nuclei to the center of the fiber are found. In the late stage, myofibrils are subject to fragmentation and disorganization, mitochondria swell. In the final stage of the disease, muscle fibers are compacted and surrounded by a hyaline-like substance.

*Death*patients with a severe course of progressive muscular dystrophy caused by a pulmonary infection.

Myasthenia

Myasthenia gravis is a chronic disease, the main symptom of which is weakness and pathological fatigue of the striated muscles. Normal contraction of muscles after their active activity decreases in strength and volume and may stop completely. Muscle rest time becomes longer in the late stage of the disease. Eye muscles (ptosis), masticatory, speech and swallowing muscles are most often affected. The disease occurs at any age, in women 3 times more often than in men.

*Etiology and pathogenesis.*The etiology is unknown. Correlation between thymus abnormalities and myasthenia occupies a significant place in pathogenesis. The development of the disease is associated with a decrease of up to 90% in the number of acetylcholine receptors per unit of muscle plate, which is associated with autosomal reactions.

*Pathological anatomy.*In patients, follicular hyperplasia or thymoma is often found in the thymus. Skeletal muscles are slightly changed or in a state of dystrophy, sometimes accumulation of lymphocytes among muscle cells is revealed. IgG and C3 are also detected in postsynaptic membranes. Lymphoid infiltrates are found in the liver, thyroid gland, and other organs.

*Complication*occur with damage to the respiratory muscles. An inadequate response of the lungs leads to the development of pneumonia and asphyxia, which, as a rule, become the cause of death.

Avitaminosis

Vitamins are part of food products and are important for the normal functioning of the body. Insufficiency or lack of vitamins of both exogenous and endogenous origin lead to the development of a number of pathological processes and diseases (hypo- and vitamin deficiency). As a result of insufficiency or absence of vitamins, the

following develop most often: rickets, scurvy, xerophthalmia, pellagra, deficiency of vitamin B^ and folic acid.

RICKETS

Rickets —a consequence of hypo- or vitamin D deficiency.
There are several forms of rickets:
1) classical form in children of different ages (from 3 months to 1 year — early rickets; from 3 to 6 years — late rickets);
2) vitamin-0-dependent rickets — a hereditary disease with an autosomal recessive type of transmission;
3) vitamin D-resistant rickets — a hereditary sex-linked (X-chromosome) disease;
4) rickets in adults, or osteomalacia.
The classic form deserves the most attentionrickets in childhood and rickets in adults.
Etiology. The cause of rickets is caused by a deficiency of vitamin D. The origin of this deficiency can be: 1) hereditary; 2) as a result of insufficient ultraviolet radiation, necessary for the formation of vitamin D3 in the body; 3) in connection with the insignificant intake of vitamin D with food; 4) impaired absorption of vitamin D in the intestine; 5) increased need for the vitamin with its normal intake into the body; 6) chronic diseases of the kidneys and liver, in which the formation of the active metabolite of vitamin D3 — 1.25 (OH)2O3 — is disturbed. In D-avitaminosis in adults, a violation of vitamin absorption due to diseases of the gastrointestinal tract and an excessive need for vitamin D, for example, during pregnancy, hyperthyroidism, renal acidosis, etc., is of great importance.
Pathogenesis. At the heart of the disease are deep disturbances in the metabolism of calcium and phosphorus, which leads to a violation of the calcification of the osteoid tissue, which loses the ability to accumulate calcium phosphate. This is explained, first of all, by the fact that with rickets, the content of inorganic phosphorus in the blood decreases (hypophosphatemia), the intensity of oxidative processes in tissues decreases with the subsequent development of acidosis. With rickets, protein and fat metabolism is also disturbed, while fatty acids have a rickets-stimulating effect.
Pathological anatomy. In children with early rickets, morphological changes are most pronounced in the bones of the skull, at the junctions of the cartilage and bone parts of the ribs, and in the metaesphyseal sections of long tubular bones, that is, in places with the most intensive growth of the skeleton. Round or oval softenings (craniotabes) appear in the bones of the skull, primarily in the occipital-parietal regions, and periosteal growths (osteophytes) appear in the area of the frontal and parietal humps. At the same time, the child's head acquires a quadrangular shape (sarii; diaotaiit). The size of the fountains increases sharply, they close late. At the joints of the cartilage and bone parts of the ribs, thickenings appear (especially noticeable on the inner surface of the VI, VII and VIII ribs), which have received the name "rickets". Epiphyses of long tubular bones become thickened - "rachitic bracelets".

In the places of enchondral ossification, the germinal zone expands sharply, and it turns into a "rachitic zone", the width of which is proportional to the severity of rickets. In the area of enchondral ossification, an excess of cartilage and osteoid tissue is formed, and calcification does not occur in the latter. Cartilage cells are arranged randomly. Osteoid tissue accumulates not only enchondrally, but also endo- and periosteally, which leads to the development of osteophytes. The cortical layer of the diaphyses thins due to lacunar resorption of the bone; it becomes less elastic and bends easily. Due to the excessive formation of osteoid tissue, which is not capable of calcification, the formation of a full-fledged bone is delayed. Sometimes microfractures of individual bone beams are possible,

In late-onset rickets in children, disorders of endosteal ossification prevail, not enchondral. Bones, especially of the lower limbs and pelvis,subject to deformation, the shape of the chest and spine changes.

In early and late rickets, anemia, enlargement of the spleen and lymph nodes, muscle atony, especially of the abdominal wall and intestines ("frog's belly") are observed.

With rickets in adults (osteomalacia), bone changes are the result of impaired calcification of new bone structures and excessive formation of osteoid tissue.

Complicationin children with rickets, pneumonia, eating disorders anddigestion, as well as purulent infections.

SCURVY

Scurvy(synonyms: scurvy, Barlow's disease) — vitamin deficiency S.

Etiology and pathogenesis. The disease occurs in the absence of vitamin C (ascorbic acid) in food or insufficient absorption. The disease manifests itself most clearly when, along with vitamin C, vitamin P is excluded from food. Insufficient intake of vitamin C in the body disrupts the function of redox enzymes and leads to significant changes in carbohydrate and protein metabolism. The increased formation of melanin and excessive pigmentation of the skin is associated with the disorder of the oxidation of aromatic amino acids (tyrosine and phenylalanine). With an insufficient amount of vitamin C, the condition of the main substance of connective tissue, collagen synthesis, fibrillogenesis, maturation of connective tissue is disturbed, which is associated with an increase in vascular and tissue penetration. It increases especially sharply with a combination of vitamin C and P deficiency. In such cases, the hemorrhagic syndrome is most pronounced. Disturbances and delays in collagen formation also explain the changes in bone tissue in scurvy, which are manifested by inhibition of proliferative processes in the areas of the most intensivebone growth and remodeling.

Pathological anatomy. Morphological changes in scurvy consist of manifestations of hemorrhagic syndrome, bone changes and complications associated with secondary infection.

*Hemorrhagic syndrome*manifests itself equally in both children and adults. Hemorrhages appear on the skin, mucous membranes, internal organs,bone marrow, under the periosteum, in the joint cavity (hemarthrosis). Ulcers appear on the skin and mucous membranes.

Bone changes in children and adults have the same manifestation. In children, they become leaders in the picture of the disease and are expressed in depressionbone formation In the germinal zone of tubular bones, the replacement of cartilaginous structures by bone slows down, the compact layer of diaphyses becomes thin, fractures easily occur. Hemorrhages in the region of the germinal growth zone lead to the separation of the epiphysis from the diaphysis (epiphysiolysis). Bone marrow is replaced by fibrous tissue. In adults, bone changes appear mainly at the border with the cartilaginous part of the ribs, where chondroplastic bone growth continues until 40-45 years of age. Here, the bone beams become thinner, the bone marrow is replaced by fibrous tissue, fibrin and blood accumulate, then the cartilaginous part of the rib can separate from the bone, the sternum in such cases sinks.

The skin with scurvy becomes dark due to the accumulation of melanin in it.

Complicationassociated mainly with the attachment of a secondary infection that develops in areas of hemorrhage. Stomatitis and gingivitis appear, teeth loosen and fall out easily; ulcerative and necrotic processes occur on the tongue and tonsils (ulcerative glossitis, phlegmonous and gangrenous angina). As a result of possible aspiration, pneumonia, abscesses or gangrene of the lungs develop; sometimes joins tuberculosis. Enteritis and colitis are possible.

XEROPHTHALMIA

Xerophthalmia— a disease that is a consequence of vitamin A deficiency.

Etiology and pathogenesis. Avitaminosis A can be of exogenous and endogenous origin and is caused by a number of reasons: its insufficient amount in food, impaired absorption of both vitamin A and fats in the intestines, excessive use of this vitamin in some pathological processes and diseases. It is known that vitamin A determines the condition of the epithelium and the synthesis of rhodopsin. With a deficiency of vitamin A, metaplasia of the prismatic and transitional epithelium takes place into a keratinized, multi-layered flat one. When the synthesis of rhodopsin is disturbed, hemeralopia (chicken blindness) appears. Metaplasia of the prismatic epithelium of the respiratory tract, especially the trachea and bronchi, is often observed in measles and influenza, which is largely associated with endogenous vitamin A deficiency. Manifestations of endogenous vitamin A deficiency can also be observed in other infectious diseases (for example,

Pathological anatomy. Changes in xerophthalmia are characterized by epithelial metaplasia and secondary inflammation of mucous membranes. Epithelial metaplasia into stratified stratum corneum is particularly evident in

the conjunctiva of the eye and the cornea. At the same time, tear glands atrophy and their secretion decreases. There is dryness of the cornea and conjunctiva, which become whitish. The transparency of the cornea decreases sharply, dystrophic and necrotic changes occur in its tissue (keratomalacia). Metaplasia of the epithelium is also observed in the mucous membranes of the respiratory (nasal passages, trachea, bronchi) and urinary tracts, in the vagina, uterus, prostate and pancreas. Inflammatory and ulcerative processes easily occur on mucous membranes changed in this way. Healing of ulcers and wounds in patients withVitamin A is significantly delayed.

PELLAGRA

Pellagra— a chronic disease that occurs when the body lacks nicotinic acid (vitamin PP) and other B vitamins.

Etiology and pathogenesis. Pellagra develops when there is a deficiency in the bodyonly nicotinic acid and other vitamins, but also tryptophan. A significant loss of nicotinic acid by the body is observed when there is insufficient protein in food products. Deficiency of nicotinic acid becomes the cause of disruption of redox processes, which is accompanied by the development of both dystrophic and atrophic changes.

Pathological anatomy. Morphological changes develop mainly in the skin, nervous system and intestines. Erythema with swelling appears on the skin of exposed parts of the body, which are gradually replaced by hyperkeratosis and atrophy, the skin becomes dry and acquires a brown color. During histological examination, in addition to atrophy and hyperkeratosis, cellular infiltrates around the vessels of the dermis, dystrophic changes in sweat glands and nerve fibers are found. Excessive production of melanin is found in the basal layer of the skin. Dystrophic changes develop in the nervous system, primarily in various areas of the brain (motor cortex, midbrain, cerebellum), spinal cord, and peripheral nerves. With a long course, dystrophic changes develop mainly in the conduction system of the spinal cord. Atrophy of the mucous membrane, cystic expansion of the glands are found in both the small and large intestines, ulceration of follicles with successive epithelization of ulcers. Atrophicchanges also develop in the stomach, liver and pancreas.

VITAMIN B12 AND FOLIC ACID DEFICIENCY

With a deficiency of vitamin B12 and folic acid, various forms of anemia develop.

PNEUMOCONIOSIS

Pneumoconiosis - dust diseases of the lungs. The term "pneumoconiosis" was proposed by Zenker in 1867. Industrial dust refers to the smallest particles of a solid substance that are formed during an industrial process, which, falling into the air, are suspended in it for a more or less long time.

A distinction is made between inorganic and organic dust. Inorganic dust includes quartz (which consists of 97-99% free silicon dioxide), silicate, and metal dust. Organic includes plant (flour, wood, cotton, tobacco, etc.) and animal (cotton, hair, etc.) products. There is

mixed dust, for example, containing coal, quartz and silicate dust in various proportions, or iron ore dust consisting of iron and quartz dust. Particles less than 5 μm in size that penetrate deep into the lung parenchyma pose the greatest danger. The shape and consistency of dust particles and their solubility in tissue fluids are of great importance. Dust particles with sharp edges injure the mucous membrane of the respiratory tract. Fibrous dusts of animal and plant origin cause chronic rhinitis, laryngitis, tracheitis, bronchitis, pneumonia. During the dissolution of dust particles, chemical compounds are formed that have an irritating, toxic, and histopathogenic effect. They have the ability to cause the development of connective tissue in the lungs, that is, pneumosclerosis.
When dust of various composition enters the lungs, lung tissue can react differently.
The reaction of lung tissue can be:

- inert, for example, with ordinary pneumoconiosis - anthracosis of coal miners;
- fibrosing, for example, with massive progressive fibrosis, asbestos with silicosis;
- allergic, with exogenous allergic pneumonitis;
- non-plastic, for example, mesothelioma and lung cancer with asbestosis.

Localization of the process in the lungs depends on the physical properties of the dust. Particles less than 2-3 μm in diameter can reach the alveoli, larger particles are retained in the lungs and nasopharynx, from where they can be removed from the lungs by mucociliary transport. An exception to this rule is asbestos, whose particles of 100 microns can settle in the terminal parts of the respiratory tract. This is because the asbestos particles are very thin (about 0.5 microns in diameter). Dust particles are phagocytosed by alveolar macrophages, which then migrate to the lymphatic vessels and go to the basal lymph nodes.

Classification. Anthracosis, silicosis, metalloconiosis, carboconiosis, pneumoconiosis from mixed dust, pneumoconiosis from organic dust are distinguished among pneumoconiosis.

ANTHRACOS

Inhalation of coal dust is accompanied by its local accumulations, which are invisible until massive pulmonary fibrosis is formed. The accumulation of coal in the lungs, which is referred to as "pulmonary anthracosis", is typical for residents of industrial cities. It can be observed practically in

- the amount of inhalable silicon and quartz, as well as the type of coal (bituminous coal is more dangerous than wood coal);
- co-infection with a tubercle bacillus or atypical mycobacteria;
- the development of a hypersensitivity reaction due to the death of macrophages and the release of antigens;

- the development of fibrosis associated with the deposition of immune complexes. But none of the theories have been proven, and some researchers believe that the determining factor is only the amount of absorbed dust.At the end of the disease, the lungs have the appearance of honeycombs, the formation of the pulmonary heart is observed. Patients die either from pulmonary and heart failure, or from joining intercurrent diseases.

Silicosis

Silicosis is a disease that develops as a result of long-term inhalation of dust containing free silicon dioxide. Most of the earth's crust contains silica and its oxides. Silicon dioxide is present in nature in three different crystalline forms: quartz, cristobalite and tridymite. Uncombined forms of silicon dioxide are called "free silicon", and combined forms containing cations make up various silicates. Silica dust is found in most industrial productions, in particular in gold, tin and copper mines, during cutting and grinding of stones, during the production of glass , in metal smelting, in the production of pottery and porcelain. In all these industries, the size of the particles matters. Sand usually contains 60% silicon oxide. But its particles are so large that they can reach the periphery of the lungs. Only small particles, entering the bronchioles and alveoli, can cause their damage. Silicon, especially its particles with a size of 2-3 nm, is a powerful stimulator of the development of fibrosis. The amount and duration of exposure to silicon also play a large role in the development of silicosis. Approximately 10-15 years of work in industrial dust conditions without respirators can cause silicosis. If the concentration of dust is significant, then its acute form may occur in 1-2 years, "acute" silicosis. In some cases, the disease may appear several years after the end of exposure to industrial dust (late silicosis). The risk group for the given disease includes the workers mentioned above professions The amount and duration of exposure to silicon also play a large role in the development of silicosis. Approximately 10-15 years of work in industrial dust conditions without respirators can cause silicosis. If the concentration of dust is significant, then its acute form may occur in 1-2 years, "acute" silicosis. In some cases, the disease may appear several years after the end of exposure to industrial dust (late silicosis). The risk group for the given disease includes the workers mentioned above professions The amount and duration of exposure to silicon also play a large role in the development of silicosis. Approximately 10-15 years of work in industrial dust conditions without respirators can cause silicosis. If the concentration of dust is significant, then its acute form may occur in 1-2 years, "acute" silicosis. In some cases, the disease may appear several years after the end of exposure to industrial dust (late silicosis). The risk group for the given disease includes the workers mentioned above professions

Pathogenesis. Now the development of silicosis is associated with chemical, physical and immune processes that occur when dust particles interact with tissue. At the same time, the value of the mechanical factor is not excluded.

According to modern ideas, the pathogenesis of silicosis includes the following stages:

- inhalation of silicon particles with a diameter of less than 2 μm with their penetration into the terminal parts of the airways;

- absorption (phagocytosis) of these silicon particles by alveolar macrophages;
- death of macrophages;
- release of the contents of dead cells, including silicon particles;
- repeated phagocytosis of silicon particles by other macrophages and their death;
- appearance of fibrous hyalinized connective tissue;
- possible development of further complications.

The exact nature of the factor or factors causing fibrosis is still unknown. Unlike coal dust, silicates are toxic to macrophages and lead to their death with the release of proteolytic enzymes and unchanged silicate particles. Enzymes cause local tissue damage with subsequent fibrosis; silicate particles are again absorbed by macrophages and the cycle repeats endlessly. According to this theory, the leading role in the pathogenesis of silicotic fibrosis is the death of koniophages with the subsequent stimulation of fibroblasts by macrophage decay products. It is believed that hydrogen bonds between the released silicic acid formed when it is absorbed by macrophage lysosomes and the phospholipids of the phagosome membrane lead to membrane rupture. Rupture of the phagosome membrane leads to the death of macrophages. All macrophage derivatives formed able to stimulate fibroblastic proliferation and activation of fibrillogenesis. Since plasma cells and immunoglobulins are found in the places of impression, participation in fibrillogenesis and immune reactions is assumed, but the mechanism of their development in silicosis has not yet been clarified. According to the immunological theory, during the influence of silicon dioxide on tissues and cells, during their disintegration, auto antigens appear, which leads to auto immunization. The immune complex arising from the interaction of an antigen with an antibody exerts a pathogenic influence on the connective tissue of the lungs, resulting in the formation of a nodule. But no specific antibodies were detected. but the mechanism of their development in silicosis has not yet been clarified. According to the immunological theory, during the influence of silicon dioxide on tissues and cells, during their disintegration, auto antigens appear, which leads to auto immunization. The immune complex arising from the interaction of an antigen with an antibody exerts a pathogenic influence on the connective tissue of the lungs, resulting in the formation of a nodule. But no specific antibodies were detected. but the mechanism of their development in silicosis has not yet been clarified. According to the immunological theory, during the influence of silicon dioxide on tissues and cells, during their disintegration, auto antigens appear, which leads to auto immunization. The immune complex arising from the interaction of an antigen with an antibody exerts a pathogenic influence on the connective tissue of the lungs, resulting in the formation of a nodule. But no specific antibodies were detected.

Pathological anatomy. With chronic silicosis, atrophy and sclerosis are found in the mucous membrane and submucosal layer of the nasal cavity, larynx, and trachea. In humans, the histological evolution of silicosis lesions is not well known, since an advanced form of the disease is detected at autopsy. According to the study of silicosis in

animals and in the case of an acute disease, the following has been established. The first response to the appearance of silicon in the acinus is the accumulation of macrophages. If the pollination is massive, then macrophages fill the lumen of the bronchioles and the surrounding alveoli. It is possible to develop a serous inflammatory reaction, similar to what can be observed in alveolar proteinosis. In some cases, the described picture is similar to gray hepatization of the lungs in case of croup pneumonia. With the slow development of the process in the early stages, multiple small nodules are found in the lung tissue, mainly in the upper parts and in the portal area, which give the lung parenchyma a fine-grained appearance, as if the tissue is covered with sand. During this period, granulomas are formed, which are mainly represented by macrophages surrounded by lymphocytes and plasmocides. These granulomas are found around bronchioles and arterioles, as well as in paraseptal and subpleural tissues. In the process of evolution, the size of the nodules increases, some of them merge and then they are visible to the naked eye. The nodules become larger and denser, and then large areas of the lungs turn into scar layers, separated from each other by areas of mixed emphysema. Pleural leaves grow together with dense scar moorings.

In the lungs, silicosis manifests itself in the form of two main forms: nodular and diffuse-sclerotic (or interstitial).

With the nodular form, a significant number of zygotic nodes are found in the lungs, which are small paired or larger sclerotic areas of round, oval or irregular shape, gray or gray-black in color (coal miners). With severe silicosis, the nodes merge into large silicotic nodes, occupying a large part of the fate or even the entire fate. In such cases, they speak of a tumorous form of pulmonary silicosis. The nodular form occurs with a high content of free silicon dioxide dust and with prolonged exposure to dust.

With the diffuse-sclerotic form, typical silicotic nodes in the lungs are absent or there are very few of them, they are often found in their bifurcation lymph nodes. This form is observed when inhaling industrial dust with a small content of free silicon dioxide. In this form, the connective tissue in the lungs grows in the alveolar membranes, peribronchially and perivascularly. Diffuse emphysema, bronchial deformation, various forms of bronchioles and bronchitis (more often catarrhal-desquamative, less often purulent) develop. Sometimes a mixed form of pulmonary silicosis is found. Silicotic nodes can be typical and atypical. The structure of typical silicone nodes is twofold: some are formed from concentrically arranged hyalinized bundles of connective tissue and therefore have a rounded shape, others are not rounded and consist of bundles of connective tissue, that go like a vortex in different directions. Atypical silicotic nodes have irregular shapes, they lack concentric and vortex-like arrangement of bundles of connective tissue. In all nodes, there are many dust particles lying freely or in macrophages, which are called dust marks or coniophages. Silicotic nodes develop in the lumen of the alveolar passages, as well as in the place of lymphatic vessels. Alveolar histiocytes phagocytize dust particles and turn into coniophages. During long-term and strong pollination, all dust cells are removed, so their accumulations are formed in the lumens of the alveoli and alveolar passages. Collagen fibers appear between the cells, a cell-fibrous nodule is formed. Gradually, dust cells die, the number of fibers increases, resulting in the formation of a typical fibrous

node. Similarly, a silicotic nodule is formed at the site of a lymphatic vessel. In silicosis, in the center of large silicotic nodules, connective tissue disintegrates with the formation of silicotic caverns. Disintegration occurs as a result of changes in blood vessels and the nervous system of the lungs, as well as as a result of the instability of the connective tissue of silicotic nodules and nodes that differ in biochemical composition from normal connective tissue. Silicone connective tissue is less resistant to the action of collagenase compared to normal. A lot of quartz dust, diffuse sclerosis and silicotic nodules are found in lymph nodes (bifurcations, basal, less often in paratracheal, cervical, supraclavicular). Occasionally, silicotic nodules are found in the spleen, liver, and bone marrow. In silicosis, in the center of large silicotic nodules, connective tissue disintegrates with the formation of silicotic caverns. Disintegration occurs as a result of changes in blood vessels and the nervous system of the lungs, as well as as a result of the instability of the connective tissue of silicotic nodules and nodes that differ in biochemical composition from normal connective tissue. Silicone connective tissue is less resistant to the action of collagenase compared to normal. A lot of quartz dust, diffuse sclerosis and silicotic nodules are found in lymph nodes (bifurcations, basal, less often in paratracheal, cervical, supraclavicular). Occasionally, silicotic nodules are found in the spleen, liver, and bone marrow. In silicosis, in the center of large silicotic nodules, connective tissue disintegrates with the formation of silicotic caverns. Disintegration occurs as a result of changes in blood vessels and the nervous system of the lungs, as well as as a result of the instability of the connective tissue of silicotic nodules and nodes that differ in biochemical composition from normal connective tissue. Silicone connective tissue is less resistant to the action of collagenase compared to normal. A lot of quartz dust, diffuse sclerosis and silicotic nodules are found in lymph nodes (bifurcations, basal, less often in paratracheal, cervical, supraclavicular). Occasionally, silicotic nodules are found in the spleen, liver, and bone marrow. Disintegration occurs as a result of changes in blood vessels and the nervous system of the lungs, as well as as a result of the instability of the connective tissue of silicotic nodules and nodes that differ in biochemical composition from normal connective tissue. Silicone connective tissue is less resistant to the action of collagenase compared to normal. A lot of quartz dust, diffuse sclerosis and silicotic nodules are found in lymph nodes (bifurcations, basal, less often in paratracheal, cervical, supraclavicular). Occasionally, silicotic nodules are found in the spleen, liver, and bone marrow. Disintegration occurs as a result of changes in blood vessels and the nervous system of the lungs, as well as as a result of the instability of the connective tissue of silicotic nodules and nodes that differ in biochemical composition from normal connective tissue. Silicone connective tissue is less resistant to the action of collagenase compared to normal. A lot of quartz dust, diffuse sclerosis and silicotic nodules are found in lymph nodes (bifurcations, basal, less often in paratracheal, cervical, supraclavicular). Occasionally, silicotic nodules are found in the spleen, liver, and bone marrow. Silicone connective tissue is less resistant to the action of collagenase compared to normal. A lot of quartz dust, diffuse sclerosis and silicotic nodules are found in lymph nodes (bifurcations, basal, less often in paratracheal, cervical, supraclavicular). Occasionally, silicotic nodules are found in the spleen, liver, and bone marrow. Silicone connective

tissue is less resistant to the action of collagenase compared to normal. A lot of quartz dust, diffuse sclerosis and silicotic nodules are found in lymph nodes (bifurcations, basal, less often in paratracheal, cervical, supraclavicular). Occasionally, silicotic nodules are found in the spleen, liver, and bone marrow.

ASBESTOS

The word "asbestos" comes from the Greek word "indestructible". About 6 million tons of this mineral are extracted annually in the world. Asbestos contains many fibrous minerals consisting of hydrated silicates. Asbestos fibers give a double refraction of the beam in polarized light, which can be used in microscopic diagnosis. They are often found in combination with silicates. In these cases, they contain calcium, iron, magnesium and sodium. According to most authors, the carcinogenicity of asbestos depends not only on its type, but on the length of the fibers. Thus, fibers larger than 5 microns do not have carcinogenic properties, while fibers smaller than 3 microns have a pronounced carcinogenic effect. The risk of lung cancer in patients with asbestosis increases approximately 10 times, and in the case of smokers, then 90 times. Patients with asbestosis are twice as likely to have cancer of the esophagus, stomach, and colon. It is now proven that asbestos potentiates the action of other carcinogens. The onset of pneumoconiosis is different. It happens that pulmonary manifestations appear after 1-2 years of contact with asbestos, but most often - after 10-20 years. The pathogenesis of pulmonary fibrosis is unknown. Asbestos fibers have a small thickness (0.25-0.5 μm, so they penetrate deeply into the alveoli in the basal parts of the lungs. The fibers are found not only in the lungs, but also in the peritoneum and other organs. The fibers damage the walls of the bronchioles and alveoli, which is accompanied by shallow hemorrhages, which are the basis for the formation of hemosideria inside macrophages. Sets consisting of asbestos fibers are sometimes covered with protein, but most often with glycosaminoglycans, on which iron-containing grains of hemosiderin are deposited, were named "asbestos bodies". Under an optical microscope, they are reddish or elongated yellowish structures in the form of rings or strung pearls, resembling the appearance of "elegant dumbbells". In the electron microscope, their appearance is even more specific; their outer contours are represented by roughness resembling the steps of a ladder, and their axis contains parallel lines. These bodies (length 10-100 and width 5-10 μm) are found in sputum and help to differentiate asbestos from fibrosing alveolitis. Histologically, interstitial fibrosis is observed in the lungs. Macroscopically, the lungs in the late stages look like honeycombs. Fibrosis and emphysema of the lungs are found mainly in the basal parts of the lungs. Patients die from pulmonary and pulmonary heart failure. which resemble the steps of a ladder and their axis contains parallel lines. These bodies (length 10-100 and width 5-10 μm) are found in sputum and help to differentiate asbestos from fibrosing alveolitis. Histologically, interstitial fibrosis is observed in the lungs. Macroscopically, the lungs in the late stages look like honeycombs. Fibrosis and emphysema of the lungs are found mainly in the basal parts of the lungs. Patients die from pulmonary and pulmonary heart failure. which resemble the steps of a ladder and their axis contains parallel lines. These bodies (length 10-100 and width 5-10 μm) are

found in sputum and help to differentiate asbestos from fibrosing alveolitis. Histologically, interstitial fibrosis is observed in the lungs. Macroscopically, the lungs in the late stages look like honeycombs. Fibrosis and emphysema of the lungs are found mainly in the basal parts of the lungs. Patients die from pulmonary and pulmonary heart failure.

Vibration diseaseoccurs in workers who use vibrating equipment in the course of their work: pneumatic hammers, machines for grinding and polishing metal and wooden products, for compacting concrete, asphalt road surfaces, driving\piles, and others. The disease is chronic. Workers develop a clinical and morphological picture of obliterating endarteritis. Vascular changes are accompanied by a violation of tissue trophism of the upper and lower extremities. Contractures of the fingers, deforming arthrosis, develop at the final stage of gangrene of the fingers, hands, and feet. Dystrophic changes up to the complete death of neurons are noted in the spinal cord. In the heads of the bones of the wrist, in the epiphyses of the radial and ulnar cysts, cystic foci of rarefaction and sclerosis are observed.

Diseases caused by the influence of electromagnetic waves of radio frequencies. Electromagnetic waves of radio frequencies are widely used in the field of radio (radiolocation, radio navigation, radio astronomy, radio linear communications, radio telephony, etc.), television, during physiotherapeutic procedures. They distinguish:— microwaves (MKH), or ultrahigh-frequency (UHF) with a wavelength from 1 mm to 1 m;—ultrashort waves (VHF), or ultrahigh-frequency waves (UHF) with a wavelength of distance up to 10 m;

— short waves (HF) or high frequency waves (HF) with a wavelength from 10 to 1000m and more.

There have been no reports of acute deaths among lairds exposed to massive exposure to radio frequency electromagnetic waves.

Chronic exposurelow intensities of electromagnetic waves of radio frequencies of various ranges occurs in industry, workers of radio-television and radio relay stations, and residents of adjacent areas. The victims have damage to the function of the nervous, cardiovascular systems and gonads.

Morphological changes are detected in synapses and sensory nerve fibers of the receptor zones of the skin of internal organs. In the brain, the neurosecretory function of the neurons of the hypothalamic region is disturbed, which is accompanied by a steady drop in blood pressure. Fatty dystrophy of cardiomyocytes occurs in the myocardium. Dystrophic changes in the germinal epithelium up to its necrosis occur in testes. The most pronounced clinical and morphological changes are noted as a result of exposure to microwaves (MKH).

Diseases caused by exposure to industrial noise (noise disease)

Noise disease refers to persistent, irreversible morphological changes in the hearing organ caused by exposure to industrial noise

With an acute overpowering effect of noise and sounds, the death of the spiral (Corti's) organ, rupture of the eardrums, and bleeding from the ears are observed.

With chronic exposure to industrial noise, atrophy of the spiral organ with its replacement by fibrous connective tissue is observed. Changes in the sensitive nerve may be absent. Stiffness is observed in the joints of sensitive bones.

Meteosensitivity and diseases caused by atmospheric pressure.

Weather sensitivityis the organism's reaction to the influence of meteorological (weather) factors. Meteosensitivity is quite widespread and occurs under any, but more often unusual for a given person climatic conditions. About a third of the inhabitants of temperate latitudes "feel" the weather. The peculiarity of these reactions is that they occur in a significant number of people simultaneously with the change in meteorological conditions or slightly ahead of them. Meteosensitivity has long caused surprise and even fear of man before an incomprehensible phenomenon of nature. People who sense the weather were called "living barometers", "storm forecasters", "weather prophets".

Already in ancient times, doctors guessed about the influence of weather on the body. In Tibetan medicine, it is indicated that "pain in the joints increases in the rainy season and in the period of high winds." Paracelsus wrote: "He who has studied the winds, lightning and weather knows the origin of diseases."

The manifestation of weather sensitivity depends on the initial state of the organism, age, the presence of any disease and its nature, the microclimate in which a person lives, and the degree of his acclimatization to it. Meteosensitivity is more often noted in people who are rarely in the fresh air, engaged in sedentary, mental work, who do not engage in physical education. It is in them that the zones of the so-called microclimatic comfort are narrowed. As a rule, meteorological fluctuations are not dangerous for a healthy person. With a sudden change in weather conditions, it becomes harder for them to concentrate. Hence, the number of accidents may increase. As a result of diseases (influenza, sore throat, inflammation of the joints, joint diseases, etc.) or overfatigue, the body's resistance and reserves decrease. That is why weather sensitivity is noted in 35-70% of patients with various diseases. So, every second patient with diseases of the cardiovascular system feels the weather. Significant atmospheric changes can cause overstrain and disruption of adaptation mechanisms. Then the oscillatory processes in the body — biological rhythms — are distorted, become chaotic. Physiological (asymptomatic) weather reaction can be compared to a calm lake, on which waves are flowing from a light breeze. A pathological (morbid) weather reaction represents a kind of vegetative "storm" in the body. Disturbances in the regulation of the autonomic nervous system contribute to its development. The number of vegetative disorders has recently been increasing, which is connected with the effect of adverse factors of modern civilization, stress, haste, hypodynamia, overeating and malnutrition, etc. In addition, the functional state of the nervous system is far from the same in different people. This determines the fact that that diametrically opposite weather reactions, favorable and unfavorable, are often noted for the same diseases. More often, weather sensitivity is observed in people with a weak (melancholic) and strong unbalanced (choleric) type of nervous system. People of a strong balanced type (sanguines) are sensitive to the weather only when the body is weakened.

The body is affected by both the weather as a whole and its individual components. Fluctuations in barometric pressure act in two ways: they reduce blood oxygen saturation (the effect of barometric "pits") and mechanically irritate the nerve endings (receptors) of the pleura (the mucous membrane that lines the pleural cavity), the peritoneum (which lines the abdominal cavity), the synovial membrane of the joints, and also vascular receptors. On the European territory of the country, atmospheric pressure is most variable in the Baltic region, in the northwest and north. It is here that weather sensitivity is most often noted in patients with cardiovascular diseases. The wind causes disturbances in the nervous system, irritating skin receptors. In recent years, the study of the influence of weather conditions onorganism, the so-called "syndromic meteopathology", which includes the symptoms of meteopathy caused by the combined effect of barometric pressure and atmospheric anomalies, such as thunderstorms, hot and dry winds, fogs, snowfall, etc. So, for example, the syndrome of the midday wind in France; the syndrome of the southwest wind in Switzerland, the syndrome of the northern winds (nords) blowing on the Absheron peninsula (Baku), according to various scientists, affect the health of approximately 75% of the population of these areas. They provoke angina attacks with coronary heart disease

Air humidity plays a role in maintaining the density of oxygen in the atmosphere, affects heat exchange and sweating. Patients with hypertension and atherosclerosis are especially sensitive to high humidity. In most cases, exacerbation of diseases of the cardiovascular system occurs at high relative humidity (80-95%). For many people, rainy days leave an impression even on their appearance, often the face becomes pale. Sudden temperature changes cause outbreaks of acute respiratory infectious diseases. In January 1780, the air temperature in St. Petersburg rose from -44° to +6° during one night, as a result, about 40,000 residents fell ill. A significant increase in cases of acute respiratory diseases was noted in Tashkent in November 1954, when the air temperature dropped from 4-15° to -21°. In addition, a sharp north wind blew, which raised masses of drops of water, sand and microbes that were in them into the air, outbreaks of infectious diseases arose in the city. An excess of positive aerophones, which is observed in hot and humid weather, has an adverse effect on the body, which can cause an exacerbation of heart diseases. In recent years, great importance has been attached to changes in solar activity and the Earth's magnetic field (geomagnetic disturbances and storms). their effect on the body is revealed 1-2 days before the weather changes, while other meteorological factors affect directly before or during the passage of air masses (cyclone or anticyclone). Unusual persistent weather, as a rule, also has an adverse effect on the body. In November 1977, warm, humid weather with heavy fog persisted for a long time in the city of Saratov. It had an oppressive effect on that raised masses of water droplets, sand and microbes contained in them into the air, outbreaks of infectious diseases arose in the city. An excess of positive aerophones, which is observed in hot and humid weather, has an adverse effect on the body, which can cause an exacerbation of heart diseases. In recent years,

great importance has been attached to changes in solar activity and the Earth's magnetic field (geomagnetic disturbances and storms). their effect on the body is revealed 1-2 days before the weather changes, while other meteorological factors affect directly before or during the passage of air masses (cyclone or anticyclone). Unusual persistent weather, as a rule, also has an adverse effect on the body. In November 1977, warm, humid weather with heavy fog persisted for a long time in the city of Saratov. It had an oppressive effect on that raised masses of water droplets, sand and microbes contained in them into the air, outbreaks of infectious diseases arose in the city. An excess of positive aerophones, which is observed in hot and humid weather, has an adverse effect on the body, which can cause an exacerbation of heart diseases. In recent years, great importance has been attached to changes in solar activity and the Earth's magnetic field (geomagnetic disturbances and storms). their effect on the body is revealed 1-2 days before the weather changes, while other meteorological factors affect directly before or during the passage of air masses (cyclone or anticyclone). Unusual persistent weather, as a rule, also has an adverse effect on the body. In November 1977, warm, humid weather with heavy fog persisted for a long time in the city of Saratov. It had an oppressive effect on there were outbreaks of infectious diseases in the city. An excess of positive aerophones, which is observed in hot and humid weather, has an adverse effect on the body, which can cause an exacerbation of heart diseases. In recent years, great importance has been attached to changes in solar activity and the Earth's magnetic field (geomagnetic disturbances and storms). their effect on the body is revealed 1-2 days before the weather changes, while other meteorological factors affect directly before or during the passage of air masses (cyclone or anticyclone). Unusual persistent weather, as a rule, also has an adverse effect on the body. In November 1977, warm, humid weather with heavy fog persisted for a long time in the city of Saratov. It had an oppressive effect on there were outbreaks of infectious diseases in the city. An excess of positive aerophones, which is observed in hot and humid weather, has an adverse effect on the body, which can cause an exacerbation of heart diseases. In recent years, great importance has been attached to changes in solar activity and the Earth's magnetic field (geomagnetic disturbances and storms). their effect on the body is revealed 1-2 days before the weather changes, while other meteorological factors affect directly before or during the passage of air masses (cyclone or anticyclone). Unusual persistent weather, as a rule, also has an adverse effect on the body. In November 1977, warm, humid weather with heavy fog persisted for a long time in the city of Saratov. It had an oppressive effect on which can cause exacerbation of heart diseases. In recent years, great importance has been attached to changes in solar activity and the Earth's magnetic field (geomagnetic disturbances and storms). their effect on the body is revealed 1-2 days before the weather changes, while other meteorological factors affect directly before or during the passage of air masses (cyclone or anticyclone). Unusual persistent

weather, as a rule, also has an adverse effect on the body. In November 1977, warm, humid weather with heavy fog persisted for a long time in the city of Saratov. It had an oppressive effect on which can cause exacerbation of heart diseases. In recent years, great importance has been attached to changes in solar activity and the Earth's magnetic field (geomagnetic disturbances and storms). their effect on the body is revealed 1-2 days before the weather changes, while other meteorological factors affect directly before or during the passage of air masses (cyclone or anticyclone). Unusual persistent weather, as a rule, also has an adverse effect on the body. In November 1977, warm, humid weather with heavy fog persisted for a long time in the city of Saratov. It had an oppressive effect on while other meteorological factors affect directly before or during the passage of air masses (cyclone or anticyclone). Unusual persistent weather, as a rule, also has an adverse effect on the body. In November 1977, warm, humid weather with heavy fog persisted for a long time in the city of Saratov. It had an oppressive effect on while other meteorological factors affect directly before or during the passage of air masses (cyclone or anticyclone). Unusual persistent weather, as a rule, also has an adverse effect on the body. In November 1977, warm, humid weather with heavy fog persisted for a long time in the city of Saratov. It had an oppressive effect onpsyche of people, reduced work capacity, caused overstrain of the nervous system.

There are three degrees of weather sensitivity. A mild degree is only a subjective malaise. With severe weather sensitivity (moderate degree), pronounced objective shifts are noted: changes in blood pressure, electrocardiogram, etc. With a severe degree of meteosensitivity, sharply expressed violations are observed, it is manifested by five types of meteopathic reactions. With the cardiac type, there are pains in the region of the heart, shortness of breath. The cerebral type is characterized by headaches, dizziness, noise and ringing in the head. The mixed type is a combination of cardiac and nervous disorders. In the asthenoneurotic type, increased excitability, irritability, insomnia, changes in blood pressure are noted. There are people who cannot clearly localize the manifestation of weather sensitivity. This is an unspecified reaction type: generalweakness, pain and aches in joints, muscles, etc.

The nature and magnitude of damage caused by atmospheric pressure depends on the magnitude (amplitude) of atmospheric pressure deviations and, mainly, from the speed of its change.

Decompression sickness most often occurs in divers (during deep-sea diving), in pilots, workers in caissons (caisson disease) as a result of saturation of blood and body tissues with nitrogen or helium and other gases during a person's stay in a high-pressure zone with its subsequent decrease - decompression . The saturation of the tissues of the body with nitrogen or helium in the zone of increased pressure continues until the pressure of these gases in the inhaled air equalizes with theirpressure in tissues. This process usually lasts several hours, and different tissues are saturated with nitrogen or helium at different rates. Blood, for example, is saturated

faster than adipose tissue, but the latter dissolves nitrogen 5 times more than blood and other tissues. Saturation of tissues with nitrogen at a pressure of up to 4 at. h (when observing the rules for creating increased pressure) does not have an adverse effect on the body. However, during a rapid transition from a high-pressure zone to a low-pressure zone, excess dissolved nitrogen does not have time to be excreted through the lungs, which results in the transition of blood and tissue gases from a dissolved state to a gaseous state with the formation of bubbles.

The immediate cause of decompression sickness is the blockage of blood vessels by gas bubbles or their suppression of adjacent tissues. Concomitant factors are of significant importance: hard physical work of cooling the body, injuries, etc.

Symptoms of the disease most often appear within the first hour after leaving the high pressure zone, but often much later. The disease is manifested by skin itching, joint and muscle pain. The most severe clinical symptoms occur when the blood vessels of the brain, lungs and other vital organs are blocked by gas bubbles?

When brain vessels are damaged, dizziness, stupor, vomiting, weakness, fainting, sometimes paresis and paralysis are observed. When pulmonary vessels are damaged, chest pains and a sharp cough occur. Depending on the severity of the disease, death can occur either a few minutes after decompression or withinone day to three weeks.

With the rapid onset of death, there is a strong emphysema of the subcutaneous tissue of the trunk, neck, and face. Crepitation is audible when palpating the skin (resembles the crunch of snow underfoot). Due to the presence of gas in the blood vessels and uneven blood filling of the vessels of the hemomicrocirculatory channel, the skin acquires a marble appearance. The blood collected in the veins remains liquid (due to hypoxia) and acquires a foamy appearance. During the microscopic examination of the internal organs in the vessels, an abundance of air bubbles (gas embolism) is noted. Swelling, perivascular hemorrhages, interstitial emphysema are found in the lungs, fatty dystrophy in the liver. There are multiple small ischemic foci of gray softening in the brain and spinal cord.

With long-term exposure to elevated atmospheric pressure, foci of rarefaction with perifocal sclerosis are found in the tubular bones, deforming osteoarthritis in the joints.

The most effective method of treatment is decompression, i.e. increasing the pressureits subsequent slow decrease."

Radiation damage

Radiation is energy contained in electromagnetic waves and particles. The types, frequencies and biological effect of electromagnetic radiation are summarized in the table. About 80% of radiation comes from natural sources, including cosmic rays, ultraviolet light, and natural radionuclides, especially radon gas. The other 20% arise from various man-made sources: sources of radio and microwave radiation, nuclear, power plants, etc. Though. that the pathological effect of high doses of radiation is probably proven, the effect of low dosessometimes it turns out to be the exact opposite. Electromagnetic radiation is divided into ionizing and non-ionizing.

Non-ionizing radiation includes radiation with a long wavelength and a low frequency of radio waves, microwave, ultraviolet and infrared radiation, visible light. This radiation will lead to vibration and rotation of the atoms of biological molecules. Short-wave radiation can ionize andknock out electrons.

Ionizing and non-ionizing electromagnetic radiation

X-ray, gamma and cosmic radiation are classified as ionizing radiation. There is also radiation of elementary particles: alpha or beta electrons, neutrons, mesons and neutrinos. The energy of these particles is measured in megaelectronvolts (MZV).

The dose of ionizing radiation is measured in the following units: — X-ray: the dose of ionizing radiation, when acting on 1 cm of air

ions carrying a charge of one electrostatic unit will be formed; — rad: dose of radiation under the influence of which 1 gram of tissue absorbs 100 Erg; —gray (Gy): radiation dose under the influence of which 1 kg of tissue absorbs 1 J of energy; — ber: radiation dose that produces a biological effect is equal to the action of 1 rad

X-ray or gamma radiation. — sievert (Zv): the dose of radiation that produces a biological effect is equal to

effects of 1 Gy of X-ray or gamma radiation; 1 Sv is equal to 100 Ber.

Cellular mechanisms of radiation damage

The acute effect of the lesion can vary from pronounced necrosis at high doses (>10 Gy), death of proliferating cells at medium doses (from 1 to 2 Gy) to the absence of histopathological effect at doses of less than 0.5 Gy. At such low doses, intracellular structures, especially DNA, are damaged; however, adaptive and reparative response mechanisms to low doses of radiation are activated in most cells. Delayed (late) effects of ionizing radiation can be observed in surviving cells: mutations, chromosomal aberrations, genetic instability. These genetically damaged cells can become the basis for the emergence of malignant tumors; fast-growing tissues are most severely affected. Most tumors are induced by ionizing radiation with a power of more than 0.5 Gy. Acute cell death, especially endothelial, can lead to delayed organ dysfunction several months or even years after exposure to radiation. In general, this delayed damage occurs as a result of several pathological processes: atrophy of parenchymal organs, ischemia as a result of vascular damage and fibrosis. Acute and delayed effects of ionizing radiation are presented in the table and described below.

Acute effects. Ionizing radiation can cause various types of DNA damage: the formation of cross-links in DNA proteins, cross-links between DNA chains, oxidation and destruction of bases, destruction of carbohydrate-phosphatechains, breaking one and two DNA chains. These damages can occur both as a result of the direct action of elementary or short-wave radiation particles, and as a result of the action of free radicals and soluble substances formed during lipid peroxidation. Acute injuries and delayed complications under the action of ionizing radiation.

Body	Acute injury	Delayed injury
Bony	Atrophy.	Hypoplasia, leukemia
Skin	Erythema	Atrophy of the epidermis and fibrosis, dermis: cancer
Heart		Interstitial fibrosis
Lungs	Edema, death of epithelial and endothelial cells	Interstitial and intraalveolar fibrosis; cancer
gastrointestinal tract	Edema, mucosal ulcersshell	Ulcers; fibrosis; strictures; cancer
Liver	Veno-occlusive diseases	Cirrhosis; liver tumors
Kidneys	Vasodilatation	Atrophy of the cortical substance, Interstitial fibrosis
Bladder	Erosions of the mucous membrane	Submucosal fibrosis; cancer
Mainbrain	Edema, necrosis	White matter necrosis, gliosis brain tumors
Testicle	Necrosis	Tubular atrophy
Ovary	Atresia of follicles	Fibrosis of the stroma
Thyroid	-	Hypothyroidism; cancer
Mammary gland	-	Fibrosis; cancer
Thymus, lymph nodes	Atrophy	Lymphoma

Acute disturbances in the genetic apparatus of cells occur even under the influence of small doses (less than 0.5 Gy). Such damages include increased expression of c-IO5, c^shi and c-tus proto-oncogenes, induction of cytokines, such as tumor necrosis factor (TNF), and activation of antioxidant protective enzymes, for example, superoxide dismutase. Free radicals, which are formed directly or indirectly under the action of ionizing radiation, can lead to the development of "oxidative stress", which leads to the activation of the transcription of some substances that enhance the synthesis of various proteins. DNA damage itself causes increased synthesis of proteins involved in DNA repair, cell division arrest, and apoptosis. As is known, the p53 tumor suppressor gene is activated in various types of DNA damage: its protein product changes into an activated form as a result of post-translational transformation. Under its influence, the cell cycle stops,

DNA repair is activated, and if the integrity of DNA cannot be restored, the mechanism of apoptosis is triggered.

Fibrosis. An important late complication under the influence of ionizing radiation, usually in the doses used for radiotherapy of tumors, is the replacement of normal parenchymal tissue by fibrous tissue, which leads to scarring of the organ and disruption of its function. These fibrotic changes can develop both as a result of acute cell necrosis in organs with incomplete regeneration, and as a result of ischemic damage due to damage to blood vessels. In addition, in the mammary gland and lungs during irradiation, damaging cytokines and growth factors that contribute to sclerosis are released, which persist for several weeks after irradiation.

Carcinogenesis. As a result of exposure to ionizing radiation, the risk of various malignant tumors increases, especially skin cancer, leukemia, osteogenic sarcomas, and lung cancer. The disease most often develops 10-20 years after exposure. Thus, Japanese survivors of the atomic bombings of Hiroshima and Nagasaki had an increased incidence of all types of leukemia, except for chronic lymphocytic leukemia. In children, there was an increased incidence of cancer of the mammary and thyroid glands andto a lesser extent - cancer of the urinary tract and urinary organs.

The mechanism responsible for late carcinogenesis is still poorly understood. The long latent period between exposure to radiation and the development of cancer is attributed by some to the occurrence of the so-called induced genetic instability. Quantitative analysis of mutated genes in irradiated cells showed that pathological genes can be transmitted in the population of cells for several generations.

Disorders of growth and development. The embryo and the child's body are very sensitive to ionizing radiation. The greatest sensitivity is observed in the following 4 phases of development:

1. Embryo implantation. When the mother's body is irradiated before implantation, the embryo dies.
2. Critical phases of embryogenesis. When the mother's body is irradiated, even with

for diagnostic purposes, from the moment of implantation to the 9th week of pregnancy is observeda large number of various developmental disorders, which in most cases are fatal. During this period, the greatest susceptibility is observed not only to radiation, but also to other teratogenic factors.

3. Fatal period. From the 9th week to the end of pregnancy, exposure to ionizing radiation leads to impaired development of the central nervous system and reproductive organs. Ischemic damage, atrophy and fibrosis are observed in the organs supplied with blood through the affected vessels.

Skin. Hair follicles and epidermis are most sensitive to the effects of ionizing radiation. Desquamation of the epidermis is often observed, its foci are replaced by atrophic epidermis with hyperkeratosis, hyper- or hypopigmentation. Vessels can

thin and expand, they are often surrounded by dense bundles of collagen fibers. Impaired wound healing, increased sensitivity to infections and ulceration are observed. These changes are called contact dermatitis. As already mentioned, skin cancer, especially basal cell and squamous cell, can develop 20 or more years after exposure.

Heart. The heart and pericardium are often damaged as a result of radiation therapy in the chest area for lymphomas, lung and breast cancer. Fibrosis of the pericardium leads to the development of constructive pericarditis. Less often, myocardial ischemia and, as a result, cardiosclerosis develop as a result of damage to the coronary arteries.

LungsLungs are easily damaged by ionizing radiation. Acute pulmonary insufficiency often develops, in the later Term — radiation pneumonia. They develop both intra-alveolar and interstitial fibrosis. The risk of lung cancer is much higher in smokers, because there is a synergistic effect of these two factors in carcinogenesis. In cigarette smoke, in addition to carcinogenic substances, two radionuclides are detected: Pb210 and Po210. Sometimes in minesdetect Ka222. These miners often have a mutation (guanine -> thymidine) at codon 249 in p53 gene suppressor tumors.

Kidneys and bladder. Kidneys are moderately susceptible to radiation damage. They gradually develop peritubular necrosis, damage to blood vessels, hyalinization of glomeruli, which will ultimately lead to hypertension and atrophy of the kidneys. Acute necrosis of the epithelium can be observed in the bladder, then submucosal fibrosis, contractures, bleeding, and ulceration develop.

Gastrointestinal tract. As a result of exposure to ionizing radiation, there is a delay in the neuropsychological development of children. The risk of childhood leukemia and tumors also increasesnervous tissue.

Postnatal period. When irradiated in childhood, there is a violation of growth and differentiation of bone tissue. The development of the nervous system, eyes and teeth can also be disturbed.

Congenital mutations. On Ogoizorpiia flies and mice it was proved that mutations thatarise under the action of ionizing radiation, can be inherited. Despite the fact that chromosomal aberrations in blood cells are found in people who survived the atomic bombing and workers of nuclear power plants, such changes are not found in their descendants. Geneticists believe that some recessive mutations can still be passed on to offspring and accumulate in the population. However, a clear relationship between the number of mutations in human germ cells and the received dose was not found.

Delayed manifestations of exposure. After several months or years, late complications may occur (carcinogenesis was discussed above). As a result of such complications, the normal function of vital organs may be disturbed: lungs, heart, kidneys, central nervous system. Infertility can also develop in both men and women. Vision may be impaired due to the development of cataracts, and intestinal obstruction is also sometimes observed as a result of the growth of connective tissue in the intestines. Fibrous structures and chronic

ulcers can be observed on the skin, in the gastrointestinal tract, bladder, and vagina. Chronic disorders in small vessels and excessive formation of connective tissue can complicate various surgical interventions. Healing is often disrupted earlier, they develop infectious processes.

Blood vessels. After the initial inflammatory reaction, accompanied by necrosis of the endothelial cells, subendothelial fibrosis, fibrosis of the muscular membrane, destruction of the internal elastic membrane, significant narrowing of the lumen of the vessel develops in the blood vessels in the irradiated area. Capillaries can become thrombosed, obliterated or, conversely, dilated (capillary ectasia). INabsorption of electrolytes and fluid. As a result of vascular damage, ischemia, ulceration, and atrophy of the mucous membrane occur. As a result of fibrosis, structures can develop that lead to intestinal obstruction.

Mammary gland. Even diagnostic chest X-rays can increase the risk of developing breast cancer. Radiotherapy of breast cancer leads to the development of a pronounced fibrotic reaction with a high polymorphism of epithelial cells.

Ovaries and testicles. Spermatogenic cells are very sensitive to radiation; even small doses can lead to disruption of meiosis and infertility. As a result of sclerosis of blood vessels, fibrosis of seminiferous tubules is observed, while Sertoli cells and interstitial cells of Leydig are not damaged. Follicles in the ovary are rapidly destroyed.

Eyes and nervous system. The lens is unstable to the action of ionizing radiation, cataracts often develop in it. The vessels of the retina and ciliary body are often damaged. Foci of necrosis and demyelination of nerve fibers can develop in the brain. As a result of irradiation of the spinal cord, sclerosis of blood vessels occurs in it, which leads to necrosis of cells, demyelination of fibers and, as a result, paraplegia. This process is called transverse myelitis.

Ultraviolet radiation

Sunlight contains radiation with wavelengths from 200 to 4000 nm, including ultraviolet, visible, and infrared. Depending on the wavelength, ultraviolet radiation is divided into three types - UV-A, UV-B and UV-C (see Table 24.4). Ultraviolet radiation makes up from 3 to 5% of the total flow of sunlight that penetrates to the earth's surface. The Earth's ozone layer plays a very important role because it completely absorbs UV-C and partially UV-B. Ordinary glasses, which completely absorb UV-B, but pass UV-A, also play a protective role against ultraviolet radiation. Ultraviolet radiation has two main effects: it accelerates skin aging and increases the risk of skin cancer.

Acute changes under the influence of UV-A and UV-B are reversible and disappear quickly. These include erythema, pigmentation, and damage to Langerhans cells and keratinocytes in the skin. At the same time, the mechanisms and mediators involved in the process differ depending on the type of radiation. Depending on the duration of exposure, erythema, swelling and acute inflammation occur as a result of the release of histamine from smooth

cells in the dermis and the synthesis of arachidonic acid metabolites. When exposed to UV-B, interleukin 1 is also released. When exposed to UV-Athere is a rapid temporary darkening of melanin as a result of its oxidation, which is most pronounced in people with dark skin. Tanning under the influence of UV-A and UV-B occurs as a result of an increase in the number of melanocytes, the elongation and spread of their appendages, and the transfer of melanin to keratinocytes. Tanning determines the resistance of the skin to UV-B and partly to UV-A. Both UV-A and UV-B lead to the destruction of Langerhans cells and, as a result, to disruption of immune processes in the skin. UV-B causes apoptosis of keratinocytes, while "sunburn cells" appear in the epidermis, not in make-up keratin.

Repeated exposure to ultraviolet radiation leads to the appearance of signs of aging in the skin (wrinkles, solar elastosis, uneven pigmentation). Unlike ionizing radiation, which activates tissue collagenization, ultraviolet radiation leads to the destruction of elastin and collagen, which results in the formation of wrinkles and a decrease in skin elasticity. These changes are irreversible. The reason for this process is an increase in the activity of the elastin gene andsynthesis of metalloproteases that destroy collagen. As a result, enzymatic destruction of type I collagen occurs.

Damage to the skin under the influence of UV-B occurs as a result of the formation of active oxygen-containing substances and damage to natural pigments, for example, melanin. Also, ultraviolet radiation leads to DNA damage, which manifests itself in the form of formation of pyrimidine dimers between adjacent pyrimidine bases in the same DNA strand. Pyrimidine-pyrimidone-(6-4)-phosphoproducts, breaks in one of the DNA strands, and crosslinks in DNA proteins can also be formed. When studying the genetic apparatus of skin cancer cells, the same changes are often found in the p53 gene: the replacement of T by T or DTC by TT. These observations confirm the role of ultraviolet radiation in the development of skin cancer.

Electromagnetic poles

Non-ionizing electromagnetic poles can have frequencies from 1 Hz to 100 Hz (microwave radiation from radars). There is evidence that exposure to loud noise with a frequency of only 50-60 Hz increases the risk of leukemia in children. There are reports of an increased incidence of leukemia and brain tumors among electricians working on high-voltage power lines.However, these facts were not proven in various experiments on animals.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Vitamin diseases, species, etiology, morphological characteristics, consequences.
2. Occupational diseases, types, etiology, morphological characteristics, consequences.
3. Radiation sickness, etiology, morphological characteristics, consequences.
4. Parathyroid osteodystrophy, etiology, morphological characteristics, consequences.
5. Osteomyelitis, etiology, morphological characteristics, consequences.
6. Fibrous dysplasia, etiology, morphological characteristics, consequences.

7. Osteopetrosis, etiology, morphological characteristics, consequences.
8. Paget's disease, etiology, morphological characteristics, consequences.
9. Muscular dystrophies, myasthenia, diseases, etiology, morphological characteristics, consequences.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

24. methods: assessment of the correctness of the performance of practical skills
25. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Pathology of the female and male reproductive system. Pathology of pregnancy, postpartum period and placenta. Diseases of the mammary gland.".

Suggested topics for essays:

1.Occupational diseases, types, etiology, morphological characteristics, consequences.

2. Radiation sickness, etiology, morphological characteristics, consequences.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 31

Topic: Pathology of the female and male reproductive system. Pathology of pregnancy, postpartum period and placenta. Breast disease.

Goal:learn to determine etiology, pathogenesis, morphologydiseasesfemale and male reproductive system, pathology of pregnancy, postpartum period and placenta, diseases of the mammary gland.

Basic concepts: Pathomorphological changes of diseasesfemale and male reproductive system, pathology of pregnancy, postpartum period and placenta, diseases of the mammary gland.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Dyshormonal diseases of genital organs and mammary gland

Dyshormonal diseases of the genital organs and mammary gland include nodular hyperplasia and adenoma of the prostate gland, glandular hyperplasia of the endometrium, endocervicosis, adenomatosis and polyps of the cervix, and benign dysplasia of the mammary gland.

Nodular hyperplasia and adenoma of the prostate gland(dyshormonal hyperplastic prostopathy) is observed in 95% of men over the age of 70. At the same time, the gland is enlarged, m□which is elastic, sometimes bumpy. The middle part of the gland, which protrudes into the lumen of the bladder, increases especially sharply, which leads to difficulty in the outflow of urine. On autopsy, the gland appears to consist of separate nodes separated by layers of connective tissue.

According to the histological structure, glandular (adenomatous), muscle-fibrous (stromal) and mixed

forms of nodular hyperplasia.

*Glandular hyperplasia*characterized by an increase in the number of glandular elements, while the number and size of particles are different. Muscular-fibrous (stromal) hyperplasia is characterized by an increase in the number of muscle fibers, among which there are atrophied glands, gland lobulation is disturbed. In mixed prosthopathy, there is a combination of tissue disorders characteristic of the first two types; possible

formation of retention cysts.

*Adenoma*prostate gland does not have any histological features.

Complications of dyshormonal hyperplastic prostopathy include diseases and deformation of the urinary canal and bladder neck, as a result of which urine output is delayed, compensatory hypertrophy occurs in the bladder wall. However, the compensation becomes insufficient, in the bubble
excess urine accumulates, a secondary bacterial infection joins, cystitis, pyelitis, and ascending pyelonephritis develop: if the inflammation becomes purulent, urosepsis may develop.

Glandular hyperplasia of the mucous membrane of the uterus- a fairly widespread disease that occurs in connection with a violation of the hormonal balance and the entry into the body of an excessive amount of folliculin or the hormone of the corpus luteum (progesterone). Mostly mature and elderly women are affected, sometimes with presence
ovarian tumors producing estrogen hormones, as well as with hormonal dysfunction of the ovaries.
The disease is accompanied by uterine bleeding.

Endometrium with glandular hyperplasia has a characteristic appearance: sharply thickened, with polypous growths. Upon histological examination, the mucous membrane corresponds to a prolonged phase of proliferation, which acquires a pathological state as a result of increased secretion of estrogens: the glands are envious, dusty or corkscrew-shaped,
elongated; growth of the stroma with hyperplasia of its cells is observed at the same time. In such cases, when glandular cysts are formed, we are talking about glandular-cystic (cystic) hyperplasia, and when signs appear atypia - about atypical hyperplasia.

With glandular hyperplasia, inflammation of the mucous membrane with subsequent sclerosis is possible, as well as

the development of cancer of the uterine body, therefore glandular hyperplasia of the endometrium is classified as a precancerous state of the uterus.

Endocervicosis -accumulation of glands in the thickness of the vaginal part of the uterus with a change in the covering epithelium.

Proliferating, simple, and healing endocervicosis are distinguished, which should be considered as stages of development. Proliferating endocervicosis is characterized by the neoplasm of gland-like structures that develop from cambial elements of the prismatic epithelium of the cervical canal (it is able to differentiate into glandular, and in squamous epithelium). With simple endocervicosis, the glands do not have signs of a neoplasm. The growth of squamous epithelium in the glands and its replacement by prismatic epithelium is typical for endocervicosis, which heals.

Under**adenomatosis**the cervix understands such a process, when under the covering epithelium of its vaginal part, glandular formations, lined by one layer of cuboidal epithelium, grow.

Polypsthe cervix arises in the wall of the canal, less often - in its vaginal part, formed by a prismatic epithelium that secretes mucus.

Endocervicosis, adenomatosis and cervical polyps should be considered*precancerous process.*

Benign dysplasia of the mammary gland(synonyms: mastopathy, fibrocystic disease) characterized by a violation of the differentiation of the epithelium, its atypia, a change in histostructure, but without penetration through the basal membrane and the possibility of reversible development. Its development is associated with a violation of the balance of estrogens.

There are two main forms of mastopathy - non-proliferative and proliferative.

For*non-proliferative form*characteristic growth of dense connective tissue with areas of hyalinosis, in which atrophic lobes and cystic dilated ducts are located. Ducts and cysts are lined with atrophic or high (apocrinized) epithelium, which forms papillary growths. This form of dysplasia can to be in the form of a single dense node (nodes) - this is fibrous mastopathy; or a whitish dense node with cysts in it (fibrocystic mastopathy) more often in one mammary gland.

*Proliferative form*is characterized by proliferation of epithelium and myoepithelium or conjoined proliferation of epithelium and connective tissue. Varieties of this form of mastopathy are adenosis (masoplasia) - proliferation of intraductal or lobular epithelium. Adenosis (masoplasia) is characterized by an increase particle sizes in connection with the proliferation of glandular epithelium. The growth of ductal or lobular epithelium leads to the formation of structures of the solid, adenomatous, and cribriform type, at the same time it grows connective tissue. In sclerosing (fibrosing) adenosis, the proliferation of myoepithelium prevails. At the same time, foci formed by myoepithelial cells and epithelial tubules appear; later sclerosis and hyalinosis of the entire gland join. Against the background of benign dysplasia of the mammary gland, it is not uncommon cancer develops, in connection with which they are classified as precancerous conditions.

Inflammatory diseases of genital organs and mammary gland

Inflammatory processes of the genital organs are quite often manifestations of the main disease, for example, tuberculosis, syphilis, gonorrhea, etc. Inflammation of the mucous membrane of the uterus (endometritis), inflammation of the mammary gland (mastitis), inflammation of the testicle (orchitis) and prostate gland (prostatitis) are of the greatest importance.

Endometritis can be acute or chronic. Acute endometritis quite often complicates or deepens childbirth or abortion. Its causative agents are staphylococci, streptococci, anaerobic bacteria, and intestinal bacteria stick and others. The endometrium is thickened, covered with a gray-yellow purulent film. When spreading purulent metritis and thrombophlebitis occur during the inflammatory process on myometrial vessels. Chronic endometritis is characterized by chronic catarrh of the mucous membrane of the uterus with mucous-purulent exudate, sometimes significant (white - fluor albus). The endometrium is full of blood, infiltrated by various cells (neutrophils, plasma cells, lymphocytes). The epithelium of the glands is in a state of desquamation and proliferation. With a long course of endometritis there is atrophy of the glands, fibrosis of the stroma and its infiltration by lymphoid cells - atrophic endometritis. When the fibrous tissue squeezes the excretory ducts of the glands, cysts filled with mucus are formed (cystic endometritis).

If hyperplasia occurs in the mucous membrane during chronic inflammation, then we are talking about hypertrophic endometritis, in which differential diagnosis with glandular hyperplasia of the endometrium is complicated.
Mastitis is an inflammation of the mammary gland, depending on the course, it can be both acute and chronic.

Acute purulent (phlegmous) mastitis is quite common in women in the postpartum period;

more often, its causative agent is staphylococcus. In most cases, chronic mastitis is a consequence of acute and purulent inflammation.

Orchitis- inflammation of the testicle, which can be both acute and chronic.
Acute orchitis in most cases is a complication of some infectious diseases (typhoid, scarlet fever, gonorrhea, tuberculosis) and especially epidemic parotitis (20-30% of cases). According to the nature of the exudate, it is a purulent inflammation;
with epidemic parotitis - diffuse intermediate inflammation with lymphocytic and plasmacytic infiltration.
Chronic orchitis can be both a consequence of acute and a manifestation of chronic infectious diseases (syphilis, actinomycosis, tuberculosis) or trauma to the testicle. Autoimmune processes (autoimmune orchitis) sometimes take part in its development. This type of orchitis is characterized by chronic diffuse or granulomatous inflammation; at penetration of spermatozoa into the stroma of the testis, peculiar spermatozoal granulomas are formed. The consequence of chronic orchitis is unfavorable (infertility).

Prostatitis- inflammation of the prostate gland, a fairly common disease in men during active sexual life. The course is both acute and chronic.
The causative agent of acute prostatitis is mostly coccal bacteria (strepto-, gono-, staphylococci). According to morphological features, catarrhal, follicular and parenchymal prostatitis are distinguished, which follow the course considered as stages of an acute inflammatory process. In the catarrhal form, purulent catarrh of the ducts of the prostatic glands, edema of the connective tissue base, and sharp hyperemia develop. This form usually turns into a follicular one, in which changes in the ducts are accompanied by a general infiltration of the gland. In the parenchyma form, leukocyte infiltration becomes diffuse; abscesses appear and granulation tissue grows.
The development of chronic prostatitis is associated with infectious diseases (gonorrhea, chlamydia, mycoplasma infection, etc.), it is characterized by lymphohistiocytic infiltration of the stroma of the gland, growth granulation and scar tissue; sometimes granulomas occur.
Atrophy of the glands is associated with proliferation and metaplasia of the epithelium of the ducts, which leads to the formation of cribriform and papillary structures. Complications of prostatitis, especially chronic prostatitis, are recurrences of the infectious inflammatory process of the urinary tract.

TUMORS OF THE GENITAL ORGANS AND BREAST GLANDS
Genital and mammary gland tumors are diverse in origin, nature of growth, and features of metastasis. These are epithelial and mesenchymal tumors, both benign and malignant; some of them have a peculiar specificity.
Cancer of the uterus. Among malignant tumors of the female genital organs, uterine cancer ranks second after cancer mammary gland. Cancer of the cervix and cancer of the body of the uterus are distinguished.

Cancer of the cervix is more common than cancer of the body of the uterus. To date, it has been established that cervical cancer

of the uterus are preceded by precancerous conditions, such as endocervicosis and severe dysplasia of the epithelium of the vaginal part of the neck. According to the nature of tumor growth, cervical cancer can be non-invasive (cancer in situ) or invasive. Cancer of the vaginal part of the neck and cancer of the cervical canal are distinguished by localization. Cancer of the vaginal part of the cervix grows exophytically, in the cavity of the vagina, ulcerates early, less often - in the wall of the cervix and its surrounding tissues. Cancer of the cervical canal, as a rule, grows endophytically in the wall of the neck, adjacent tissue and grows into the wall

bladder and rectum. When the tumor is ulcerated, vaginal-bladder or vaginal-rectal fistulas (fistulas) are formed.

According to histological structure, cervical cancer is squamous, glandular (adenogenic) and *glandular-squamous*with different degrees of differentiation. In addition, they are also allocated *endometrioid adenocarcinoma*cervix

Metastases occur early and spread primarily through lymphatic pathways to pelvic, inguinal, and extra-abdominal lymph nodes; later hematogenous metastases are also observed.

Cancer of the uterine body occurs more often in women over 50 years of age. In the development of cancer of the uterine body, a significant place is occupied by a violation of the hormonal balance (estrogen content), which causes hyperplastic changes in the epithelium of the mucous membrane of the uterus with its subsequent malignancy. The development of cancer is preceded by precancerous changes, which include endometrial hyperplasia and polyps.

Cancer of the uterine body grows mostly exophytically, looks like a cauliflower or a polyp on a wide stem (exophytic growth). Sometimes the tumor occupies the entire cavity of the uterus, is subject to ulceration and necrosis with successive decay; endophytic tumor growth is rarely observed.

According to the histological structure, cancer of the uterine body is an adenocarcinoma, which can be highly moderately or poorly differentiated; undifferentiated cancer is rare.

Metastases of cancer of the uterine body are observed, first of all, in the lymph nodes of the small pelvis, hematogenous metastases are rare.

Malignant tumors of the uterus also include chorioepithelioma (see Tumors of exocrine glands and epithelial coverings).

Ovarian cancer. Among tumors of the female genital organs, ovarian cancer ranks second after cervical cancer. I can develop from normal components of the ovary (covering mesothelium, ovum and

its derivatives, granulosa cells), its rudimentary formations (duct of the primary kidney, or Wolff's duct), as well as embryonic remains. However, the vast majority of malignant ovarian tumors are the result of malignancy of benign epithelial serous or mucinous tumors. Ovarian cancer has a lumpy appearance

nodes of different sizes, that is, they are malignant serous and pseudomucinous tumors (see Tumors of exocrine

glands and epithelial covers).

Tumor metastases are lymphatic and hematogenous, occur in lymph nodes, peritoneum,

internal organs.

Breast cancer. It ranks first among all malignant neoplasms in women. In most cases, breast cancer develops against the background of precancerous changes. This is primarily benign dysplasia of the mammary gland and ductal papilloma. Breast cancer is microscopically nodular and diffuse,

as well as cancer of the nipple and nipple field (Paget's disease). Nodular cancer is characterized by the development of a node in

diameter up to several centimeters; in some cases, the knot is dense with layers of connective tissue that penetrates into the adjacent fatty tissue, in others it is soft, juicy on dissection, and easily disintegrates.

*Diffuse cancer*covers almost the entire gland, sometimes the tumor grows into the skin and forms on its surface

a mushroom-shaped node with ulceration - a cancerous ulcer. In some cases, the tumor spreads over the surface of the gland and then the gland becomes as if covered with a shell (shell cancer).

According to histological structure, the following types of breast cancer are distinguished: 1) non-infiltrating: intralobular and intraductal; 2) infiltrating.

The spread of breast cancer is associated with germination in soft tissues. Lymphogenic metastases appear in regional lymph nodes: inguinal, anterior thoracic, subclavian, supraclavicular, peristernal; hematogenous - in bones, lungs, liver, less often - kidneys.

Prostate cancerranks second among oncological diseases in men and

observed in old age. Hormonal factors play a significant role in the development of cancer of this gland, and, first of all, a violation of the secretion of androgens. Sometimes the development of cancer is preceded by nodular hyperplasia of the prostate gland. Macroscopically - the gland is enlarged, lumpy, dense. On autopsy, it looks like white cords

connective tissue, interwoven with each other, between them is cancerous tissue

gray-yellow color. Microscopically - the structure of adenocarcinoma, less often - undifferentiated cancer.

Cancer of this gland spreads to nearby organs, first of all, it grows in the bladder,

rectum, seminal vesicles. Cancer metastases are observed both in the lymph nodes of the small pelvis,

iliac and inguinal, as well as hematogenous - in internal organs, especially in bones. Testicular cancer is rare. Seminoma, embryonal cancer and teratoblastoma are more common. Sometimes chorionepithelioma develops from teratoid tumors.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Dyshormal diseases of female genital organs, etiology, morphological characteristics, consequences.
2. Dyshormal diseases of male genital organs, etiology, morphological characteristics, consequences.
3. Inflammation of the female genital organs, etiology, morphological characteristics, consequences.
4. Inflammation of the male genital organs, etiology, morphological characteristics, consequences.
5. Inflammation of the mammary gland, morphological characteristics, consequences.
6. Breast dysplasia, etiology, morphological characteristics, consequences.
7. Paget's tumor, etiology, morphological characteristics, consequences.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

A histological examination of a scraping of the walls of the uterine cavity of a 45-year-old woman with ovarian and menstrual cycle disorders revealed an increase in the number of endometrial glands, some of which are convoluted, some of which are cystically enlarged. Diagnose the disease.
Correct answer Gland - cystic hyperplasia of the endometrium
B Adenocarcinoma of the endometrium
Atypical endometrial hyperplasia
Placental polyp
Endometrial glandular polyp
Microscopic examination of the cervical biopsy revealed cellular and nuclear atypia of the multilayered squamous epithelium, pathological mitoses, as well as horn pearls in the depth of the epithelial layers. Your diagnosis:
Correct answer Squamous cell carcinoma with keratinization

BTransitional cell cancer
Squamous cell carcinoma without keratinization
Glandular cancer
Anaplastic cancer.
A histological examination of a scraping of the uterine mucosa of a 54-year-old patient with a clinical diagnosis of ovarian-menstrual cycle disorder revealed the growth of atypical glandular structures consisting of cells with hyperchromic nuclei, mitotic figures, and atypia. Atypical glandular structures grow into the myometrium. Which pathological process is characterized by microscopic changes?
Correct answer Adenocarcinoma of the uterus
BAcute endometritis
Chorioepithelioma of the uterus.
Placental polyp
Glandular hyperplasia of the endometrium
In a 46-year-old woman, during a palliative operation for stomach cancer, the presence of Krukenbergian metastases in the ovaries ("Krukenbergian ovarian cancer") was established. which of the following routes of metastasis led to ovarian damage?
Correct answer Implantation
BLymphogenic orthograde
CLymphogenic retrograde
DHematogenous
Canalicular
In connection with acute pain in the iliac region, a young woman had her fallopian tube removed with a local expansion of its middle third, filled with blood. During histological examination, chorionic villi, large fields of erythrocytes with an admixture of leukocytes were found in the opening of the tube. Your diagnosis:
Correct answer Tubal pregnancy
BAcute purulent salpingitis
Bleeding in the fallopian tube
D Hemorrhagic salpingitis
Purulent salpingitis
The autopsy of a 73-year-old man revealed an enlarged, soft, elastic, slightly bumpy prostate gland, which on cross-section consists of separate nodes separated by layers of connective tissue. Microscopy revealed an increase in the number of glandular elements. The size of the particles and the number of glandular elements in them are different. What process takes place in the prostate gland?
Correct answer Glandular nodular hyperplasia
BMuscular - fibrous (stromal) nodular hyperplasia
C Mixed nodular hyperplasia
Adenocarcinoma
Undifferentiated cancer
During histological examination of the prostate gland surgically removed from a 72-year-old man who complained of difficult urination, an increase in the number of

glandular and muscular elements was found. The lobular structure of the gland is disturbed. What is the most likely process in the prostate gland?
Correct answer Mixed form of prostatopathy
BMuscular-fibrous hyperplasia
Glandular hyperplasia
Prostatitis
Adenocarcinoma
The patient was 42 years old, suffered from menometrorrhagia, and a supravaginal amputation of the uterus was performed. Macroscopic examination revealed multiple intramural and submucosal dense nodes in the uterus, 1 to 5 cm in size, whitish in section, fibrous structure. Microscopically, they are represented by randomly arranged bundles of smooth muscle fibers. Your diagnosis?
Correct answer Multiple leiomyoma
B Chorion carcinoma
CFibroma
D Polyp
Endocervicosis
A 57-year-old female patient developed periodic uterine bleeding. For diagnostic purposes, the uterine cavity was scraped. In the obtained material, glandular complexes of various sizes and shapes, formed by atypical cells with hyperchromic nuclei, with numerous mitoses (including irregular ones) are observed among the blood elements. Your diagnosis:
Correct answer Cancer of the body of the uterus (adenocarcinoma)
BUterine fibromyoma
Chorioepithelioma
Glandular hyperplasia of the endometrium
Endometritis
The 23-year-old patient's condition deteriorated sharply the next day after giving birth, her body temperature rose to 39.0C, discharge from the uterus was yellow-green with an unpleasant smell. Microscopic examination of the endometrial scraping revealed continuous neutrophilic granulocytes. Your diagnosis:
Correct answer Acute endometritis
BGlandular hyperplasia of the endometrium
Cancer of the body of the uterus
D Physiological state
Sepsis

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;

4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

26. methods: assessment of the correctness of the performance of practical skills
27. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.

"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Pre- and perinatal pathology. Asphyxia of newborns. Birth injury.".

Suggested topics for essays:

1.Ovarian tumors, types, etiology, morphological characteristics, consequences.

2. Tumors of the prostate gland, etiology, morphological characteristics, consequences.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine

2.www.ama-assn.org– American Medical Association /American Medical Association

3.www.who.int- World Health Organization

4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine

5.http://bma.org.uk– British Medical Association

6.www.gmc-uk.org- General Medical Council (GMC)

7.www.bundesaerztekammer.de– German Medical Association

8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 32

Topic: Pre- and perinatal pathology. Asphyxia of newborns. Birth trauma.

Goal:to know the pathological changes of the fetus and the newborn in the pathology of pregnancy. Morphological changes in fetal asphyxia; methods of determining asphyxia.

Basic concepts:classification of diseases of pregnancy, postpartum period; classification of litter pathology; principles of diagnosis of pathology of pregnancy, postpartum period and litter; characteristic morphological features of any of the forms of the aforementioned pathology.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

DISEASES OF PREGNANCY AND THE POSTPARTUM PERIOD

Neurohumoral changes that occur during pregnancy can lead to disruption of its normal development, which creates prerequisites for the occurrence of pregnancy pathology.

Pregnancy pathology includes: 1) gestosis (pregnant toxicosis), 2) ectopic pregnancy, 3) spontaneous abortion; 4) premature birth; 5) bladder drift. After childbirth or abortion, placental polyps, chorionepithelioma, and congenital infection of the uterus develop.

Preeclampsia(from the Latin gesto - to carry, to be pregnant), or toxicosis of pregnant women - a group concept that unites dropsy of pregnant women, nephropathy, preeclampsia and eclampsia.

Etiology and pathogenesis. The causes of preeclampsia have not been established. Among the numerous theories of pathogenesis (renal, hormonal, coagulation,

neurogenic, etc.), the most evident is the immunological one, which is based on the weakening of the mother's immune recognition of fetal antigens when the barrier properties of the placenta are disturbed. Insufficient immune recognition of fetal antigens by the mother, as well as insufficient production of suppressive factors (T-suppressors, blocking antibodies, etc.), are associated with the relative homozygosity of the pregnant woman, husband and fetus according to D-antigens of histocompatibility. The lack of suppressive factors leads to the development of immune cell and immune complex reactions. Immune complexes appear not only in the blood of pregnant women, but also in the vessels of the placenta, the changes of which resemble the reaction of transplant rejection. With immune complex reactions ulcers during gestosis and damage to a number of internal organs, in particular the kidneys (nephropathy of pregnant women). The sensitivity to angiotensin increases sharply, which leads to widespread angiospasm and arterial hypertension.

A major role in the pathogenesis of preeclampsia is played by blood coagulation disorders, which are largely associated with the release of thromboplastin by the placenta. The syndrome of disseminated intravascular coagulation (DVS-syndrome) develops, which is especially pronounced in eclampsia.

*Eclampsia*among the manifestations of toxicosis of pregnant women, the most clinically significant and dangerous, develops in the second half of pregnancy (late toxicosis of pregnant women), less often - during childbirth and the postpartum period.

Pathological anatomy of eclampsia. The changes are represented by disseminated thrombosis of small vessels, numerous small necrosis and hemorrhages in internal organs. At autopsy, swelling, jaundice, pronounced changes in the brain, lungs, heart, liver and kidneys are found. In the brain, edema, blood clots in small vessels, hemorrhages are found, more frequent in the subcortical nuclei, in the lungs - edema and congestive hemorrhagic pneumonia, in the heart - blood clots in vessels, focal necrosis of the myocardium and hemorrhages. The liver is enlarged, variegated, with numerous hemorrhages. Microscopic examination reveals thrombi in small vessels, hemorrhages, and foci of necrosis. The kidneys are enlarged, flaccid, their cortical layer is swollen, variegated, and the medulla is sharply full-blooded. Sometimes they find - symmetric necrosis of the cortical substance of the kidneys.

Deathoccurs from liver or kidney failure, as well as from DIC and hemorrhages in vital organs.

Ectopic pregnancy -development of the fetus outside the uterine cavity: in the tube (tubal pregnancy), in the ovary (ovarian pregnancy) or in the abdominal cavity (peritoneal pregnancy). Tubal pregnancy is the most common. The development of an ectopic pregnancy is associated with those changes in the fallopian tubes that prevent the progress of a fertilized egg through them (chronic inflammation, congenital anomalies, tumors, etc.).

*tubal pregnancy,*usually observed in one pipe. If the egg is attached and develops in the ventral end of the tube, it is said to be an ampullary tubal pregnancy, if in the uterine end of the tube (isthmus region), it is an interstitial tubal pregnancy. During

growth, the fetal egg can break the tube and be placed between the leaves of the broad ligament, then an ectopic interligamentous pregnancy occurs.

During a tubal pregnancy, a decidual reaction develops in the mucous membrane of the tube, where the egg is attached and formed, which is characterized by the appearance of large and light-colored decidual cells both in the mucous membrane and in the wall of the tube. The fetal membrane also appears in the mucous membrane, and the villi of the chorion penetrate the muscle layer and its vessels, destroying the tissue elements of the tube. In connection with this, in the first months of tubal pregnancy, bleeding into the tubal cavity and the release of the fetus into the tubal cavity are possible - incomplete tubal abortion. The dead fetus and its blood-soaked membranes are ejected through the fimbrial end into the abdominal cavity - a complete tubal abortion.

A rupture of the pipe wall and bleeding into the abdominal cavity are possible, which can lead to the death of a woman. When the tube ruptures, the dead fetus can end up in the abdominal cavity, where it dies and mummifies ("paper fetus") or calcifies (lithopedion); secondary abdominal pregnancy rarely develops.

During the operation to remove the tube with the fetal egg, the basis for the diagnosis of ectopic pregnancy is the detection of chorionic villi and decidual cells, not to mention the elements of the fetus. A decidual reaction is also found in the mucous membrane of the uterus (staple).

Spontaneous abortion and premature birth. They are abortions that occur at different times. Termination of pregnancy and removal of the fetus from the uterus before the 14th week from the moment of conception is marked as an abortion (miscarriage), from the 14th to the 28th week - as a late abortion, from the 28th to the 29th week - premature birth.

In an involuntary abortion, the entire fetal egg (fetus and membranes), which may be preserved or damaged, is thrown out of the uterus with blood clotting. With premature birth, the fetus is born first, and then the shell with the baby's place. During the histological examination of the fragments of the fetal egg, which were isolated independently or removed with an abrasive (scraping of the uterine cavity), the membranes of the fetus, chorionic villi and decidual tissue are revealed. Abortion often occurs when the fetus dies as a result of incomplete placement of the fetal egg in the mucous membrane of the uterus, failure of the mucous membrane itself, in the presence of hemorrhages, tumors, etc.

*Artificial abortion*performed according to medical indications in a medical institution. Abortion carried out in unsanitary conditions, outside a medical hospital, may cause infection of the uterus, development of sepsis; it may be subject to legal proceedings (criminal abortion).

Trophoblastic disease. Trophoblastic disease is a group concept. It includes cystic fibrosis, invasive cystic fibrosis, choriocarcinoma, and trophoblastic tumor of the placental bed. Placenta tissue is the source of the disease. Trophoblastic disease is relatively rare. Thus, there is 1 case of cystic fibrosis per 1,000 births, and 2 cases of choriocarcinoma per 100,000 births or abortions. Compared to Europe, the frequency

of trophoblastic disease is much higher in Asian and African countries. Differences in morbidity may be of a racial nature, or may be due to a large number of deliveries and the age of pregnant women (it has been established that the frequency of trophoblastic disease is increased in pregnant women younger than 16 and older than 35). The share of choriocarcinoma among malignant neoplasms of female genital organs is only 2.1%.

Cystic prolapse is manifested by vaginal bleeding in the first trimester, which can be accompanied by the release of cystic villi, and at the same time, an increase in the size of the uterus and an unusually high level of chorionic gonadotropin are observed. With cystic drift, cluster-like clusters consisting of numerous bubbles filled with transparent liquid are microscopically visible. Bubbles can be freely located in the uterine cavity and be released from the vagina. Microscopically, a sharp swelling of the villi is revealed, often with a lumen in the center of the villi of cavities (cisterns) filled with liquid. The degree of trophoblast proliferation can be different. With complete cystic drift, the entire placenta is affected; the fruit is usually absent. With a partial, as a rule, there is no noticeable increase in the volume of the placenta, vesicular villi are distributed among morphologically normal placental tissue. There is usually fruit, but it dies early.

*Complete bladder drift.*In this type of drift, there is a diploid set of chromosomes, all of parental origin. It is assumed that the chromosomes of the sperm double, and the nucleus of the egg is inactivated or dies. Rarely, dispersible fertilization is observed. In partial cystic drift, the karyotype is triploid, and the additional, third set of chromosomes is of parental origin. If the additional set of chromosomes is of maternal origin, hydropic transformation of the villi does not develop. Thus, the cystic transformation of the placental villi with the formation of a cystic drift is due to the predominance of parental chromosomes in the karyotype of the embryo.

After removal of cystic tissue, a woman's recovery most often occurs, but the possibility of disease progression is quite high. The risk of developing choriocarcinoma after complete cystic drift is about 5%. The frequency of choriocarcinoma after partial drift has not been established, but it is known to be significantly lower than with complete drift.

Invasive cystic drift is characterized by the growth of villi in the myometrium. Clinically, it is manifested by bleeding that occurs a few weeks after the removal of the cyst. Hemorrhagic foci of various sizes are determined microscopically in the myometrium. The liquid tissue of the trophoblast penetrates the entire wall of the uterus and spreads to the adjacent organs. Microscopically, swollen villi are found in the myometrium, more often in vessels. The degree of trophoblast proliferation can be different. The invasive nature of the introduction is not considered a sign of true neoplasia. A normal trophoblast has the capacity for invasive growth, and the villi of a normal placenta can penetrate deep into the myometrium. However, with invasive cystic drift, metastases can be observed, more often in the lungs and vagina. These metastases regress spontaneously or after a single course of chemotherapy.

Choriocarcinoma. This is a malignant tumor of the trophoblastic epithelium. About 50% of such neoplasms develop after pregnancy complicated by cystic fibrosis, 25% - after abortion, 2.5% - after ectopic pregnancy and 22.5% - after clinically normal pregnancy. Choriocarcinoma can occur immediately after termination of pregnancy, after a few weeks and even after 15-20 years. The most characteristic symptom is vaginal bleeding. Relatively often, the disease is manifested by signs caused by metastases. The development of pulmonary hypertension associated with the growth of metastatic nodes in the pulmonary arteries is possible. Choriocarcinoma is hormonally active because the trophoblast synthesizes chorionic ronadotropin. in connection therefore, regardless of the size of the primary tumor, an increase in the uterus and thickening of its mucous membrane with a pronounced decidual reaction are always noted. Choriocarcinoma is one of the most malignant tumors, but it is well treated with a combination of hysterectomy and chemotherapy. The exception is cases when it develops after a normal pregnancy. In these cases, the prognosis is extremely unfavorable.

Choriocarcinoma has the appearance of a juicy yellowish-white or variegated spongy node on a wide base. When located under the mucous or serous membranes, the node can shine through in the form of a dark cherry formation. Microscopically, choriocarcinoma consists of cytotrophoblast cells and polymorphic giant syncytiotrophoblast elements. There are never true villi in the tumor. The degree of atypism and mitotic activity in tumor cells varies significantly. With the help of immunohistochemical methods, chorionic gonadotropin can be found in these cells. There is no stroma and vessels in the tumor. The rapid growth of the tumor is accompanied by multiple foci of necrosis and hemorrhages. Choriocarcinoma is characterized by widespread early hematogenous metastases in the lungs (80%), vagina (30%), brain, liver, and kidneys.

Trophoblastic tumor of the placental bed is rare. Usually, this neoplasm develops after a normal pregnancy, but in the anamnesis of sick women, a high incidence of cystic drift is noted. The uterus is enlarged, white-yellow or yellowish-brown masses are visible in the myometrium, exploding into the cavity in the form of polyps. Microscopically, the tumor consists mainly of mononuclear cells of the intermediate trophoblast with an admixture of multinucleated cells that resemble multinucleated cells of the placental bed. Cells form islands and rods penetrating between muscle fibers. Hemorrhages and necrosis are not characteristic. Tumor cells secrete placental lactogen, chorionic gonadotropin is found in only a small part of them. The outcome of the disease is often favorable. A malignant course with metastases is observed in 10-20% of patients. Unlike choriocarcinoma, cells of trophoblastic tumors of the placental bed are insensitive to chemotherapy. The main treatment is surgical.

*Placental polyp*is formed in the mucous membrane of the uterus at the place of parts of the droppings stuck in it after childbirth or abortion. A polyp consists of villi, coils of fibrin, decidual tissue, which undergo organization, a connective tissue area appears in the uterus. Placental polyp interferes with the postpartum involution of the uterus, supports inflammation in the mucous membrane and is the cause of bleeding.

Congenital infection of the uterus -a very dangerous complication of the postpartum period, and streptococcus, staphylococcus, and Escherichia coli are the most important pathogens. Infection of the uterus leads to purulent endometritis, which can occur during or after childbirth. Obstetric infection occurs exogenously (non-observance of the rules of asepsis) or endogenously (an outbreak of an earlier infection during childbirth). In the most severe cases, endometritis can become septic. The inner surface of the uterus becomes dirty gray, covered with purulent plaque. The infection spreads along the course of lymphatic vessels and veins (lymphogenous and hematogenous), lymphangitis, phlebitis and thrombophlebitis develop. Endometritis is joined by metritis and perimetritis, which leads to peritonitis. As a result, the uterus turns into a septic focus, which determines the generalization of the infection.

PATHOLOGY OF THE FEED

The litter, consisting of the placenta, fetal membranes and umbilical cord, is an important intermediate element of the mother-fetus functional system. Its main role consists in timely and adequate supply of constantly growing needs of the fetus.

Age-related (involutive) changes and compensatory-adaptive processes

After the morpho-functional peak of activity at the 36th week of pregnancy, age-related changes naturally occur in the placenta, which reach a maximum during a carried-over pregnancy. Microscopically, whitish-yellow foci of necrosis, more often in the marginal areas, and small calcifications are visible in the placenta on the maternal surface; the placenta is pale, the borders of the cotyledons are smoothed. Microscopically, dystrophic changes are the main ones. They are expressed by strengthening the processes of fibrinoid transformation of the trophoblast and the inflow of fibrin from maternal blood. The result of this is the gluing of several or many villi, blocking the access of maternal blood to the villi of the chorion. Entire groups of chorionic villi die and form ischemic placental infarcts, in the areas of which calcium salts are deposited. Fibrosis of the stroma of the villi and sclerosis of their vessels is also observed.

Violation of implantation and placentation processes

Malformations of the shape of the placenta. The main changes in form, which negatively affect the fetus, the course of pregnancy and childbirth, include the placenta, surrounded by a shaft and surrounded by a rim. The process in terms of the nature of the changes is unambiguous, but with a shaft-shaped placenta it is expressed more sharply. It is a consequence of detachment and twisting of the edges of the placenta in the early stages of pregnancy. Microscopically, the shaft consists of necrotized villi and decidual tissue impregnated with fibrinoid, which gradually undergo hyalinosis. With a shaft-shaped placenta during pregnancy, bleeding is observed, premature births and the birth of a dead fetus are more common. Windowed placenta, bilobed, multipartite placenta and with additional lobes do not have serious

thanatogenetic significance, but are indirect signs of a disturbance at the stage of implantation and placentation.

Defects in the development of localization of the placenta. These defects include marginal or central placenta previa in relation to the internal opening of the uterus. Placenta previa occurs as a result of blastocyst implantation in the lower segment of the uterus. The reasons for such implantation are unclear, it is more common in multiple pregnancies and in multiparous women. It is registered in approximately 0.25-0.5% of all births, accompanied by a high level of fetal and newborn mortality (17-19%). The main danger is premature detachment of such a placenta, massive bleeding and death of the fetus, or severe intracranial trauma of the newborn during emergency extraction through an insufficiently dilated cervix. The placenta is often irregular in shape, flattened, windowed or with additional lobes.

Defects of detachment of the placenta.*Growth of the placenta*it is manifested by ingrowth of chorionic villi into the myometrium, difficulty in its separation and/or massive uterine bleeding, which sometimes requires extirpation of the uterus. The defect occurs as a result of insufficient development of the basal layer of the shell in the area of implantation of the ovum. Insufficient development of decidual tissue can be associated with endometritis, repeated scraping of the uterine cavity, etc.

*Premature exfoliation.*Detachment of the placenta, which occurs before the birth of the fetus, is called premature. Premature detachment can occur with defects in the development of the location of the placenta and a normally located placenta. It can also be a consequence of nephritis, hypertensive disease of pregnant women or abdominal trauma, short umbilical cord, late opening of the amniotic sac, rapid confluence of amniotic fluid with polyhydramnios.

Blood circulation disorders of the placenta

Diffuse ischemiaplacenta is observed in hemolytic disease in combination with edema, in posthemorrhagic conditions, as postmortem changes in connection with intrauterine death of the fetus. The decline of the capillaries of the terminal villi, the formation of syncytial buds is revealed. Diffuse hyperemia is observed in hypoxic conditions of the mother (diseases of the cardiovascular system), in the case of complications of blood flow through the umbilical vein - coiling of the umbilical cord, true knots of the umbilical cord, etc. Bleeding can be from the maternal part of the placenta during presentation or premature detachment of the placenta and from the fetal part in the form of hemorrhages into the stroma of the villi in nephropathy, infectious diseases of the mother, and into the amniotic fluid - hemamnion in case of rupture of the vessels of the umbilical cord.

Edema of the placentaobserved in hemolytic disease, infectious processes, diabetes and maternal nephropathy. The maternal surface of the placenta is pale, its mass is increased. Swelling of the stroma of the villi is accompanied by an increase in their volume by 2-3 times, in all such cases, a combination with immaturity of the villous tree is found, therefore, the swelling of the villi should be differentiated from

the presence of embryonic and intermediate immature villi with characteristic stromal channels and Kashchenko-Hoffbauer cells.

Thrombosis of the intervillous spaceoccurs with physiological aging of the placenta, with toxicosis of pregnant women, with infectious diseases of the mother. It is important to determine the age of occurrence of blood clots: fresh or old, with hemolysis of erythrocytes, fibrin deposition. Microscopically, this is the so-called red infarction of the placenta.

heart attack -the focus of necrosis of the villi, arising as a result of a violation of their nutrition with frequent disorders of the maternal blood flow, in particular in the spiral arteries of the uterus. Infarcts in the form of whitish-yellowish foci occur in small numbers with physiological aging of the placenta, large in volume - with diseases of the mother, which leads to vascular spasms, thrombosis (hypertensive disease, severe toxicosis of pregnancy, diabetes, etc.). Microscopically, complexes of necrotized villi surrounded by coagulated blood can be seen. A diagnostic sign of a long-standing heart attack can be considered the presence of clusters of syncytial buds, calcifications, and fibrinoid on the periphery of the necrotic zone. The volume of distribution of white infarcts is of great importance in the assessment of placental insufficiency. If it occupies more than 20-30% of the placental parenchyma,

Classification of placental insufficiency

The concept of "placental insufficiency or dysfunction" is interpreted inconsistently in the literature. Thus, E. Hovorka (1970) proposed to distinguish three types of placental insufficiency, depending on the pathogenesis of hypotrophy of newborns:

1) placenta in the case of primary deficiency of the body weight of the newborn, in cases of disorders of utero-placental blood circulation that are detected early, in case of chronic diseases of the mother (hypertensive disease, nephritis, etc.) with characteristic chronic heart attacks, intervillous thrombi in the hypoplastic organ;

2) placenta with secondary deficiency of the body weight of newborns - with late-onset blood supply disorders, most often in cases of carried-over pregnancy;

3) placenta in undifferentiated forms of newborn body weight deficiency, when signs of primary and secondary newborn body weight deficiency are simultaneously detected in the absence of hypertension, nephropathy and ongoing pregnancy.

Placental insufficiency is often defined as the inability of the placenta to maintain adequate exchange between the mother and the fetus, and therefore acute, subacute, chronic respiratory and chronic metabolic forms have been distinguished.

*Acute placental insufficiency*characterized by placental dysfunction that develops over several hours as a result of widespread hemorrhage or partial detachment. Histologically, retroplacental hematoma with collapse of the intervillous space, reactive hyperemia of fetal vessels, destruction of the epithelial cover of the villi against the background of immaturity of the villous tree, often of the type of chorioangiomatosis are determined. Most often, intrauterine death or acute asphyxia of the fetus occurs.

*Subacute placental insufficiency*develops over several days, causing intermittent placental dysfunction. By the nature of the lesion, this form is close to the previous one, but the areas of hemorrhage are small, they are characterized by thrombi in the intervillous spaces of different ages. Violation of utero-placental blood circulation is detected in the presence of villous immaturity, but detachment of the placenta does not occur. Intrauterine hypoxia and fetal hypotrophy develop.

*Chronic respiratory placental insufficiency*characterized by gas diffusion disturbances at the level of the placental barrier for weeks as a result, mainly, of the pathological immaturity of the villi, without pronounced disturbances of blood circulation in the placenta. Microscopically, small foci of necrosis, immature villi without syncytiocapillary membranes and syncytial buds are visible. A latent form of hypoxia develops in the fetus.

*Chronic metabolic placental insufficiency*are long-term (months) violations of the placenta's function with a compensatory increase in its mass, pathological immaturity of its villi, diffuse sclerosis of their stroma, hemorrhage and widespread heart attacks. Depending on the volume of the placenta lesion, intrauterine hypotrophy and hypoxia develop, or fetal death occurs.

In our country, primary and secondary placental insufficiency are distinguished, taking into account the duration of the pathological factors during pregnancy.

*Primary placental insufficiency*occurs during the period of egg implantation, placentation and early embryogenesis. Disturbances in the development of the mass, the shape, location, maturation, and vascularization, which are revealed in this case, lead to insufficiency of the placenta, the threat of termination of pregnancy and the death of the fetus during the first half of pregnancy.

*Secondary placental insufficiency*develops when the placenta has already formed as an organ. There are two forms of such deficiency: acute (disruption of maternal-placental blood circulation, hemorrhage, widespread heart attacks, etc.) and chronic, which occurs in late toxicosis, foci of latent infection, cardiovascular and renal diseases of the mother, etc. Under the influence of pathogenic factors on the immature placenta, the imperfection of compensatory reactions causes absolute placental insufficiency and intrauterine death of the fetus.

Pathology of the placenta in various diseases of the mother

Placenta in late toxicosis of pregnant women. The complex pathogenesis of toxicosis in pregnant women causes a variety of changes in the placenta. Among them, it is advisable to distinguish villus maturation disorders, common hemorrhagic heart attacks, immune disorders and compensatory and adaptive processes.

According to Z.P. Zhemkova, P.I. Topchieva (1973), out of 138 placentas of full-term newborns from mothers with late toxicosis of pregnancy (without other pathology), in 11.3 cases placental pathology of the type of maturita retardata and dissociated maturation disorder was found. In 43.9% of full-term fetuses that died in the antenatal period, similar ripening disorders were observed. A constant sign in all forms and degrees of pathological immaturity of the placenta is the insufficient

development of villi vessels, which indicates the early manifestations of the disease, which later manifest as late toxicosis of pregnant women. The same characteristic signs of toxicosis are multiple and widespread placental infarcts of various ages: the earlier the toxicosis develops and the more severe it is, the greater the number of chronic infarcts in the placenta

Placenta in hypertensive disease and chronic nephritis of pregnant women. The commonality of pathogenetic mechanisms of these diseases with toxicosis of pregnant women also explains the undeniable similarity of histological changes in the placenta. Therefore, some authors consider it impossible to differentiate the pathology of the placenta in these diseases and combine it into one group - the so-called toxemic placentas. They are also similar in the presence of typical complications: widespread heart attacks and premature detachment of a normally located placenta, which are based on changes in the spiral arteries of the uterus, which are easily damaged due to the lack of a sufficiently developed elastic framework in their wall. Changes in the form of plasmorrhagia, secondary lipoidosis and thrombosis, as well as fibrinoid lesions of the vessels of the fetal villi, destruction of the capillary endothelium dominate in the hypertensive disease of the mother. In fibrinoid lesions of placental vessels, the main role is played by intrauterine hypoxia, vasospasm,

Placenta with anemia in pregnant women. Iron-deficiency anemia in pregnant women is a frequent and common pathology that leads to many complications: with a mild degree of anemia, complications during childbirth make up 10%, with a severe degree - 70%. The placenta undergoes changes, mainly as a result of the deterioration of the oxygen supply of the mother's erythrocytes. With moderate and severe anemia in pregnant women, dyscirculatory, alternative and compensatory reactions are found in the placenta. Typical accumulations of maternal erythrocytes in the intervillous space, hemorrhage or white infarcts. In many terminal villi, dystrophy and desquamation of the syncytial cover, sclerosis of the stroma, a large number of immature villi with a two-layer structure of the syncytium, and a central arrangement of capillaries are observed. Compensatory and adaptive mechanisms are mainly manifested by angiomatosis of immature villi, an increase in the number of terminal villi, the presence of syncytial buds. At the same time, as the severity of anemia increases, the area of the syncytial cover decreases. It is important to emphasize that newborns from mothers suffering from iron-deficiency anemia are less adaptive in the first hours and days of extrauterine life.

Placenta in pregnant women with diabetes. When pregnant women have diabetes, there is variability in the mass and histological structure of the placenta, which is mainly explained by the degree of severity of the mother's main disease and the term of pregnancy. At the same time, E. Govorka singles out three variants of the placenta in terms of mass: excessively large, medium, very small.

An excessively large placenta (550-800 g) is observed during full-term pregnancy in mothers whose diabetes began around the age of 20, lasted no more than 10 years, and was not accompanied by vascular complications (micro- and macroangiopathy, etc.). Histologically, such a placenta most often corresponds to the variant of pathological immaturity - the type of embryonic villi. Placental tissue is dominated by large, multilobed villi with a two-layer syncytium, a loose stroma with characteristic

channels containing Kashchenko-Hoffbauer cells, and narrow, centrally located capillaries. Nuclear forms of fetal erythrocytes are sometimes visible in their lumen. Thickening of the walls of arterioles in the supporting villi and in the composition of the chorionic plate is also common. They also describe severe changes in the spiral arteries of the uterus with expansion of their subendothelial zone due to the formation of fibroblasts and fibrin deposits, similar to diabetic angiopathy of other localizations. The body weight of the child reaches, as a rule, 5000-6000 g.

The average placenta (400-500 g) is found in full-term pregnancy in mothers suffering from diabetes in compensated forms. The structure of such a placenta corresponds to the dissociated variant of pathological immaturity with a characteristic alternation of mature and immature cotyledons. Along with the observations described above, there are also terminal villi. The prognosis for the newborn is good, the body weight of the newborn does not exceed 3700-4500 g.

A very small placenta (less than 300 g) is observed in premature pregnancies of 28-30 weeks in mothers who suffered from juvenile diabetes with a disease history of more than 20 years. A very characteristic combination with late toxicosis of pregnant women, and the histological picture of small placentas resembles that of toxicosis. Changes in the wall of the arterial vessels of the chorionic plate and umbilical cord, as well as the spiral arteries of the uterus in the form of plasmatic impregnation, sclerosis, fibrinoid necrosis, damage to the endothelium, and proliferation of myofibroblasts of the subintimal layer prevail.

Placenta in isoimmune conflict between mother and fetus. In this pathology, the placenta has large dimensions, its weight is 450-600 g. There are cotyledons of various sizes, separated by deep furrows; the surface of the fruit is pale yellow, the parenchyma is loose, swollen, and poorly drained. Swelling and yellowish color are also found in the fruit membranes and in the thickened umbilical cord. The histological picture of such a placenta corresponds to that of large placentas with maternal diabetes.

Litter inflammation

Infectious damage to the litter is important in perinatal pathology, as it can lead to the death of the fetus or to the disease of the newborn. There are inflammations of: intervillous space - intervillusitis; villi - villousite; basal plate - basal deciduit; chorionic plate - chorioamnionitis. Occasionally, the entire placenta is affected - spilled placentitis. Accordingly, inflammation of the umbilical cord - funiculitis, fetal membranes - parietal amnionitis.

The etiology of litter inflammation is related to viruses, plasmodia, protozoa, fungi, bacteria, as well as chemical irritants - meconium, its proteolytic enzymes, changes in the pH of amniotic fluid, etc. However, not every inflammation of the litter is accompanied by infection of the fetus, and, in addition, infection of the fetus, for example, with some viral infections, can occur without inflammation of the litter. The ways of spreading the infection can be different. Most often, an ascending route of infection is observed with early water withdrawal and a long dry period; hematogenous infection from the mother's blood through the spiral arteries into the intervillous space or during the transition of the process to the chorionic villi is less common.

Morphological diagnosis of placentitis differs in a number of features.

*Firstly,*the inflammatory reaction is expressed moderately, in particular, leukocyte infiltration. Leukocytes can come from the blood of the mother - in the basal plate, intervillous space, or from the blood of the fetus - in the capillaries of the villi, in the umbilical vessels, or be of mixed origin. The assessment of leukocyte infiltration requires some caution, since aseptic accumulations of leukocytes in the umbilical cord and chorionic plate (of fetal origin) are formed during prolonged labor accompanied by intrauterine hypoxia of the fetus. They are found in the droppings of dead fetuses that suffered from oxygen starvation for a long time, as well as in live children born asphyxiated. In fact, this is a peculiar reaction of rejection of the placenta by the maternal tissue - the uterus. In controversial cases, the decisive argument in the diagnosis is a virological or bacteriological examination of placenta tissue,

*Second,*by accompanying signs of delayed development of the villous tree, one can judge the early effect of an infectious agent, for example, in toxoplasmosis, listeriosis, syphilis, etc.

*B-third,*with an infectious lesion of the litter, circulatory disorders, alteration and productive changes of the epithelium of the villi and fruit membranes often dominate.

*B-fourths,*the use of relevant methods: immunofluorescence, bacterioscopy, detection of viral inclusions provides significant help in the etiological diagnosis of litter lesions.

The most common type of placentitis are viral and mycoplasma lesions, which are clinically manifested as SARS during pregnancy. Changes similar to those found in the respiratory organs of a fetus or newborn are found in the placenta.

*With mycoplasma infection*characteristic hypertrophy of syncytiotrophoblast villi with vacuolization of their cytoplasm and the presence of inclusions in vacuoles; by the immunofluorescent method, mycoplasmas are also detected in the cells of the stroma of the villi, in the basal plate. In the intervillous space and the basal lamina, lymphoid infiltrates with an admixture of leukocytes are constantly observed, while their presence is rarely found in the chorionic plate and stroma of the villi. The prognosis for the life of the fetus depends on the prevalence of inflammatory and alternative changes in the tissue of the placenta.

*With hermetic and adenoviral infection in*in the tissue of the placenta, cells with large, hyperchromic nuclei appear in the chorionic plate, in the epithelium of the villi, in the basal plate, and in the cells of the septa. With cytomegaly in the placenta, typical cytomegaloviruses are found in the stroma of the villi; foci of inflammation do not have clear boundaries, are more often located under the syncytial cover of villi, larger ones occupy the entire stroma of individual villi.

*When the placenta is damaged by RNA viruses,*in addition to the proliferation of syncytiotrophoblast villi in para-influenza and MS infection, the formation of papillary structures in the epithelium of the amnion and fetal membranes is characteristic. Small foci of acidophilic necrosis, areas of disorganization of the stroma of villi and vessel walls are observed during influenza. Fuchsinophilic inclusions, often cytoplasmic, rarely intranuclear, are constantly detected in smears-scrapes from the amnion, villous chorion, and basal plate. Lymphoid infiltrates with an admixture of leukocytes in the

composition of the chorionic plate are constantly detected, as well as swelling of the endothelium, proliferation of cells of all layers and narrowing of the lumen of fetal vessels, although such pathology of the endothelium is hardly specific.

In 1980, SH Sander described a peculiar hemorrhagic endovasculitis of the placenta, in 43 out of 70 such observations, the pregnancy ended in stillbirth, in 15 out of 28 live-born children, distress syndrome developed or there was a lag behind the gestational period. Hyperplasia of the inner and middle membranes of fetal vessels with narrowing of the lumen and thrombosis, as well as erythrocyte fragments, diapedesis of fragments and whole erythrocytes, deposition of hemosiderin in the stroma of the villi are characteristic. The appearance of nuclear forms of erythrocytes in the vessels of the villi was noted. The nature of vascular lesions and intranuclear inclusions suggest a viral origin of the disease.

The most favorable prognostic factors for the fetus and the newborn are combined viral-mycoplasma-bacterial lesions of the placenta, proceeding according to the type of basal deciduit or spilled placentitis.

*For purulent bacterial infection*characteristic serous-purulent, purulent inflammation, sometimes with the development of phlegmon or abscesses.

In case of listeriosis, yellowish-gray foci of necrosis with histioleukocyte infiltration on the periphery are found, granulomas are found in the composition of the chorionic plate, in Warton's coldness of the cord and vessels. Listerella are clearly visible on semi-thin sections of the villi and in the basal plate.

In tuberculosis, foci of caseosis, nodules with epithelioid and giant cells are observed, the basal plate is more often affected. Changes in the placenta in congenital syphilis are characterized by swelling or fibrosis of the stroma in the terminal and stem villi, focal polymorphic cellular infiltrates with or without necrosis inside the villi. Mesenchymal cells and Kashchenko-Hofbauer cells are part of the infiltrates. The diagnosis is clarified when spirochetes are detected in the tissue of the placenta and with the help of serological tests of the mother and fetus.

With toxoplasmosis, cysts, pseudocysts and free parasites are found in the area of widespread necrosis with calcifications of the placenta tissue.

In case of candidiasis, inflammatory infiltrates consist of polymorphonuclear leukocytes and mononuclear cells. Many fungi are usually found, more often in the chorionic plate, fruit membranes.

In malaria, the causative agent is detected in large quantities in the intervillous space and in the vessels of the decidual membrane, as well as in the erythrocytes of the mother, and malaria pigment deposits are found in the tissues.

Tumors of the placenta

True tumors of the placenta are represented by hemangiomas, angiofibromas and occasionally teratomas. Placental hemangiomas occur relatively often, approximately 1 case per 100 births. Their sizes vary from microscopic nodules to large foci resembling hematomas or heart attacks. Cavernous or capillary forms of hemangiomas are usually diagnosed histologically. They should be differentiated from the option of

pathological immaturity - chorioangiomatosis of the placenta and compensatory angiomatosis of the villi. Often, angiomas have the character of angiomyxoma or angiofibroma. Large chorioangiomas are often combined with polyhydramnios, disorders of fetal development. Intrauterine mortality in widespread placental hemangiomas reaches 8-25%, and concomitant polyhydramnios leads to impaired fetal kidney function, sometimes to edema and hypertrophy of the heart.

Quite large teratomas of the placenta with various tissue components of all three germ layers are occasionally observed. It is assumed that such teratomas are the so-called amorphous fetus in multiple pregnancy. Sometimes metastatic nodes are found in the placenta: melanoblastoma of the mother, various forms of cancer. There were cases of congenital leukemia with marked leukemic infiltration of the stroma of the villi, but without the transition of leukemic cells into the maternal vascular bed.

Pathology of the umbilical cord and fetal membranes

Anomalies of the length of the umbilical cord. In perinatal pathology, both shortening and excessive lengthening of the umbilical cord are important. At 34-42 weeks of pregnancy, the length of the umbilical cord increases from 53 to 57-60 cm, this parameter is closely correlated with the length of the fetus.

An umbilical cord 40 cm long or less is considered short. A rare syndrome of umbilical cord insufficiency is known - aplasia or a rare shortening of the cord up to 8 cm. Such a case is characterized by a combination with underdevelopment of the anterior abdominal wall and internal organs, therefore this syndrome is more often called "eventeration", "umbilical-fetal dysplasia". The development of the spine, limbs, lungs, heart, and genitourinary system of the fetus is repeatedly disrupted. Although the time of fetal damage is established (3rd week of pregnancy), the cause of umbilical cord aplasia is unknown. Fetuses die around 15-25 weeks of pregnancy. Shortening of umbilical cords from 10 to 20 cm in 60 cases is accompanied by premature birth, in 36% - the birth of dead fetuses, with the length of the umbilical cord of 25-35 cm, such complications are less - 32% and 14%, respectively.

Excessive elongation of the umbilical cord (more than 62 cm) is sometimes found during pregnancy that is transferred, but it has no serious thanatogenic significance.

Umbilical cord cysts. There are false cysts in Varton's cold, up to 1-1.5 cm in size; most often they are found in cords with twists in dead fetuses, but they also occur in full-term newborns. True cysts are formed from the remains of the yolk or allantoic duct. Cysts of the yolk duct have a typical localization - in the triangle between the vessels of the umbilical cord. They usually have microscopic dimensions and are lined with cuboidal epithelium. Cysts of the allantoic duct consist of a flat epithelium, a connective tissue membrane, and a concentric layer of Warton jelly. Occasionally, tumors are found in the cord: teratoblastoma, etc.

Forms of compression of the umbilical cord. There are prolapse, entanglement, entanglement, knots and clamping of the umbilical cord.

*Prolapse of the umbilical cord*closely related to premature rupture of the fetal membranes and occurs most often before or during childbirth. Fetal tachycardia and

bradycardia develop. If this formidable complication is not diagnosed in time, the fetus dies intranatally as a result of asphyxiation.

*Convolutions or true knots*umbilical cords are detected in the presence of a small fetus, a long umbilical cord and polyhydramnios. Such complications occur in 0.4-0.5% of all births. The timing of the formation of umbilical cord knots is difficult to determine, since during pregnancy the knots usually do not tighten due to blood pressure and pulsation of the umbilical cord vessels. The entanglement of the umbilical cord and the formation of its true knots represent a danger in childbirth, when their tightening leads to the death of the fetus. Serious difficulties arise when assessing the thanatogenic role of cord twisting in stillbirths. It is believed that the signs of intrauterine acute twisting of the umbilical cord are compression or obliteration of the umbilical vein and the presence of strangulations on the body of the fetus.

It is known that the umbilical cord is clamped by Simonart's amniotic cords - amniotic cord syndrome. S. Heifetz, analyzing 6 of his own observations and 57 cases described in the literature, singled out a triad of signs: separation of the amnion from the placenta, adhesions between the fetus and the remains of the amnion, as well as deformations or severe defects in the development of the fetus. In 58 observations, fetuses died in the antenatal period, in 3 - in the intranatal period, and 2 newborns died during the first week.

Abnormalities of attachment of the umbilical cord. The most clinical significance is the marginal u membrane attachment of the umbilical cord. It should be emphasized that these placentation disorders often accompany variants of placental insufficiency. Based on the analysis of 1000 placentas in singleton pregnancies, P. Uyanwah-Akrot, H. Fox (1979) concluded that marginal and membrane attachment of the umbilical cord has a pathogenetic connection with an increase in the frequency of miscarriages, malformations, fetal hypoxia, intrauterine death, prematurity and etc.

Aplasia of one of the umbilical arteries. This disorder is a rare but serious malformation of the umbilical cord; diagnosed by the absence or obliteration of one of the two arteries on the section of the cord. Its attachment is atypical - marginal or membrane. Lobular placenta: in 21% of cases - very small (100 g less than the gestational norm), in 18.6 - surrounded by a shaft, in 32.6% - with the presence of heart attacks. B 80-90% of observations reveal severe defects: fetuses without a heart, Down's disease, malformations of the genitourinary organs, etc. With aplasia of the umbilical artery, the number of premature babies increases, perinatal mortality increases to 16.5%, chromosomal disorders are not uncommon, in particular, trisomy of the 18th pair of chromosomes.

Pathology of fruit membranes. Premature rupture of the fetal membranes, which can occur starting from the 28th week of pregnancy, has the greatest clinical significance. Early rupture of membranes increases the frequency of pre- and neonatal infections. Pathology of the fetus is caused most often by the accompanying loss of the umbilical cord.

Polyhydramnios is a frequent symptom of late toxicosis of pregnant women, placental transfusion in multiple pregnancies. At 30-37 weeks of pregnancy, the volume of amniotic fluid is 450-500 ml, before childbirth - 600 ml. An increase in the amount of water up to 2 liters is more often combined with fetopathy - hemolytic disease, diabetic fetopathy, sometimes with embryopathy.

low tide -a decrease in the amount of amniotic fluid to 500 ml or less - is combined with hypoplasia of the fetus and placenta and with embryopathies. The etiology and pathogenesis of polyhydramnios and hypohydramnios have not been established.

*Amniotic adhesions (Simonart's cords)*are dense connective tissue hyalinized cords or threads that go from the amnion to the surface of the fetus. In full-term fetuses, they cause the formation of furrows or amputation of fingers, toes, forearms, lower legs, shoulders, and thighs. They are less often attached to the body. Embryos are allowed to have a teratogenic effect of cords with the development of hypoplasia or malformations of the limbs. They are especially common in low water conditions.

Rare defects include an incomplete amnion, as a result of which the embryo is partially located outside the amniotic cavity, which is accompanied by its fusion with the chorion and severe developmental defects.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Gestosis, morphological characteristics, consequences.
2. Pathology of the placenta, classification.
3. Pathology of the placenta in various diseases of the mother, morphological characteristics, consequences.
4. Ectopic pregnancy, classification, consequences.
5. Trophoblastic disease, morphology, consequences, causes of death.
6. Litter inflammation, morphological changes, consequences.
7. Birth injury, types, consequences.
8. Pathology of the umbilical cord and fetal membranes, types, morphological changes, consequences.
9. Pathology of fruit membranes, morphological changes, consequences.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

The newborn did not take his first breath. During the pathological autopsy of the body, it was established that the lungs did not expand with free airways. Which of the following could be the reason for this?

Correct answer Absence of surfactant
BNarrowing of the bronchi
C Rupture of the bronchi
DThickening of the pleura
An increase in the size of the alveoli
At the autopsy of the stillborn, a decrease in the volume of the skull, thickening of the bones of the skull, and a decrease in the mass and volume of the brain were found. What congenital brain defect should be considered?
Correct answer Microcephaly
Anencephaly
Porencephaly
Hydrocephalus
Meningocele
During the autopsy of the brain of the stillborn, cysts of various sizes were found, communicating with the lateral ventricles of the brain. What birth defect should you think about?
Correct answer Porencephaly
BMicrocephaly
Hydrocephalus
Meningoencephalocele
Encephalocystocele
An autopsy of the heart of a stillborn revealed a defect of the interventricular septum, stenosis of the pulmonary artery, and hypertrophy of the wall of the right ventricle of the heart. What kind of combined congenital heart disease should you think about?
Correct answer Triad of Fallot
Tetrad of Fallot
Fallot's pentad
DDefect of the atrial septum
Stenosis of the pulmonary artery
A macroscopic examination of the liver of a stillborn revealed multiple cysts of various sizes filled with a clear liquid. What congenital liver disease should be considered?
Correct answer Polycystic liver
Agenesis of intrahepatic bile ducts
CHypoplasia of the intrahepatic bile ducts
Hyperplasia of intrahepatic bile ducts
Stenosis of the intrahepatic bile ducts
The autopsy of the stillborn revealed the absence of one kidney. To what type of congenital kidney defects should this case be classified?
Correct answer Agenesis
BHypoplasia
Dysplasia
D Adhesion of kidneys
ELarge cystic kidneys

A macroscopic examination of the kidneys of a stillborn revealed their significant increase, a large number of large cysts with transparent contents in the cortical layer. Cysts were also found in the tissue of the liver and pancreas. To what type of congenital kidney defects should this case be classified?
Correct answer Large cystic kidneys
BKidney dysplasia
C Kidney hypoplasia
DSmall cystic kidneys
E Kidney enlargement
There is a limited hemorrhage in the cavity of the parietal bones of the infant's skull under the periosteum. Diagnose the process:
Correct answer External cephalohematoma
Obstetric tumor of the fetal head
Epidural hemorrhage
Phlegmon of the soft tissues of the head
ESubdural hemorrhage
In the thickness of the parietal lobe of the left hemisphere of the brain of the deceased infant, a grayish-dirty area of mushy consistency 1x2 cm with indistinct borders was found. What pathological process has developed?
Correct answer Ischemic heart attack
BHemorrhagic heart attack
Bleeding in the tissue of the brain
D Cephalohematoma
Epidural hematoma
During the autopsy of the brain of the dead baby, blood was found in the cavity of the lateral ventricles. Name the cause of death.
Correct answer Intraventricular hemorrhage
Epidural hemorrhage
CSubdural hemorrhage
DSubependymal hemorrhage
ELeptomeningeal hemorrhage
Macroscopic examination of the deceased infant revealed hemorrhages in the lung tissue, pleura, subcapsular hematomas in the liver, adrenal glands, kidneys, and gastrointestinal tract. What disease should you think about?
Correct answer Hemorrhagic disease of infants
BHemolytic disease of infants
CValid ground
D Pulmonary infarction
E Kidney hematoma

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

28. methods: assessment of the correctness of the performance of practical skills
29. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .

"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Final lesson.(Subsections Diseases of the respiratory organs. Diseases of the digestive organs. Diseases of the endocrine system. Diseases of the urogenital system. Diseases of the musculoskeletal system. Diseases of pregnancy and the postpartum period. Diseases of the pre- and perinatal period. Pathomorphology of hypo- and vitamin deficiency. caused by human activity and the influence of the external environment) Practical skills".

Suggested topics for essays:

1. Trophoblastic disease. Morphological characteristics.
2.Birth trauma, types, morthological characteristic.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization

4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 33

Topic: Final lesson.(Subsections Diseases of the respiratory organs. Diseases of the digestive organs. Diseases of the urogenital system. Diseases of the endocrine system. Diseases of the musculoskeletal system. Diseases of pregnancy and the postpartum period. Diseases of the pre- and perinatal period. Pathomorphology of hypo- and vitamin deficiency. caused by human activity and the influence of the external environment). Practical experience.

Goal:to test the ability to describe micro and macro drugs with a grant of the conclusion of the pathological process according to the topics of the final control.

Basic concepts:see above according to the topics of the final control.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

see above according to the topics of the final control.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Describe the micropreparation, draw a conclusion.
2. Describe the macropreparation, draw a conclusion.

3. Definition of this pathological process.
4. Complications and causes of death of this pathological process.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

see above according to the topics of the final control.

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

30. methods: assessment of the correctness of the performance of practical skills
31. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "General concepts of human infectious pathology. Classification of infectious diseases. Intestinal infectious diseases.".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 34

Topic: General concepts of human infectious pathology. Classification of infectious diseases. Intestinal infectious diseases.

Goal: To be able to determine the signs of the general infectious process from the macro- and microscopic picture. Morphological variants of local and general reactions in infections. Learn to determine the etiology, pathogenesis, morphology of intestinal infections, as well as their complications, causes of death.

Basic concepts: General characteristics of the infectious process. Classification of infectious diseases. Intestinal infectious diseases, klassification,morphological characteristics, complications, result.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Infectious diseases can occur in the presence of three components:
• sources of pathogens (infected person or animal);
• factors that ensure the transmission of pathogens from an infected organism to a healthy one;
• people susceptible to infection.
The penetration and activation of the pathogen in the body is an infectious disease.
Infectious agents can be classified according to the complexity of their structure:
- prions
- viruses
- rickettsiae
- chlamydia
- mycoplasmas
- bacteria
- mushrooms
- Simpler
- helminths.

Infectious agents damage tissues in three ways:
• they can come into contact with or penetrate host cells and directly cause cell death;
• they can secrete endo- or exotoxins, which lead to the death of cells located at a distance; secrete enzymes that damage blood vessels and indirectly cause ischemic necrosis;
• they stimulate the development of immune cellular and humoral reactions directed against the infectious agent, but capable of causing additional tissue damage.
Tissue changes during infections
A. Cell necrosis. B. Dystrophic cell changes. B. Formation of intracellular inclusions. D. Formation of giant cells.
Typhoid fever is an acute infectious disease, a classic intestinal infection, a typical anthroponosis.
Stages:The first stage is characterized by brain-like swelling of group follicles, furrows and convolutions are formed on their surface, resembling the surface of the brain. The second stage is necrosis of group follicles, which is based on necrosis of typhoid granulomas. The third stage is the formation of ulcers as a result of sequestration and rejection of necrotic masses. The fourth stage is the stage of clean ulcers. The fifth stage is the healing of ulcers and the appearance of scars in their place.
Complications: Among intestinal complications, intestinal bleeding and ulcers are the most frequent and dangerous. Bleeding occurs in the third week and can be fatal. The

ulcer leads to peritonitis. The reason for this can be necrotic changes in the mesenteric lymph nodes and rupture of the spleen capsule.
Among the extraintestinal complications, the most important are: pneumonia, purulent inflammation of the larynx, waxy necrosis of rectus abdominis muscles, osteomyelitis, intramuscular abscesses.
Pneumonia is caused by pneumococcus, staphylococcus.
Purulent perichondritis of the larynx with the development of bedsores near the entrance to the esophagus occurs in weakened patients. Waxy necrosis of the rectus abdominis muscles is quite common. Purulent osteomyelitis and intramuscular abscesses are late complications. Death of patients occurs from complications.
Dysentery (shigellosis) is an acute infectious disease caused by a group of microbes from the genus Shigella. It is characterized by an inflammatory process in the large intestine.
Stages: The first stage is acute catarrhal inflammation; the second stage is necrosis; the third stage - the formation of ulcers; the fourth stage is the stage of regeneration and scarring.
Complication.
1. microperforation of ulcers with the development of focal or diffuse peritonitis. This can cause the formation of an abscess or phlegmon of pelvic tissue - phlegmonous paraproctitis. Paraproctitis does not heal for a long time, with the formation of fistulas. With dysentery, there are no perforations visible to the eye.
2. Narrowing - stricture of the rectum on the basis of deep scarring of ulcers. The scarring process can spread to the surrounding tissue with the appearance of periproctitis. Rectal prolapse is possible in children.
3. Relapses of necrotic-ulcerative processes of the intestinal wall with the transition of the disease into a chronic form.
4. Bleeding from dysenteric ulcers is very rare.
Extraintestinal complications.
1. Focal pneumonia. Pneumonia and lung abscesses occur in weakened patients. There are such pneumonias with dysentery and in small children.
2. In children, as a complication - purulent otitis, followed by mastoiditis. These purulent processes, in turn, can lead to pyome.
3. Metastatic liver abscesses. With amoebic dysentery, they occur more often. Thrombophlebitis of the branches of the portal vein and, against this background, a liver abscess.
4. The result of dysenteric intoxication is serous arthritis and synovitis.
5. Exhaustion of the body, dropsy of the serous cavities, edema of the tissue. Nephrosis.
6. Development of inflammatory amyloidosis - in chronic form of dysentery.

2.3. List of questions to check basic knowledge on the subject of the lesson.
1. General characteristics of the infectious process, classification.

2. Definition of the primary infectious complex, morphological characteristics.
3. Classification of the infectious agent.
4. Local changes in infectious diseases, morphological characteristics.
5. General changes in infectious diseases, morphological characteristics.
6. Typhoid fever, morphological characteristics, complications, causes of death.
7. Salmonellosis, morphological characteristics, complications, causes of death.
8. Dysentery, morphological characteristics, complications, causes of death.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

During the autopsy of the deceased, the soft brain membranes are dull, on their surface there are layers of greenish-yellow color, which capture almost the entire convex surface of the cerebral hemispheres. During the histological examination, a sharp fullness of the meninges with diffuse leukocyte infiltration is observed. Which of the diseases listed below is the most likely?

The correct answer is Men Ingococcal infection

B Kand

C Sibirka

D Tuberculosis

E Flu

An autopsy of an elderly man who had suffered from an acute intestinal disorder for the past 2 weeks revealed changes in the rectum and sigmoid colon: a brownish-green film was noted on the surface of the mucous membrane. The wall of the intestine is thickened, the cavity is sharply narrowed. Microscopically, necrosis of the mucous membrane penetrating to different depths is revealed, necrotic masses are penetrated by fibrin threads, with leukocyte infiltration. What is the most likely diagnosis?

The correct answer is F Ibrinous colitis

B Catarrhal colit

Ulcerative colitis

Follicular colitis

E-

The autopsy of a man who died on the 5th day of typhoid fever revealed the following changes: the group follicles of the ileum are enlarged, filled with blood and protrude above the mucous membrane, furrows and convolutions are visible on their surface. Histologically: full blood and swelling of the tissue, the presence of granulomas, which consist of large cells with light cytoplasm and contain typhoid bacilli. What period of local changes in typhoid fever can we think about?

Correct answer The stage of cerebral swelling

Stage of necrosis

Stage of ulcer healing
Stage of clean ulcers
Stage of formation of ulcers
In typhoid fever, necrotized Peyer's patches of the small intestine are colored yellow-brown. What pigment permeates the necrotic tissue?
Correct answer Bilirubin
Lipofuscin
Hemoglobin
DIndol
EMelanin
At the autopsy of the deceased, 67 years old, signs of fibrinous inflammation were found in the large intestine. Your possible diagnosis:
Correct answer Dysentery
Balantidiasis
Typhoid fever
Amebiasis
Cholera
The mucous membrane of the large intestine in a person who died of dysentery at autopsy is full of blood, covered with a gray film that comes off with effort. What type of inflammation developed in the intestines of the patient?
Correct answer Diphtheritic inflammation
BHemorrhagic inflammation
Catarrhal inflammation
D Serous inflammation
ECrupose inflammation
Patient K., 16 years old, died as a result of disseminated (disseminated) fibrinous-purulent peritonitis. At autopsy, in the lower part of the small intestine, an ulcer was found, repeating the shape of a Peyer's plaque, with perforation of the intestinal wall. Microscopic examination revealed the effacement of the pattern of lymphoid tissue, its displacement by proliferating monocytes forming granulomas. What complication of the disease was the cause of death?
Correct answer Typhoid fever
Brucellosis
Non-specific ulcerative colitis
Dysentery
Cholera
At the autopsy of the corpse of a person suffering from typhoid fever, changes were found in the small intestine: group lymphoid follicles are enlarged, protruding above the surface of the mucous membrane, gray-red, juicy, the surface has the appearance of convolutions and furrows. Microscopic examination reveals the formation of typhoid granulomas. Indicate which of the listed stages of typhoid fever is most probable?
Correct answer Cerebral edema
B Necrosis

CClean ulcers
DHealing
Ulcer formation
The uterus removed during the operation was delivered for histological examination. Under the mucous membrane, multiple nodes of a rounded shape, clearly separated from the surrounding tissue, were found. Microscopically, the tumor consists of bundles of smooth muscles with tissue atypism. Your diagnosis?
Correct answer Leiomyoma
BFibromyoma
C Leiomyosarcoma
Chorionepithelioma
Uterine cancer
During the autopsy of a patient who died of widespread peritonitis, multiple oval-shaped ulcers located along the intestine were found in the distal parts of the small intestine. The bottom of the ulcers is clean, smooth, formed by a muscular or serous membrane, the edges of the ulcers are even, rounded. Two ulcers have perforating holes with a diameter of up to 0.5 cm. What disease should be suspected?
Correct answer Typhoid
Cholera
Salmonellosis
D Tuberculosis
E Dysentery
At the autopsy of the deceased L., 62 years old, signs of hypovolemic shock were found. Violated skin turgor, pronounced symptoms of "washing hands", "gladiator's pose". In the lumen of the intestine, there is a small amount of "rice broth" type liquid. What disease led to the patient's death?
Correct answer Cholera
B Typhoid fever
Salmonellosis
D Tuberculosis
E Dysentery
During the autopsy of the corpse of a 50-year-old man, several ulcers measuring 4 to 6 cm in diameter were found in the terminal part of the small intestine. The edges of the ulcers moderately rise above the surface of the mucous membrane, the walls of the ulcers are covered with crumbling grayish-yellow masses. Which of the complications of typhoid fever are most common in this stage of the disease?
Correct answer Perforation of the ulcer
BParaproctitis
CIntestinal bleeding
Peritonitis
Penetration of ulcer

At the autopsy of the patient P., who died of spilled peritonitis, a perforation of the intestinal wall was found in the region of the transition of the small intestine into the large intestine. What infectious disease caused peritonitis?
Correct answer Typhoid
Cholera
Salmonellosis
D Tuberculosis
E Dysentery
On the autopsy of the body of a man suffering from typhoid fever, defects were found in the ileum, which are located along the length of the intestine, their edges are even, the bottom is formed by a muscle layer. Which of the stages of typhoid fever is diagnosed?
Correct answer Clean ulcers
BHealing
Formation of ulcers
D Cerebral swelling
ENecrosis
At the autopsy of the body of a man who died from intra-intestinal bleeding, necrosis of group and solitary follicles, imbibition of dead tissues with bile and blood is observed in the ileum; in the lower part of the intestine - phenomena of sequestration and rejection of necrotic masses with the formation of defects. Which of the listed diagnoses is the most likely?
Correct answer Typhoid fever, stage of "dirty" ulcers
B Typhoid fever, stage of "pure" ulcers
Typhoid fever, stage of necrosis
Typhoid form of salmonellosis
Crohn's disease
A 71-year-old man had diarrhea with mucus and blood in his stool for 10 days. The patient was hospitalized in serious condition, died 2 days later. The autopsy of the deceased revealed: diphtheritic colitis with multiple ulcers of irregular shape of different depths in the sigmoid and rectum. Bacteriological examination revealed Shigella. What is the patient's main disease?
Correct answer Dysentery
B Typhoid fever
Salmonellosis
DNonspecific ulcerative colitis
Yersiniosis
As a result of heart failure, the deceased has traces of a rash in the form of spots and dots on the skin. In the region of the sacrum, spinous processes of the vertebrae - bedsores. Microscopic examination of the central nervous system: destructive-proliferative endothrombovasculitis with the presence of Popov's granuloma in the vessels of the microcirculatory bed and small arteries, interstitial myocarditis in the heart. Which of the listed diagnoses is the most likely?

Correct answer Typhus
B Typhoid fever
CKu-fever
D Nodular periarteritis
EVIL infection
During the autopsy of the body of a 9-year-old child, numerous irregularly shaped defects of different depths with uneven edges, as well as gray-white films, which are tightly soldered to the underlying tissues, were found in the rectal mucosa. What disease should we think about?
Correct answer Dysentery
B Salmonellosis
Cholera
Typhoid fever
EAmoebiasis

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

32. methods: assessment of the correctness of the performance of practical skills

33. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Viral airborne infections.Corona virus disease. HIV infection and AIDS. Rabies. Childhood infections.".

Suggested topics for essays:

1.Yersiniosis. Morphological characteristics.

2.Campylobacteriosis.Morphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 35

Topic: Viral airborne infections.Corona virus disease. HIV infection and AIDS. Rabies.

Goal:to study clinical and morphological manifestations of viral infections and coronavirus disease.; interpret morphogenesis, clinical and morphological manifestations, complications, causes of death in viral diseases, AIDS, rickettsiosis; morphological characteristics of rickettsioses; causes of death in rabies; morphological characteristics of airborne viral and prion infections; interpret morphogenesis.

Basic concepts: General characteristics of viral airborne infections.Corona virus diseases, morphological characteristics, complications, causes of death. AIDS, rickettsioses; morphological characteristics, complications, causes of death.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Features of viruses:
1. Intracellular parasitism.
2. Fabric tropism.
3. Latent mode of existence.
4. Resistance of viruses to leukocytes.

Eye infection
I. Incubation stage.
II. Stage of primary manifestations:
A - acute febrile phase;
B - asymptomatic phase;
B - persistent generalized lymphadenopathy.
III. Stage of secondary diseases:
A - loss of body weight less than 10%, superficial fungal, bacterial, viral lesions of the skin and mucous membranes, shingles, repeated pharyngitis, sinusitis;
B - progressive loss of body weight more than 10%, unexplained diarrhea or flatulence for more than 1 month, hairy leukoplakia, pulmonary tuberculosis, repeated or persistent bacterial, fungal, viral, protozoal lesions of internal organs (without dissemination) or deep lesions of the skin and mucous membranes, repeated or disseminated shingles, localized Kaposi's sarcoma;
B - generalized bacterial, viral, fungal, protozoan and parasitic diseases, pneumocystis pneumonia, esophageal candidiasis, atypical mycobacteriosis, extrapulmonary tuberculosis, cachexia, disseminated Kaposi's sarcoma, CNS lesions of various etiologies.
IV. Terminal stage.
There are also three immunological categories depending on the level of CD4 lymphocytes:
1) more than 0.5 x 109 / l CD4 cells in 1 mm 3 of blood;
2) from 0.2 to 0.5 x 109 / l in 1 mm 3;
3) less than 0.2 x 109 / l in 1 mm 3.

rickettsioses- a group of infections of humans, many warm-blooded animals and some arthropods, which are intracellular parasites.
Reservoir of rickettsial infections in nature - wild and domestic animals, they are a source of infection of arthropod bloodsuckers - fleas, ticks.
People develop diseases with characteristic rashes on the skin and peculiar lesions of blood vessels in the form of vasculitis and thrombovasculitis of varying severity. Infection occurs through damaged skin, into which the causative agents of lice and fleas are rubbed with feces.
Classification of rickettsioses (6 groups):
1) typhus group, which includes epidemic typhus transmitted by lice, and endemic or rat typhus transmitted by fleas.
2) a group of tick-borne spotted fevers (rocky mountain spotted fever, Marseilles, North Australian tick-borne typhus and North Asian tick-borne typhus).
3) a group of endemic fevers, the causative agents of which are transmitted by tick larvae (tsutsugamushi fever).
4) group of pneumotropic rickettsioses or Ku-fever group.
5) a group of paroxysmal rickettsioses (Volyn or trench fever).
6) group of animal rickettsioses.
These rickettsioses are characterized by:
1) severe vascular pathology;
2) febrile condition;
3) rash.
Elementary forms of damage to vessels are:
1) warty endovasculitis (destruction of necrotized endothelium, formation of wall coagulation thrombus in the form of a wart);
2) proliferation of endothelial and intimal cells;
3) necrosis of the entire thickness of the vessel wall, when all three membranes are necrotized. The vessel shrinks, obturates, thromboses.

2.3. List of questions to check basic knowledge on the subject of the lesson.
1. General characteristics of viral airborne infection, classification.
2. AIDS, morphological characteristics, complications of the cause of death.
3. Rickettsiosis, classification, morphological characteristics, complications of the cause of death.
4. Corona virus disease, morphological characteristics, complications of the cause of death.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

During the patho-anatomical examination of the body of a 5-year-old boy who died of acute pulmonary heart failure, it was found: serous-hemorrhagic tracheobronchitis with areas of mucosal necrosis, multiple foci of hemorrhagic pneumonia in the lungs. What disease is it about?
The correct answer is Flu
B Kand
C Scarlet fever
D Diphtheriai
E Croupous pneumoniai
A 12-year-old child, who was treated in the infectious department for influenza, developed a severe headache, nausea, dizziness, and meningeal signs on the 5th day of the illness. Death came a day later from growing cerebral edema. During the dissection of the skull, the soft meninges are swollen, full of blood, diffusely soaked with a bright red liquid. Convolutions and furrows are smoothed. What complications of flu should be considered?
Correct answer Hemorrhagic meningitis
BHemorrhage in the brain
CVenous hyperemia of the brain membranes
Purulent leptomeningitis
Serous meningitis
A 42-year-old man died of severe intoxication and respiratory failure. On cross-section, the lung tissue in all departments is variegated, with multiple small foci of hemorrhages and foci of emphysema. Histologically in the lungs: hemorrhagic bronchopneumonia with abscessation, eosinophilic and basophilic inclusions in the cytoplasm of bronchial epithelial cells. What is the most likely diagnosis?
Correct answer Flu
Parainfluenza
Adenovirus infection
Respiratory syncytial infection
Staphylococcal bronchopneumonia
A 78-year-old man died as a result of cachexia. He suffered from progressive dementia for the last 2 years of his life. At the autopsy, macroscopically, a slight decrease in brain mass and moderate atrophy of the gyri were noted. Microscopically, optically empty oval-shaped vacuoles (spongiosis) were detected in all parts of the cortex of the large hemispheres of the brain, in the region of the subcortical nuclei, the cerebellar cortex, and the thalamus, as well as the loss (loss) of neurons of the III-VI layer in combination with the proliferation of astroglia. There are also rounded eosinophilic structures with a positive dye reaction to amyloid. Such a microscopic picture is characteristic of:
Correct answer Prion encephalopathy
BViral encephalitis
Alcohol intoxication
Duremic encephalopathy

Hepatic encephalopathy
A 23-year-old patient died of cerebral edema. Microscopically, hematuria, edema, perivascular infiltrates consisting of lymphocytes, plasma cells, and macrophages, lysis of neurons, hyperplasia of astrocytes and microglial cells, and small nodules were found in the brain tissue. Such a microscopic picture is characteristic of:
Correct answer Prion encephalopathy
BViral encephalitis
Alcohol intoxication
Parasitic infection
Uremic encephalopathy
At the autopsy of a patient who died of typhoid fever, it was found that the muscles of the front abdominal wall and the inner surface of the thighs are dense, whitish-yellow in color, and externally stearin- (paraffin)-like. The described changes in the muscles are a manifestation of:
Correct answer Zenker's necrosis
Apoptosis
CFibrinoid necrosis
D Collective necrosis
Caseous necrosis
A 34-year-old patient was admitted to the hospital with complaints of chills, an increase in body temperature up to 40 degrees, headache, weakness. By the end of the 5th day, a small roseolous rash (exanthema) appeared on the skin, a collapse developed. Diagnosed with typhoid fever. What morphological changes are at the heart of exanthema:
Correct answer Destructive-proliferative endothrombovasculitis
BVenous hyperemia
Arterial hyperemia
Periarteritis
ESegmentary fibrinous necrosis of arterioles
During the flu epidemic, a 67-year-old man complained of chills, temperature rise to 390, headache, cough, severe shortness of breath. Humid rales were detected in the lungs. 2 days later, despite intensive therapy, the patient died of pulmonary and cardiac failure. What type of inflammation will be observed in the trachea and bronchi, which will confirm the diagnosis of influenza at autopsy?
Correct answer Catarrhal tracheobronchitis
B Serous-desquamative tracheobronchitis
CDiphtheritic tracheobronchitis
DFibrinous and hemorrhagic tracheobronchitis
Phlegmous tracheobronchitis
A 43-year-old patient died on the 5th day of illness during an influenza epidemic. The mucous membrane of the respiratory tract and the gastrointestinal tract is soaked with hemorrhagic exudate and dotted with small focal hemorrhages. In the larynx and trachea – fibrinous-hemorrhagic inflammation with large foci of necrosis in the

mucous membrane, fibrinous-hemorrhagic panbronchitis. There are massive hemorrhages and small foci of hemorrhagic pneumonia in the lungs. Full blood, edema, hemorrhagic impregnation and small focal hemorrhages were found in the brain tissue. The leading role in thanatogenesis was played by:
Correct answer Influenza toxicosis
BCardiovascular complications
Encephalopathy
DPneumonia
ENecrotic and infectious complications
The 27-year-old patient died due to severe intoxication and respiratory failure. On cross-section, the lung tissue in all departments is variegated with multiple small focal hemorrhages and foci of emphysema. Histological examination: hemorrhagic pneumonia with abscess in the lungs, eosinophilic and basophilic inclusions in the cytoplasm of the bronchial epithelium. Diagnose the disease detected at the section:
Correct answer Flu
BRespiratory syncytial infection
Adenovirus infection
Parainfluenza
Staphylococcal bronchopneumonia
A 6-year-old boy developed sharp pain when swallowing, severe swelling of the neck, body temperature rose to 39.0 degrees. Grayish-yellow films appeared on the tonsils, which were removed due to force. Marked signs of general intoxication. Which of the following diseases are we talking about:
Correct answer Diphtheria
Measles
Scarlet fever
Meningococcal nasopharyngitis
Influenza
During hospitalization during the flu epidemic, a 58-year-old patient complained of chills, temperature rise to 38.5 degrees for 10 days, shortness of breath, cough with sputum. The phenomena of respiratory failure progressed, after 7 days death occurred from cardiopulmonary failure. Diagnose the disease detected at the section:
Correct answer A severe form of influenza with pulmonary complications
B. Influenza with bacterial and septic complications
Influenza with hemorrhagic cerebral edema
Influenza with the advantage of toxicosis
Moderate flu
A 5-year-old child was taken to the hospital's reception department in a state of asphyxiation. In the larynx there are white-yellow films that block the respiratory tract and separate with force. A tracheotomy was performed. What kind of inflammation developed in the larynx?
Correct answer Diphtheritic
B Catarrhal

C Diphtheroid
D It's big
EMixed
A 9-year-old child who had the flu developed a severe headache, nausea, dizziness, and meningeal signs on the 5th day of the illness. Death came a day later due to swelling of the brain, which was increasing. During the dissection of the cranial cavity, the soft meninges are swollen, full of blood, and diffusely soaked with a bright red liquid. What complications of flu should be considered?
Correct answer Hemorrhagic meningitis
BHemorrhage in the brain
CVenous hyperemia of the brain membranes
Purulent leptomeningitis
Serous meningitis
Define opportunistic infection:
Correct answer Malovirulent infections that can lead to disease only in immunodeficient states
BLowly contagious respiratory infections
Highly contagious respiratory infections
Extremely rare tumor diseases that occur only in immunodeficient states.
EHemotransmissible diseases that occur only in immunodeficient states.
Opportunistic infections that develop during AIDS do not include:
Correct answer Cysticercosis
B Toxoplasmosis
Pneumocystosis
D Cytomegalovirus infection
Candidiasis
At the autopsy of the deceased, acute pneumocystis pneumonia was found, purple-red spots, plaques and nodules (Kaposh's sarcoma) were found on the skin of the distal parts of the lower limbs. For which disease are these changes characteristic?
Correct answer AIDS
B Influenza
Measles
Diphtheria
Anthrax
Patient M. is 32 years old and has been diagnosed with AIDS. When examining a biopsy of enlarged lymph nodes, sharply enlarged follicles with large bright centers were found, in which immunoblasts and numerous mitoses were detected. Such a morphological picture is characterized as:
Correct answer Follicular hyperplasia
B Diffuse hyperplasia
Immunoblastic lymphoma
DDepletion of lymphoid tissue
Follicular hypoplasia

At the patient's re-examination after a long time, a biopsy of enlarged lymph nodes revealed erosion of their structure due to hyperplasia of lymphocytes, plasma cells, immunoblasts, eosinophils and a sharp reduction in the number of follicles. Follicles are small, atrophic, often with hyalinosis in the center. Such a morphological picture is characterized as:
Correct answer Depletion of lymphoid tissue
BFollicular hyperplasia
Lymphoma
Diffuse hyperplasia
Follicular hypoplasia
A 42-year-old patient was brought to the surgical department in an extremely serious (terminal) condition. From the anamnesis: he has been suffering from drug addiction for more than 10 years. When testing blood for HIV, the result is positive. At autopsy: diffuse fibrinous-purulent peritonitis, interloop abscesses, left-sided pyoovarium and pyosalpingus with perforation, bilateral lower lobe bronchopneumonia, cachexia, lymph nodes are reduced in size, dense, their structure is not determined. Name the period of AIDS:
Correct answer AIDS
BPre-Breakfast
CSnid-Associated complex
D Persistent generalized lymphadenopathy
EIncubation
A 22-year-old patient suffering from drug addiction was admitted to the infectious department with signs of infectious mononucleosis. When testing blood for HIV, the result is positive. After 2.5 weeks, the patient's condition normalized. Name the period of HIV infection:
Correct answer Sneed-Associated complex
BPre-Breakfast
AIDS
D Persistent generalized lymphadenopathy
EIncubation
For the Sneed-Associated complex, the most typical manifestation is:
Correct answer Secondary intercurrent infections: acute respiratory viral infections, candidiasis, pyoderma, herpes, tuberculosis
Opportunistic infections:
CNeuro-Snead
D Mononucleosis-like infectious disease
EOnko-Snead
For the pulmonary variant of AIDS, the typical manifestation is:
Correct answer Pneumocystis pneumonia
BHemorrhagic pneumonia:
Friedlander's pneumonia
Caseous pneumonia

EViral pneumonia
The intestinal-diarrheal syndrome characteristic of the terminal stage of AIDS is caused by opportunistic intestinal infections:
Correct answer Cryptosporidiosis
BColi - Bacillary infection
C Salmonellosis
D Cytomegalovirus infection
E Dysenteric amoebiasis
A 35-year-old woman is suffering from HIV infection at the stage of SNID. Reddish-red spots, bright red nodules of various sizes appeared on the skin of the lower extremities and the mucous membrane of the palate. One of the nodules was taken for histological examination. Many thin-walled vessels lined with endothelium were found in a chaotic manner; bundles of spindle-shaped cells with the presence of hemosiderin. What tumor has developed in the patient?
Correct answer Kaposi's sarcoma
BHemangioma
Burkitt's lymphoma
D Lymphangioma
EFibrosarcoma

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

34. methods: assessment of the correctness of the performance of practical skills
35. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Childhood infections.".

Suggested topics for essays:

1.Corona virus disease. Morphological characteristics.

2.AIDS.Morphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 36

Topic: Childhood infections.

Goal:to study clinical and morphological manifestations, complications, causes of death in children's infections.

Basic concepts: General characteristics dinfectious diseases (diphtheria, meningococcal infection, whooping cough, scarlet fever), morphological characteristics, complications, causes of death.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Diphtheria- an acute infectious disease, which is characterized by the formation of fibrinous inflammation in the center of primary fixation of the pathogen and general intoxication associated with the absorption of exotoxin.
Features of diphtheria are that it:
1. belongs to the number of characteristic so-called local infections, the causative agents of which, as a rule, are found only in the area of the primary localization of the process;
2. the lifelong symptom complex of diphtheria and all the underlying anatomical changes are the result of exposure to the body of diphtheria toxin, so there is not a single symptom in the entire picture of the disease that could not be obtained experimentally with the help of one poison without the participation of living pathogens.
The patho-anatomical characteristic is that the most constant reaction of local tissues to the penetration of the causative agent into them is fibrinous inflammation in the form of diphtheritic or croupous.
Scarlet fever is an acute infectious disease with initial damage most often to the pharynx. The name comes from the word "scarlatina" - bright red. The causative agent of the disease is hemolytic streptococcus.
Complication: There is damage to the mucous membrane, underlying tissues, ethmoid bone (ethmoiditis). The transition of the process from the vein to the eyes, meninges and brain tissue is possible with the development of purulent meningitis or brain abscess, the spread of infection through the Eustachian tube, into the middle ear, developing otitis, thrombophlebitis of the memoid sinus, purulent meningitis or brain abscess.
Meningococcal infection.
It is one of the forms of meningococcal infection with predominant damage to the soft meninges of the brain and spinal cord. In addition to meningitis, nasopharyngitis and meningococcemia of a meningococcal nature are distinguished.
Meningococcal meningitis is more common in children under the age of five.
With a prolonged course, complications may occur. Circulation of the cerebrospinal fluid may be disrupted if the outflow channels are clogged with exudate or obliterated, and when it is organized, internal hydrocephalus develops in the brain.

The transition of the inflammatory process to the arteries of the brain with their thrombosis and disruption of the blood supply to areas of the brain in which there are foci of softening is possible.
Meningococcemia may develop with the most severe course of the disease. In this case, foci of inflammation appear in various organs and tissues. Damage to soft meninges sometimes does not have time to develop and patients die within 24-48 hours. Typical vascular changes, hemorrhage in various organs, in the skin.
Hemorrhage in the adrenal glands, which is accompanied by acute adrenal insufficiency, is especially dangerous. Vascular thrombosis and necrotic changes in the skin are observed.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. General characteristics of viral airborne infection, classification.
2. AIDS, morphological characteristics, complications of the cause of death.
3. Rickettsiosis, classification, morphological characteristics, complications of the cause of death.
4. Corona virus disease, morphological characteristics, complications of the cause of death.
5. Diphtheria, morphological characteristics, complications, causes of death.
7. Scarlet fever, morphological characteristics, complications, causes of death.
8. Meningococcal infection, morphological characteristics, complications, causes of death.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

A 5-year-old girl has a high temperature and a sore throat. Objectively: swelling of the soft palate, gray films on the tonsils, which are difficult to separate, leaving deep bleeding tissue defects. What disease is most likely?
Correct answer Diphtheria of the pharynx
Angina of Symanovsky-Vincent
Lacunar angina
Infectious mononucleosis
ENecrotic angina
The disease in an 8-year-old child began acutely - with an increase in temperature to 39 degrees, sore throat and an increase in submandibular lymph nodes. In the larynx, against the background of edema and hyperemia, there are grayish-white membranous layers, which are tightly welded to the underlying tissues and upon removal of which

tissue defects are formed. Marked phenomena of intoxication, according to the ECG, dystrophic changes in the myocardium are sharply expressed. Make a diagnosis:
Correct answer Diphtheria
BWhooping cough
Measles
Meningococcal infection
Scarlet fever
An 8-year-old child became acutely ill. Death occurred 2 days after the onset of the disease. At the autopsy, it was found that the soft meninges were sharply filled with blood, on the basal surface of the brain they were impregnated with a thick cloudy yellowish-greenish exudate. Brain tissue is swollen. Make a diagnosis:
Correct answer Meningococcal infection
BWhooping cough
Measles
Diphtheria
Scarlet fever
In a child after measles, during examination, in the soft tissues of the cheeks and perineum, vaguely demarcated swollen areas of red-black color, which fluctuate slightly, were found. What complication did the child develop?
Correct answer Wet gangrene (noma)
B Trophic ulcer
Dry gangrene
DGas gangrene
Bedsores
The disease in a 6-year-old child began acutely, the clinical picture was dominated by headache, nausea, and vomiting. After 2 days, death came. At autopsy, the meninges are full of blood, and on the basal and convex surface of the brain, they are impregnated with a thick cloudy yellowish-greenish exudate, the brain is swollen. For which disease is this picture characteristic?
Correct answer Meningococcal infection
Scarlet fever
Whooping cough
Measles
Diphtheria
An 18-year-old girl developed sharp pain when swallowing, an increase in lymph nodes in the neck, and a temperature rise to 38 degrees. On the mucous membrane of the tonsils, there are white-yellow films that are difficult to separate with the formation of a defect in the underlying tissues. The patient's condition progressively worsened. The patient died on the 8th day of the disease due to increasing symptoms of heart failure. What histological changes are most likely to be found in cardiomyocytes?
Correct answer Fatty dystrophy
BBalloon dystrophy
Mucosal dystrophy

DHydropic dystrophy
EHyaline-drip
The child was taken to the sanatorium in a state of asphyxiation. When examining the larynx, whitish films obscuring the lumen, which are easily separated, were found. The doctor suspected diphtheria. What form of inflammation of the larynx are we talking about?
Correct answer Croupous inflammation
B Purulent inflammation
CDiphtheritic inflammation
Catarrhal inflammation
E Serous inflammation
A 5-year-old girl fell ill with diphtheria. On the third day, she died of asphyxiation as a result of true croup. At the autopsy, it was established that the mucous membrane of the larynx, trachea, and bronchi is thickened, swollen, cloudy, covered with grayish films that are easily separated. What pathological process is evidenced by morphological changes in the larynx:
Correct answer Croupous inflammation
B Catarrhal inflammation
Purulent inflammation
D Serous inflammation
EDiphtheritic inflammation
At the autopsy of the patient, it was found: the soft meninges of the upper hemispheres of the brain were sharply full-blooded, yellow-green in color, impregnated with purulent and fibrinous exudate in the form of a pincer. What disease is characterized by such a picture:
Correct answer Meningococcal meningitis
Meningitis with typhoid fever
CInfluenza meningitis
Tuberculous meningitis
Meningitis with anthrax
In a 28-year-old patient, the tonsils are significantly enlarged, full-blooded, painful, on their surface there are dense dirty-gray films that spread to the hard palate, are tightly connected to the underlying tissues, and bleeding develops when trying to separate them. What pathological process causes these morphological changes:
Correct answer Diphtheritic exudative inflammation
B Croupous exudative inflammation
Catarrhal exudative inflammation
Purulent exudative inflammation
EHemorrhagic exudative inflammation
An 11-year-old child had a sudden rise in temperature to 39 degrees with chills, headache, vomiting, restlessness and motor restlessness. A few days later, a hemorrhagic rash appeared on the body, damage to the vascular membrane of the eyes and joints, which was joined by oliguria, and adrenal insufficiency developed, which

served as the cause of death. Meningococcus was found in throat tests. What is the most likely diagnosis:
Correct answer Meningococcemia
B Meningococcal nasopharyngitis
Meningococcal meningitis
DProgressive hydrocephalus
EProgressive cerebral cachexia
In a 5-year-old child, during the examination of the throat, it was found: the mucous membrane of the throat and tonsils is hyperemic, the tonsils are enlarged, covered with dense, whitish films that are difficult to remove. A deep tissue defect remains at the removal site. The soft tissues of the neck are swollen, the regional lymph nodes of the neck are enlarged and painful. What disease can you think of?
Correct answerCorrect answer Diphtheria
Scarlet fever
CKir
Mumps
Adenovirus infection
During the examination of the patient's tonsils, the dentist found an overlay in the form of grayish films that were difficult to remove. What disease can you think of?
Correct answer Diphtheria of the pharynx
B Influenza
CKir
Follicular angina
Scarlet fever

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

36. methods: assessment of the correctness of the performance of practical skills
37. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Tuberculosis.".

Suggested topics for essays:
1.Diphtheria. Morphological characteristics.
2.MeaslesMorphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 37

Topic: Tuberculosis.

Goal:to study clinical and morphological manifestations of tuberculosis, complications, causes of death.

Basic concepts: General characteristics of tuberculosis, classification. Primary tuberculosis, hematogenous tuberculosis, secondary tuberculosismorphological characteristics, complications, causes of death.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;
Tuberculosis

Tuberculosis is a common infectious disease caused by Mycobacterium tuberculosis. Most often, the lungs are affected, but other organs and tissues can also be involved in the process.
Modern clinical classification of tuberculosis.
A. Main clinical forms.
Gr. I. Tuberculosis of respiratory organs:
Primary tuberculosis complex.
Tuberculous bronchoadenitis.
Hematogenously disseminated tuberculosis.
Focal tuberculosis of the lungs.
Infiltrative-pneumonic tuberculosis of the lungs.
Pulmonary tuberculosis.
Cavernous tuberculosis of the lungs.
Fibrous-cavernous tuberculosis of the lungs.
Cirrhosis of the lungs.
Other forms (pleurisy, bronchitis, tracheitis, laryngitis).
Gr. II. Tuberculosis intoxication in children and adolescents.
Gr. III. Tuberculosis of other organs (CNS, intestines, kidneys, skin, bones).
B. Nature of the tubercular process.
and). open forms (BC +).
b). closed forms (BK-).
Phases of the process.
and). infiltration, decay, insemination.
b). compaction, scarring, calcification, resorption.

B. Complications of tuberculosis.
Atelectasis
Pulmonary - heart failure.
General amyloidosis.
Bleeding.
Pneumothorax.
Secondary tuberculosis is characterized by:
1. limitation of the process by the lungs.
2. unilateral lung damage.
3. predisposition to a chronic course.
One of the main features of secondary tuberculosis is:
1. rarity of hematogenous and lymphogenous metastases to other organs and systems.
2. the forms of secondary tuberculosis are quite diverse, some of them are genetically related to each other and to phases in the development of the following more severe forms. A.I. Strukov and I.A. Kusevytsky, on the basis of clinical, radiological and patho-anatomical studies, singled out 8 forms of secondary pulmonary tuberculosis:
1. acute focal.
2. fibrinous - focal.
3. infiltrative - pneumonic
4. tuberculoma, as a form of tuberculosis that is pathogenetically related to infiltrative-pneumonic tuberculosis.
5. caseous pneumonia.
6. sharp cavernous.
7. fibrous-cavernous.
8. cirrhotic tuberculosis.
The causes of death of patients with fibro-cavernous tuberculosis and those who died from this disease in a hospital are divided into 4 groups:

- progression of fibrous-cavernous tuberculosis;
- progression of fibrous-cavernous tuberculosis in the presence of a secondary no less serious disease, which in itself could cause death and can only conditionally be classified as concomitant.
- specific and non-specific complications in fibro-cavernous tuberculosis regardless of its phase.
- combined forms of tuberculosis

2.3. List of questions to check basic knowledge on the subject of the lesson.
1. General characteristics of tuberculosis, classification.
2. Primary tuberculosis, classification, morphological characteristics, complications of the cause of death.
3. Hematogenous tuberculosis, classification, morphological characteristics, complications of the cause of death.

4. Secondary tuberculosis, classification, morphological characteristics, complications of the cause of death.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

The autopsy of a man who died of acute post-hemorrhagic anemia as a result of pulmonary bleeding revealed: macroscopically, the tops of the lungs are deformed, on cross-section there are multiple whitish-gray cells with a diameter of 10-15 mm and multiple pathological cavities with a diameter of up to 15 mm; microscopically - in the walls of the cavities, the growth of connective tissue with the presence of an infiltrate consisting of epithelioid cells, multinucleated giant cells, and lymphocytes. What is the most likely diagnosis?

Correct answer Secondary f ibrous-cavernous tuberculosis

B Primary tuberculosis without signs of progression

Progressive primary tuberculosis complex

DHematogenously disseminated pulmonary tuberculosis

Hematogenous miliary tuberculosis of the lungs

A 47-year-old patient has been suffering from pulmonary tuberculosis for the past 3 years, complains of shortness of breath, heaviness in the area of the right side of the chest; body temperature - 37.7oC. Right-sided exudative pleurisy was detected. What type of cells is expected in a pleural punctate?

Correct answer Lymphocytes

B Neutrophils

Erythrocytes

Atypical cells

Eeosinophils

An autopsy of a 47-year-old man who died of pulmonary hemorrhage revealed a 5.5 cm round cavity with uneven edges in the 2nd segment of the right lung; its inner surface is covered with compacted yellowish masses that pass into the lung tissue. In histological examination, the inner layer consists of melted caseous masses, necrotized lung tissue. What process developed in the lungs?

Correct answer Acute cavernous tuberculosis

Disintegrating lung cancer

Chronic cavernous tuberculosis

DAcute abscess

Chronic abscess

An enlarged supraclavicular lymph node was removed from a young man. Histologically: in the center - a center of serous necrosis, on the periphery - a shaft of

epithelioid cells and lymphocytes, among which Pirogov-Langhans cells are found. Diagnose the process in the lymph nodes:
Correct answer Tuberculous lymphadenitis
Syphilitic lymphadenitis
Lepromatous lymphadenitis
D Lymphogranulomatosis
Virkhov's metastases
A 13-year-old child has swelling in the branch of the lower jaw. Enlarged and painless submandibular and cervical lymph nodes. Mantoux reaction is positive. The radiograph of the branch of the lower jaw shows bone resorption with clear contours, small sequestrations. During histological examination of a biopsy of one of the lymph nodes, foci of caseous necrosis were found, which are surrounded by shafts of epithelioid cells and lymphocytes, between which Pirogov-Langhans giant cells are located. What is the disease of the branch of the lower jaw in a child?
Correct answer Tuberculosis
B Chronic osteomyelitis
Ewing's sarcoma
Acute osteomyelitis
Osteoblastoclastoma
An autopsy of a 47-year-old man who died of pulmonary hemorrhage revealed a 5.5 cm round cavity with uneven edges in the 2nd segment of the right lung; its inner surface is covered with compacted yellowish masses that pass into the lung tissue. In histological examination, the inner layer consists of melted caseous masses, necrotized lung tissue. What process developed in the lungs?
Correct answer Acute cavernous tuberculosis
Disintegrating lung cancer
Chronic cavernous tuberculosis
DAcute abscess
Chronic abscess
In a 30-year-old man, histological examination of a cervical lymph node biopsy revealed granulomas consisting of epithelioid, lymphoid, multinucleated giant cells of the Pirogov-Langhans type. Necrosis is determined in the center of the granuloma. What causative agent must be detected in the necrosis zone to confirm the diagnosis of tuberculosis:
Correct answer Mycobacterium Koch
Staphylococcus
Salmonella
Bacillus Volkovich-Frisch
E Treponema pallor
The child's lymph nodes increased dramatically after a sore throat: paratracheal, bifurcation, and cervical. Microscopic examination of the cervical lymph node revealed foci of necrosis, limited by lymphocytes, epithelioid cells, and Pirogov-Langhans cells. Specify the most likely pathology:

Correct answer Tuberculosis
BSarcoidosis
Rhinoscleroma
DSap
Syphilis
In a 28-year-old man, a histological examination of the cervical lymph node revealed a pattern disturbance due to the growth of epithelioid, lymphoid cells and macrophages with horseshoe-shaped nuclei, in the center of some clusters of cells - structureless areas of pale pink color with fragments of nuclei. What disease is characterized by these changes:
Correct answer Tuberculosis
Actinomycosis
Syphilis
D Tumor metastasis
Lymphogranulomatosis
A patient with fibrous-cavernous tuberculosis died with increasing symptoms of renal failure. At the autopsy, the smell of urine, hypertrophy of the left ventricle, fibrinous pericarditis, fibrinous hemorrhagic enterocolitis. kidneys are slightly reduced in size, very dense, with multiple cords. Histologically, on preparations stained with congo rot, there are pink masses in glomeruli and vessel walls, death and atrophy of most nephrons, nephrosclerosis. Describe the kidneys in this pathology:
Correct answer Amyloid shrunken kidneys
BSecondary shrunken kidneys
C Pyelonephritic shriveled kidneys
D Atherosclerotic shrunken kidneys
EPrimarily shrunken kidneys
In a patient with subfebrile temperature, a biopsy of an enlarged cervical lymph node revealed numerous granulomas, which contain caseous necrosis in the center, surrounded by epithelioid cells, giant multinucleated Pirogov-Langhans cells, and lymphocytes. Which disease is characterized by the following pathohistological changes:
Correct answer Tuberculosis
Banal lymphadenitis
Lymphosarcoma
D Lymphogranulomatosis
Elympholeukosis
Microscopic examination of a skin biopsy revealed granulomas consisting of epithelioid cells surrounded mainly by T-lymphocytes. Single giant multinucleated cells of the Pirogov-Langhans type are located among the epithelioid cells. Areas of caseous necrosis are determined in the center of some granulomas. There are no blood vessels. For which disease are the described granulomas characteristic:
Correct answer Tuberculosis
BSap

CLeprosy
Syphilis
Rhinoscleroma
A 63-year-old man has been suffering from fibro-cavernous tuberculosis of the lungs for 24 years and was admitted to the nephrology department with symptoms of uremia. The intravital test for the presence of amyloid in the kidneys was positive. What form of amyloidosis occurs in this case:
Correct answer Secondary systemic
BLimited (local)
Senile (senile)
DFamily born
EPrimary system
During the autopsy of the body of a 9-year-old girl, a 15 mm diameter caseous necrosis center was found in the apex of the right lung, the bifurcation lymph nodes were enlarged and contained small coagulation-type necrosis centers. Microscopically, epithelioid cells, lymphocytes and isolated giant cells were located in the lung tissue and lymph nodes around the necrotic masses. Diagnose the disease:
Correct answer Primary tuberculosis
BHematogenous tuberculosis with predominant lung damage
CHematogenous generalized tuberculosis
DSecondary focal tuberculosis
ESecondary -fibrous-focal tuberculosis
In a 72-year-old man, an enlargement and deformation of the right knee joint was detected at the autopsy. A histological examination of the tissues of the joint and adjacent areas revealed: massive foci of caseous necrosis, which are surrounded by a shaft of epithelioid, lymphoid cells with the presence of giant macrophages. Diagnose the disease:
Correct answer Tuberculous arthritis
B Deforming osteoarthritis
Syphilitic arthritis
Rheumatoid arthritis
Gonorrheal arthritis
Microscopic examination of the cervical lymph node revealed clusters of epithelioid cells, lymphocytes and giant multinucleated Pirogov-Langhans cells. Caseous necrosis in the center. Specify the most likely pathology:
Correct answer Tuberculosis
Syphilis
Sarcoidosis
Rhinoscleroma
ESap

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

38. methods: assessment of the correctness of the performance of practical skills
39. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .

"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Sepsis. Syphilis.".

Suggested topics for essays:

1.Secondary tuberculosis. Morphological characteristics.

2.Visceral syphilis.Morphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine

2.www.ama-assn.org– American Medical Association /American Medical Association

3.www.who.int- World Health Organization

4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine

5.http://bma.org.uk– British Medical Association

6.www.gmc-uk.org- General Medical Council (GMC)

7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 37

Topic: Sepsis. Syphilis.

Goal:to study clinical and morphological manifestations of sepsis, complications of syphilis, causes of death.

Basic concepts: General characteristics of the villageEpsis, morphological characteristics, complications, causes of death. Syphilis, morphological characteristics, complications, causes of death.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

Sepsis (from the Greek Sepsis - putrefaction) is a common infectious disease,which onearises in connection with the existence of a focus of infection in the body and has a number of differences from other infectious diseases.
Classification. With sepsis, it is necessary to take into account the following signs: 1) etiological; 2) the nature of the entrance gate (location of the septic fire); 3) clinical and morphological.
. In case of sepsis, local and local are distinguishedchanges. localchangesdevelop in the cell of penetration of the pathogen (entrance gate) or at a certain distance from it. Fires are formed,whichrepresent a purulent focusinflammation(In some cases, there is no septic focus).WITHof a septic focus, the infection quickly spreads through the lymphatic and blood vessels. The spread of infection through the lymphatic system is

accompanied by the development of lymphangitis, lymphothrombosis, and lymphadenitis, and the spread through the circulatory system (through the veins) leads totodevelopment of phlebitis and thrombophlebitis. Purulent thrombophlebitis often occurs, which leads totothrombus melting and thrombobacterial embolism.
general changes in sepsis are dystrophic in nature,inflammatoryand hyperplastic processes. Dystrophic*changes* develop in the parenchymal (liver, kidneys, myocardium, muscles, central nervous system) and manifestdifferenttypes of dystrophy and necrobiosis, which often ends in necrosis.
Incendiary changes represented by intermediate (interstitial) processes (intermediate septic nephritis, hepatitis, myocarditis). In the valves of the heart, acute polyposis-ulcerative endocarditis with melting of the tissue and detachment of the valves can occur.Incendiary changesalso occur in blood vessels (vasculitis), which leads to the appearance of numerous hemorrhages. However, hemorrhagic syndromeconnectedwith sepsis not only with vasculitis, but also with intoxication, increased vascular and tissue permeability, anemia, etc.
Four clinical and anatomical forms of sepsis are distinguished by clinical and morphological features: septicemia, septicopyemia, septic (bacterial) endocarditis and chronosepsis.
Syphilis (Syphilis named after the hero of the poem by doctor J. Fracastoro the shepherd Syphilus) is a chronic infectious venereal disease with damage to the skin, mucous membranes, internal organs, bones, nervous system and successive changes in the stages (periods) of the disease. 3 periods of syphilis: primary, secondary and tertiary (humous).

Congenital syphilis develops when the fetus is infected in utero through the placenta from a sick mother. It is divided into 3 forms:

- syphilis of stillborn premature fetuses;

- early congenital syphilis of newborns and infants;

- late congenital syphilis of children of preschool and school age, as well as adults.

2.3. List of questions to check basic knowledge on the subject of the lesson.
1. General characteristics of tuberculosis, classification.
2. Primary tuberculosis, classification, morphological characteristics, complications of the cause of death.
3. Hematogenous tuberculosis, classification, morphological characteristics, complications of the cause of death.
4. Secondary tuberculosis, classification, morphological characteristics, complications of the cause of death.
5. Sepsis, classification, morphological characteristics, complications, causes of death.

7. Syphilis, classification, morphological characteristics, complications, causes of death.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

On the mucous membrane of the right palatine tonsil, a painless ulcer with a smooth lacquered bottom and smooth cartilage-like edges is observed. Microscopically: an inflammatory infiltrate consisting of lymphocytes, plasma cells, a small number of neutrophils and epithelioid cells and the presence of endo- and perivasculitis. What disease is it about?

Correct answer Syphilis

Actinomycosis

Tuberculosis

Diphtheria of the pharynx

EVencin's ulcerative-necrotic tonsillitis

A 23-year-old man developed a perforation of the hard palate, in the area of which a dense formation with clear boundaries was found. After the operation, a microscopic examination of this formation revealed: a significant caseous necrosis center surrounded by granulation tissue with endovasculitis, a cellular infiltrate consisting of lymphocytes, epithelioid cells, with a predominance of plasma cells. What is the most likely disease in the patient?

Correct answer Syphilis

B Tuberculosis

CScleroma

DSarcoma

E Leprosy

A 14-year-old patient was diagnosed with Hutchinson's triad: elongated teeth, parenchymal keratitis and deafness. For which disease are the detected changes characteristic?

Correct answer Syphilis

B Toxoplasmosis

Leprosy

D Tuberculosis

Opistorchosis

3 days after the criminal abortion, the patient's temperature rose to 40 degrees, petechial hemorrhages appeared in the skin, confusion of consciousness. After 2 days,

death came. Histologically revealed: interstitial inflammation of the myocardium, liver and kidneys, septic spleen. What form of sepsis is most likely:
Correct answer Septicemia
BChroniosepsis
CSepticopemia
D Chernogubov's disease
Prolonged septic endocarditis
An employee of a livestock farm became acutely ill and died due to increasing symptoms of intoxication. At the autopsy, it was found that the spleen was enlarged, flaccid, dark cherry-colored on the section, pulp scraping was abundant. The soft meninges of the vault and the base of the brain are swollen, soaked with blood, and have a dark red color ("cardinal's cap"). Microscopically: serous-hemorrhagic inflammation of the membranes and tissues of the brain with destruction of the walls of small vessels. Make a diagnosis:
Correct answer Anthrax
Brucellosis
Cholera
D To the plague
ETularemia
At the autopsy of a child suffering from purulent omphalitis, liver abscesses, an enlarged spleen, which gives abundant scraping of the pulp, purulent meningoencephalitis, and purulent ulcerative colitis were found. Diagnose the disease:
Correct answer Septicopemia
BChroniosepsis
Meningococcal meningitis
Dysentery
ENonspecific ulcerative colitis
An aneurysm of the ascending part of the aorta was revealed at the section. Microscopically, an inflammatory infiltrate of lymphocytes, plasma cells, fibroblasts and giant multinucleated cells was found in the wall of the aorta. What is the most likely diagnosis:
Correct answer Syphilis
Atherosclerosis
Rheumatism
Hypertensive disease
Tuberculosis
A 35-year-old patient suddenly developed diarrhea with watery stools. Later, profuse repeated vomiting joined, signs of dehydration began to appear - the skin became wrinkled, the patient suffered from severe thirst. Suffocation, hoarseness of voice, convulsions appeared, a comatose state developed, and death came. At autopsy, the lumen of the small intestine is sharply expanded, filled with a liquid similar to rice broth. The wall of the intestine is swollen, with multiple small focal hemorrhages. What is the most likely diagnosis:

Correct answer Cholera
Amebiasis
C Dysentery
Typhoid fever
Tuberculosis
The hunter's axillary lymph nodes on the right increased dramatically after a flea bite. Their sizes reached 6-7 cm in diameter, soldered together, soft consistency, motionless. During the examination of the biopsy, foci of necrosis, hemorrhages, proliferation of reticular cells were found. The tissue of the lymph nodes is saturated with serous exudate and contains a large number of microorganisms. Your diagnosis:
Correct answer Plague
BLympholeukosis
Lymphogranulomatosis
D Lymphosarcoma
Anthrax
A 45-year-old patient, a resident of a rural area, complained of malaise, insomnia, irritability, headache, pain in muscles and joints, decreased appetite, low-grade fever for a long time. Death occurred due to multiple organ failure. At autopsy, granulomas formed by epithelioid macrophages with an admixture of plasma cells, eosinophils, and single cells of the Pirogov-Langhans type were found in many organs. In addition to granulomatosis, systemic productive-destructive vasculitis was detected. Your diagnosis:
Correct answer Brucellosis
Btularemia
Cholera
D Plague
Anthrax
In an 18-year-old patient, the inguinal lymph nodes are enlarged, painless, palpably dense. In the area of the mucous membrane of the genitals, there is a small ulcer with thickened edges, a grayish "varnish" bottom. What is the most likely diagnosis:
Correct answer Syphilis
B Tuberculosis
CLeprosy
D Trophic ulcer
Gonorrhea
Patient M. is 14 years old and diagnosed with Hutchinson's triad: barrel-shaped teeth, parenchymal keratitis and deafness. Which disease is characterized by the detected changes:
Correct answer Syphilis
B Toxoplasmosis
Leprosy
D Tuberculosis
Opistorchosis

Liver biopsy of a 39-year-old patient revealed granulomas consisting of epithelioid, lymphoid, plasma cells and isolated giant macrophages of the Pirogov-Langhans cell type. In granulomas, there are many small vessels with signs of endo- and perivasculitis, in some there are foci of caseous necrosis. Which disease is characterized by the detected changes:
Correct answer Syphilis
BLeprosy
Tuberculosis
Rhinoscleroma
ESap
A 25-year-old man developed a saucer-shaped ulcer 0.8 cm in diameter on the back of his tongue on the left side. The bottom and edges of the ulcer are dense, their surface is smooth, shiny, painless on palpation. Microscopic examination shows an infiltrate of lymphoid, plasmatic, and epithelioid cells at the border of the ulcer and in the area of its bottom, with the presence of a significant number of vessels with endovasculitis. What is the most likely diagnosis?
Correct answer Primary syphilis
BDecubital ulcer
Cancerous ulcer
D Tuberculosis
EAft Setton
In a 30-year-old patient, an ulcer with a smooth lacquered surface, red color, cartilage-like consistency was found on the lower lip. Biopsy of the affected area revealed lymphoplasmacytic infiltration, phenomena of vasculitis. What is the most likely diagnosis?
Correct answer Primary syphilis
Secondary syphilis
Congenital syphilis
Visceral syphilis
Rubber

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

40. methods: assessment of the correctness of the performance of practical skills
41. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	Evaluation criteria
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .
"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "Final lesson.(Subsection Pathomorphology of infectious diseases).Practical experience.".

Suggested topics for essays:

1.Visceral syphilis.Morphological characteristics.

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory
9.http://www.webpathology.com/- Web Pathology

Practical lesson No. 39

Topic: Final lesson.(Subsection Pathomorphology of infectious diseases).Practical

experience.

Goal:to test the ability to describe micro and macro drugs with a grant of the conclusion of the pathological process according to the topics of the final control.

Basic concepts:see above according to the topics of the final control.

Equipment:a set of macro preparations (or their images in electronic form), a set of micro preparations (or their images in electronic form), a microscope, a set of tables, a multimedia projector, a laptop

Plan: 1. Organizational activities(greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge

2.1. Frontal survey of knowledge of terminology on the topic:
emphasize the definition or provide an explanation.

2.2. block diagram on the topic as a list of didactic units of the topic;

see above according to the topics of the final control.

2.3. List of questions to check basic knowledge on the subject of the lesson.

1. Describe the micropreparation, draw a conclusion.
2. Describe the macropreparation, draw a conclusion.
3. Definition of this pathological process.
4. Complications and causes of death of this pathological process.

3.0 Formation of professional skills(mastering the skills of diagnosing a pathological process based on the description of microscopic and macroscopic changes in tissues and organs, followed by a sketch of micropreparations and a description of the macropreparation in the album):

3.1 test tasks

see above according to the topics of the final control.

3.2. algorithm for describing a macropreparation and a micropreparation

Description of macropreparation:

1. Specify the name of the organ or ego part;
2. Specify the dimensions of the body (length, width, thickness);
3. Specify the surface of the organ, the condition of the capsule, overlap;
4. Specify the consistency of the organ;
5. The type and structure of the organ at autopsy;
6. Indicate the presence of a pathological formation (if any);
7. Conclude.

Description of the micropreparation:

1. Specify the name of the body;
2. Specify the color;
3. Specify what changes in cells;
4. Conclude.

3.3. Evaluation criteria

When assessing the mastery of each topic, the student is given grades on a 4-point (traditional) scale ("2", "3", "4", "5").

Evaluation of theoretical knowledge on the subject of the lesson:

- methods: survey, solving a situational clinical problem, tests
- the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

Assessment of practical skills on the subject of the lesson:

42. methods: assessment of the correctness of the performance of practical skills
43. the maximum score is 5, the minimum score is 3, the unsatisfactory score is 2.

The grade for one practical session is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.

Rating	**Evaluation criteria**
"5"	The student is fluent in the material, takes an active part in discussing and solving situational clinical problems, tests, confidently demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, expresses his opinion on the subject of the lesson, demonstrates clinical thinking.
"4"	The applicant has a good command of the material, participates in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills during micro- and macroscopic diagnosis of pathological processes in organs and tissues according to the algorithm, with some errors, expresses his opinion on the topic of the lesson, demonstrates clinical thinking .

"3"	The applicant does not have sufficient knowledge of the material, is unsure of participating in the discussion and solution of the situational clinical problem, tests, demonstrates practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues with significant errors.
"2"	The applicant does not possess the material, does not participate in the discussion and solution of the situational clinical problem, does not demonstrate practical skills of micro- and macroscopic diagnosis of pathological processes in organs and tissues.

4. Summary:

At the end of the lesson, a general assessment based on the sum of theoretical knowledge and practical skills on the topic is presented and emphasized, with further recording in an electronic journal.

The topic of the next lesson is emphasized: "PND (final educational activity).Preparation for the exam".

5. List of recommended literature (main, additional, electronic information resources):

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p

2. Fundamentals of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.

3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

1. Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - 936p.

Electronic information resources

1.http://moz.gov.ua- Ministry of Health of Ukraine
2.www.ama-assn.org– American Medical Association /American Medical Association
3.www.who.int- World Health Organization
4.www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5.http://bma.org.uk– British Medical Association
6.www.gmc-uk.org- General Medical Council (GMC)
7.www.bundesaerztekammer.de– German Medical Association
8.http://library.med.utah.edu/WebPath/webpath.html- Pathological laboratory

9.http://www.webpathology.com/- Web Pathology