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

CONFIRMED by
Acting vice-rector for scientific and pedagogical work
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METHODOLOGICAL DEVELOPMENT TO INDEPENDENT WORK OF STUDENTS ON PATHOMORPHOLOGY

Faculty, course Stomatological, II- III
Educational discipline Pathomorphology
(name of academic discipline)

Approved:
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Plan

Topic No. 1: "Disruption of ion-osmotic and water balance, acid-alkaline state."
Topic No. 2: "Diseases of the musculoskeletal system. Parathyroid osteodystrophy, osteoporosis, Paget's disease, fibrous dysplasia, osteomyelitis, joint diseases, muscular dystrophies, myasthenia. Osteo- and cartilage tumors"
Topic #3: "Organ-specific epithelial tumors"
Topic #4: "Features of childhood tumors. Embryonic tumors. Germinogenic tumors. Teratomas and teratoblastomas. "Adult-type" tumors.
Topic #5: "Systemic vasculitis: periarteritis nodosa, Takayasu's arteritis, temporal (giant cell) arteritis, Wegener's granulomatosis, thromboangiitis obliterans, Kawasaki disease, Schoenlein-Henoch purpura, Raynaud's disease and syndrome. Sjögren's syndrome"
Topic #6: "Cerebrovascular diseases. Postresuscitation encephalopathy and brain death syndrome. Neurodegenerative (neurodystrophic) (Alzheimer's disease) and demyelinating diseases (multiple sclerosis). Neuritis (neuropathy)"
Topic #7: "Hypothalamic-pituitary disorders. Adrenal gland pathology. Pathology of the thyroid gland. Pathology of the endocrine apparatus of the pancreas"
Topic No. 8: "Clinical and morphological features of the organs of the dento-maxillofacial system and the oral cavity"
Topic #9: "Dental manifestations of other diseases"
Topic #10: "Precancerous changes and tumors of the lips, tongue, soft tissues of the oral cavity"
Topic No. 11: "Disorders of the face, neck and oral cavity"
Topic #12: "Diseases of the female and male reproductive system. Pathology of pregnancy, postpartum period and placenta. Diseases of the mammary gland"
Topic #13: "Pre- and perinatal pathology"
Topic #14: "Pathology of changes in diseases related to nutrition. Radiation sickness, drug sickness. Occupational diseases"
Topic #15: "Diseases caused by protozoa and helminths"

Topic No. 1: "Disruption of ion-osmotic and water balance, acid-alkaline state."

Goal:as a result of independent study of this topic, students should know

classification and essence of changes associated with ion-osmotic balance disorders, as well as etiology, pathogenesis, pathological anatomy of these pathological conditions. Know the possible consequences and complications of diseases associated with this pathology.

Basic concepts:

The student should know:

1. features of ion-osmotic, water balance and acid-alkaline state disturbances during dehydration;
2. peculiarities of ion-osmotic, water balance and acid-alkaline state disturbances in edema;

:The student should be able to:

1. to determine macro- and microscopic changes of internal organs in case of violation of ion-osmotic, water balance and acid-alkaline state.

Topic content:

Violation of tissue fluid content

The tissue fluid is poor in proteins (1-2% no more) and is bound in the cells with protein colloids, and in the connective tissue - with proteins and glycosaminoglycans of the main substance. Disturbances in tissue fluid content are expressed either in its increase or decrease.

Swelling or increased tissue fluid content

Edema - This is an excess accumulation of tissue fluid. This liquid, or transudate (from lat.*trans* –through, sudo, sudatum-ooze) transparent, contains no more than 2% protein.

Edema can develop in all tissues, but it is most easily visible in the subcutaneous tissue. An early clinical sign of swelling in it is pastiness - the presence of a depression (dimple) (when pressing with a finger, a dimple remains, which does not disappear after pressing). Visible swelling of the skin occurs only when a large amount of excess fluid has accumulated. In the early stages, so-called hidden swellings are detected by weighing patients. The concept of edema also includes the accumulation of fluid in reshaped (preexisting) cavities, for example, in the pleural cavity (hydrothorax, pleural effusion), the abdominal cavity (ascites), and the pericardial cavity (hydropericardium). Anasarca means massive swelling of subcutaneous tissue and internal organs, including cavities. Edema can be classified as:

limited (caused by a local violation of the native mechanism in the tissue);

general or generalized (caused by retention of sodium ions and water in the body).

In the cell, a violation of fluid distribution in generalized edema is caused by gravity, that is, fluid accumulates mostly around the lower legs in ambulatory ("walking") patients and in the lumbar region in "lying down" patients.

Local swelling

Fluid exchange through the normal capillary wall is limited and regulated by opposite forces:

capillary hydrostatic pressure directs liquid from vessels;

the osmotic colloidal pressure of the plasma pushes it back.

Normally, the difference between tissue hydrostatic and colloid osmotic pressure is close to zero and does not affect fluid exchange. The liquid passes through the capillary wall, mainly through the gaps between the endothelial cells (pores), through which only small non-protein molecules can pass (ultrafiltration). Almost all the protein remains in the vessel. The small amount of protein that leaves the capillary is quickly removed through the lymphatic vessels with a small amount of fluid that cannot return to the venule.

Types of edema:

Allergic edema: acute allergic reactions cause a local release of vasoactive substances, such as histamine, which expand the lumen of the vessels of the microcirculatory bed and cause an increase in capillary permeability. Allergic is most often localized in the skin, where it appears in the form of

blisters (hives). Rarely, large areas of the skin, larynx and bronchioles can be taken into account, recalling the narrowing of the respiratory tract (angioedema). Despite the general disorders, angioedema is best considered as a form of limited edema, because it is caused by local disorders of fluid exchange, and not by the retention of sodium ions and water in the body.

*Edema due to venous stasis:*the degree of expressiveness of venous stasis depends on the intensity of collateral venous circulation in this area. In those cases when venous stasis is accompanied by a complete failure of the drainage function of the veins, severe edema and hemorrhages develop, since the hydrostatic pressure is increased, accompanied by the rupture of capillaries (for example, massive edema and hemorrhages in orbit in cavernous sinus thrombosis). When the drainage function of the veins is partially impaired, the swelling is less pronounced (for example, on the face with obstruction of the superior vena cava). In the case of obstruction of the veins of the limbs, the swelling may sometimes be unexpressed, because the collateral circulation ensures adequate drainage function of the veins.

*Edema due to lymphatic stasis:*when the lymphatic drainage is disturbed, a small amount of protein that comes out of the capillaries by pinocytosis and as a result of ultrafiltration is not removed and accumulates in the interstitial space. Over time and with the degree of protein accumulation, the osmotic colloid pressure in the interstitial tissue increases and edema develops. Initially, lymphatic edema is loose (soft) edema. But in the distant period, the swollen tissue undergoes fibrosis and the damaged area becomes hard, dense, no depressions remain on it. Fibrosis may be accompanied by epidermal thickening and skin similar to elephant skin (elephantiasis).

General edema.

General edema occurs as a result of an increase in the total number of sodium ions and water in the body when they are retained by the kidneys, when the level of glomerular filtration is reduced or the excretion of aldosterone is increased.

*Cardiac edema:*heart failure is accompanied by a decrease in left ventricular blood output. A decrease in the release of blood into the large circle of blood circulation leads to a decrease in the filtration pressure in the glomeruli, stimulation of the juxtaglomerular apparatus and the removal of renin. Renin, in turn, stimulates the production of aldosterone (secondary aldosteronism) by angiotensin, ensuring the retention of sodium ions and water, which leads to the occurrence of general edema. If left ventricular heart failure exists for a long time, the water that is retained tends to accumulate in the lungs due to increased pulmonary venous pressure. These hydrostatic factors play a minor role in the genesis of cardiac edema compared to retention of sodium ions and water in the body, but they are important in terminating the distribution of retained fluid.

*Hypoproteinemic edema:*with hypoproteinemia, the osmotic colloid pressure of the plasma decreases. As a result of the loss of fluid in the vascular system and a decrease in plasma volume, a reflex spasm of the renal vessels occurs, which leads to hypersecretion of renin, secondary aldosteronism, retention of sodium ions and water by the kidneys, and the development of general edema.

*Renal edema:*in acute glomerulonephritis, the level of glomerular filtration is markedly reduced, which leads to the retention of sodium and water ions and the development of moderate edema. Unlike other types of general edema, with acute glomerulonephritis, swelling usually first appears in the tissues around the eyes on the eyelids, and then they spread to the hands and feet.

Other kidney diseases, which are accompanied by nephrotic syndrome and significant loss of protein in the urine, lead to hypoproteinemia and are accompanied by massive general edema.

The value of general edema

In most cases, edema does not primarily cause any dysfunctions of parenchymal cells. Severe and chronic swelling of the skin can disrupt wound healing and increase susceptibility to infections. Swelling of internal organs is often manifested by various symptoms, for example, swelling of the liver in acute hepatitis or heart failure is manifested by pain caused by stretching of the liver capsule. Swelling of some organs is life-threatening.

*Pulmonary edema:*the pulmonary circulation functions at a low hydrostatic pressure (for the pulmonary artery, the systolic pressure is 20 mm Hg). When it becomes higher than the colloid-osmotic pressure of the plasma, a small amount of fluid leaves the pulmonary capillaries. The outflow

of fluid from the pulmonary capillary of the alveoli is called pulmonary edema. Lungs with edema are heavy, enlarged in size, acquire a dough-like consistency, a large amount of transparent, pink liquid with fine foam flows from the cut surface. Swollen fluid first accumulates in the interstitial tissue (stroma), and then in the alveoli. Edema disrupts gas exchange in the lungs and in severe cases, causes hypoxia and death.

*Edema of the brain:*cerebral edema is observed in a wide variety of brain injuries, for example, traumatic injury, infectious diseases, neoplasms, and vascular disorders. The liquid collects mainly in the extracellular space of the white matter. Swollen fluid physically breaks nerve connections, causing acute brain disorders that pass over time. Increased intracranial pressure leads to headache and swelling of the optic disc of the optic nerve (papilloedema). The brain is enlarged, the subarachnoid spaces and ventricles are expanded and filled with clear fluid. Edema of the brain is often combined with its swelling, which mostly dominates. When the brain swells, there is a sharp hydration of its substance, the gyri are smoothed, the cavities of the ventricles are reduced. With cerebral edema, fluid accumulates around vessels and cells (perivascular and pericellular edema), with swelling of the brain, swelling of astrocytes, destruction of glial fibers, and breakdown of myelin are noted. There is an increase in intracerebral and intracranial pressure. A significant increase in pressure can displace the temporal region down into the foramen of the cerebellar tent (herniation of the tent) or displace the tonsils of the cerebellum into the large occipital foramen (tonsillar hernia), which can cause death by compression of the vasomotor center in the brainstem. Immediate help is needed: the infusion of mannitol removes fluid from the brain. High-dose corticosteroids are also effective in reducing brain swelling. There is an increase in intracerebral and intracranial pressure. A significant increase in pressure can displace the temporal region down into the foramen of the cerebellar tent (herniation of the tent) or displace the tonsils of the cerebellum into the large occipital foramen (tonsillar hernia), which can cause death by compression of the vasomotor center in the brainstem. Immediate help is needed: the infusion of mannitol removes fluid from the brain. High-dose corticosteroids are also effective in reducing brain swelling. There is an increase in intracerebral and intracranial pressure. A significant increase in pressure can displace the temporal region down into the foramen of the cerebellar tent (herniation of the tent) or displace the tonsils of the cerebellum into the large occipital foramen (tonsillar hernia), which can cause death by compression of the vasomotor center in the brainstem. Immediate help is needed: the infusion of mannitol removes fluid from the brain. High-dose corticosteroids are also effective in reducing brain swelling.

*Edema of serous cavities:*the accumulation of swollen fluid within the pericardial sac and pleural cavity can disrupt the normal functioning of the heart and expansion of the lungs. Fluid, accumulating in the abdominal cavity (ascites), stretches the abdominal wall and does not significantly disrupt the normal function of the abdominal organs.

Dehydration of the bodymay be due to various reasons: dehydration due to burns, dehydration due to intestinal infections, etc.

Let's focus on dehydration in cholera and follow the changes in the body using the example of this disease.

Cholerogen exotoxin activates the adenyl cyclase enzyme in epithelial cells, as a result of which the synthesis of cyclic 3-5 adenosine monophosphate (3-5 AMP) increases, the level of which determines the volume of intestinal juice secretion. As a result, the mucous membrane of the small intestine begins to secrete a large amount of isotonic fluid, which does not have time to be absorbed into the large intestine. Diarrhea occurs, and later vomiting. The fluid secreted by the small intestine has little protein (less than 200 mg per 10 ml) and the following electrolyte concentrations: sodium 140 mmol/l, potassium 13 mmol/l, bicarbonate 32 mmol/l, chloride 92 mmol/l. An adult suffering from cholera loses more than 1 liter of fluid per hour. Acute extracellular isotonic dehydration develops, which is accompanied by a decrease in the mass of circulating blood and hemoconcentration, which leads to hemodynamic disorders and disruption of tissue metabolism. Thickening of blood, increase in its viscosity, slowing of blood flow, disturbance of peripheral blood flow, tissue hypoxia, accumulation of deoxidized metabolic products lead to the development of acute kidney failure, impaired cardiac function of the brain and other organs, as well as blood coagulation

processes (increased fibrinolytic and anticoagulant activity blood). Diarrhea and vomiting lead not only to hypovolemia, but also to salt deficiency, primarily potassium. Loss of potassium during cholera can reach 1/3 of its content in the body. Potassium deficiency is accompanied by sharp muscle weakness (myocardial dysfunction, damage to renal tubules) tissue hypoxia, the accumulation of deoxidized metabolic products lead to the development of acute kidney failure, impaired cardiac function of the brain and other organs, as well as blood coagulation processes (increased fibrinolytic and anticoagulant activity of blood). Diarrhea and vomiting lead not only to hypovolemia, but also to salt deficiency, primarily potassium. Loss of potassium during cholera can reach 1/3 of its content in the body. Potassium deficiency is accompanied by sharp muscle weakness (myocardial dysfunction, damage to renal tubules) tissue hypoxia, the accumulation of deoxidized metabolic products lead to the development of acute kidney failure, impaired cardiac function of the brain and other organs, as well as blood coagulation processes (increased fibrinolytic and anticoagulant activity of blood). Diarrhea and vomiting lead not only to hypovolemia, but also to salt deficiency, primarily potassium. Loss of potassium during cholera can reach 1/3 of its content in the body. Potassium deficiency is accompanied by sharp muscle weakness (myocardial dysfunction, damage to renal tubules) Diarrhea and vomiting lead not only to hypovolemia, but also to salt deficiency, primarily potassium. Loss of potassium during cholera can reach 1/3 of its content in the body. Potassium deficiency is accompanied by sharp muscle weakness (myocardial dysfunction, damage to renal tubules) Diarrhea and vomiting lead not only to hypovolemia, but also to salt deficiency, primarily potassium. Loss of potassium during cholera can reach 1/3 of its content in the body. Potassium deficiency is accompanied by sharp muscle weakness (myocardial dysfunction, damage to renal tubules)

There are 4 degrees of body dehydration: 1- loss of fluid in the volume of 1-3% of body weight, 2- loss of 4-6%, 3- loss of 7-9%, 4- loss of fluid in the volume of 10% of body weight and more.

Pathomorphological changes in organs and tissues are different depending on the clinical form of cholera. Those who died from cholera algide, as a result of sharp dehydration and demineralization, have a characteristic "Hippocrates face" with sunken eyes, sharpened features, earthy skin color, which sometimes acquires a cyanotic shade. The convulsive contraction of the muscles of the limbs gives the corpse a characteristic "boxer" pose. There is wrinkling and cyanosis of the skin, especially of the fingers ("washing hands"). Corpse spots are red-purple. On the cut, the skin, subcutaneous tissue and muscle tissue are dense, the protruding blood has a tar-like consistency, reminiscent of currant jelly. The intestine is filled with liquid that looks like cloudy soup. Redistribution of blood, accumulation of blood in large veins and desolation of the capillary network are noted in the dead. Centers of ischemia and centers of stasis capillary full blood are determined at the same time. This is especially characteristic of the gastrointestinal tract, lungs, and liver. Dystrophic changes are often found in the myocardium and liver. The kidneys are reduced, their capsule is easily removed, the glomeruli are filled with blood, fatty and vacuolar tubule dystrophy is noted. Acute dehydration of the body can lead to hypovolemic shock.

Acid-alkaline state.

The acid-alkaline state - the ratio between substances that have an acidic and alkaline reaction - is of essential importance in the vital activity of the body. The result of this balance is the constancy of the blood pH value of 7.36-7.44. Only within these limits can cells actively function and their enzyme systems work. Under conditions of normal life, the body is exposed to the effects of toxic or basic compounds in connection with the intake of various foods and the formation of metabolic products, changes in the work of excretory systems. The regulation of the acid-base state is supported by powerful systems - chemical and physiological, and their regulation. Chemical regulation of the acid-base state is carried out by the buffer systems of blood and tissues, which return it to the normal range. Physiological regulation of the acid-base state is carried out primarily by the lungs and kidneys. When carbon dioxide accumulates in the blood, the lungs release its excess, and when it decreases, they reduce its release. The role of the kidneys in regulating the acid-base state is that when acidic compounds accumulate in the body, the kidneys retain basic compounds and release acidic ones, and when basic compounds accumulate, they retain acidic compounds and release basic ones. With

excessive accumulation of basic and acidic compounds, as well as with a violation of the mechanisms of the acid-base state, its disturbances occur. Violation of the acid-alkaline state due to the appearance of an excess of acidic products is called acidosis, due to an excess of alkaline products - alkalosis. Acidosis occurs when carbon dioxide and carbonic acid accumulate in the body as a result of impaired external breathing or an increase in the level of carbon dioxide in the environment. This form of acidosis is called gas acidosis. Non-gaseous acidosis occurs with metabolic disorders, diabetes, hypoxia, starvation, kidney failure, acid poisoning. Severe acidosis is life-threatening. At first, acidosis increases breathing, and later causes its oppression, disorder of nervous activity, up to acidotic coma. Alkalosis is also divided into gaseous and non-gaseous alkalosis. Gas alkalosis develops as a result of increased release of carbon dioxide from the body through the lungs during their hyperventilation. Non-gaseous alkalosis is metabolic, occurs when a large amount of alkaline substances, vegetable food, medicines are introduced into the body, loss of acidic products during vomiting, toxicosis of pregnancy, poisoning. An uncompensated shift of the acid-alkaline state in one direction or another usually has serious consequences for the course of biochemical processes in the body as a whole. With alkalosis, the tone of blood vessels decreases, the volume of circulating blood decreases, blood flow decreases, which leads to severe disorders of nervous activity, suppression of the activity of the heart and lungs.

1. Theoretical questions

Questions for self-control
1 General idea about edema
2 Local edema
3 General swelling
4 Cardiac edema
5 Hypoproteinemic edema
6 Renal edema
7 The value of total swelling
8 Meaning of dehydration
9 Organ changes during dehydration.

2 Practical tasks

1. Prepare an essay on the topic: "The role of ions in maintaining osmotic pressure"

2. Make a graph of the logical structure "Types of edema".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

1 Pathomorphology: nats. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.
2 The basics of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.
3 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

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.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
http://www.webpathology.com/- Web Pathology

Topic No. 2: "Diseases of the musculoskeletal system. Parathyroid osteodystrophy, osteoporosis, Paget's disease, fibrous dysplasia, osteomyelitis, joint diseases, muscular dystrophies, myasthenia. Osteo- and cartilage tumors"

Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases of the musculoskeletal system at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:

The student should know:

1. Classification and essence of changes associated with diseases of the musculoskeletal system.
2. **Etiology, pathogenesis, pathological anatomy of these pathological conditions.**
3. Outcomes, complications of diseases associated with diseases of the musculoskeletal system.

:The student should be able to:

1. Classify diseases of the musculoskeletal system.
2. To characterize the etiology, pathogenesis and morphological essence of these diseases

Topic content:

Diseases of the musculoskeletal system

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:
Monoosseous - pathological changes occur in only one bone. It can develop at any age.
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).
Complications are 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.
Complications: 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.
Complications: 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 is one of the most frequent joint diseases 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:

1 Pain in the joints during exercise, narrowing of the joint space and osteophytes (radiologically) are noted.
2 Pain in the joints becomes constant, the narrowing of the joint space and the development of osteophytes are more pronounced.
3 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 are 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:

1. 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.
2. 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).
3. 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.

The death of patients with a severe course of progressive muscular dystrophy occurs from 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. Complications arise when the respiratory muscles are damaged. An inadequate response of the lungs leads to the development of pneumonia and asphyxia, which, as a rule, become the cause of death.

1. Theoretical questions

Questions for self-control

1. Describe the macro- and microscopic changes in the human body during parathyroid dystrophy,
2. Describe macro- and microscopic changes in the human body during primary hematogenous osteomyelitis,
3. Describe the macro- and microscopic changes in the human body during fibrous dysplasia,
4. Describe the macro- and microscopic changes in the human body during osteopetrosis,
5. Describe the macro- and microscopic changes in the human body during Paget's disease,
6. Describe the macro- and microscopic changes in the human body during osteoarthritis,
7. Describe the macro- and microscopic changes in the human body during progressive muscular dystrophy,
8. Describe the macro- and microscopic changes in the human body during myasthenia gravis.

2 Practical tasks

1. Prepare an essay on the topic: "Disruption of the parathyroid gland"

2. Make a graph of the logical structure "Types of bone tissue diseases".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

4 Pathomorphology: nats. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.
5 The basics of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.
6 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

Electronic information resources

9 http://moz.gov.ua- Ministry of Health of Ukraine
10 www.ama-assn.org– American Medical Association /American Medical Association
11 www.who.int- World Health Organization
12 www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine

13 http://bma.org.uk– British Medical Association
14 www.gmc-uk.org- General Medical Council (GMC)
15 www.bundesaerztekammer.de– German Medical Association
16 http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
http://www.webpathology.com/- Web Pathology

Topic #3: "Organ-specific epithelial tumors"

Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:

The student should know:

1. definition of the concept of "tumor";
2. morphological characteristics of tumors, signs of morphological atypism;
3. principles of tumor classification;
4. types of epithelial tumors according to existing principles of classification;
5. about the biological essence of tumor growth.

:The student should be able to:

1. to determine the morphological features of various types of atypism;
2. determine the type of tumor growth;
3. distinguish between mature and immature tumors on the basis of tissue and cellular atypism, the nature of tumor growth, in relation to the surrounding tissues, explain the probable cause and mechanism of development;
4. on the basis of morphological signs, be able to distinguish benign and malignant tumors from flat and iron epithelium;
5. explain the features of metastasis of malignant tumors from the epithelium;
6. determine the localization of possible metastases of malignant epithelial tumors according to the ways of its metastasis;
7. interpret modern concepts of carcinogenesis of malignant and benign tumors;
8. interpret the morphogenesis and histogenesis of tumors.

Topic content:

1. Morphological features of epithelial tumors without specific localization: benign (papilloma, adenoma) and malignant (cancer). Tumors of this type develop from flat or glandular epithelium, which does not perform any specific function.

Papilloma is an epithelial tumor of flat or transitional epithelium. Tissue atypism is manifested in uneven development of epithelium and stroma with excessive formation of small blood vessels.

Adenoma is a tumor of glandular organs and mucous membranes lined with prismatic epithelium. Adenomas have an organoid structure, consist of cells of prismatic or cubic epithelium, which forms iron formations, sometimes with papillary growths. If the stroma predominates, it is called a fibroadenoma. Epithelium preserves complexity and polarity, located on its own membrane. Types

of adenomas: 1. acinar (growing from the alveolar parenchyma); 2. tubular (growing from ducts of glands); 3. trabecular (has a beam structure); 4. papillary (in the form of papillary formations); 5. adenomatous polyp.

Malignant, immature tumors from the epithelium - cancer. Cancer usually has the appearance of a nodule of soft or dense consistency with indistinct borders. A cloudy liquid - cancer juice - separates from the cut surface of the tumor. Sharply expressed tissue and cellular atypism is determined microscopically.

2. Histological variants of cancer: Microscopic forms of cancer: 1. "cancer in place"; 2. squamous cell carcinoma with and without keratinization; 3. adenocarcinoma; 4. mucous (colloid); 5. solid; 6. small cell; 7. fibrous (skin); 8. medullary (adenogenic).

1. "Cancer in place" - the initial form of cancer without invasive growth, but with pronounced atypism. This is only the initial stage of cancer growth. Then it becomes infiltrating.

2. Squamous cell cancer consists of strands of atypical epithelial cells that grow into the underlying tissue. Tumor cells can retain the ability to keratinize and then "cancer pearls" appear.

3. Adenocarcinoma (glandular cancer) develops from the prismatic epithelium of mucous membranes and glandular epithelium. Its histological structure resembles an adenoma, but in contrast to it, there is a sharply expressed tissue and cellular activity. Tumor cells form iron formations that grow into the surrounding tissue, destroying it.

4. Mucous (colloid cancer). The tumor has the appearance of a colloidal mass. Consists of atypical cells.

5. Solid cancer grows in the form of trabeculae separated by layers of connective tissue. It grows quickly and metastasizes.

6. Small cell cancer is a form of undifferentiated cancer that consists of monomorphic lymphocyte-like cells that do not form any structures. There is little stroma. Growth is fast. Early metastases.

7. Fibrous cancer (skin) is a form of undifferentiated cancer that consists of atypical hyperchromic cells located among the layers and strings of the stroma, which prevails over the parenchyma. The tumor is highly malignant. There are often early metastases.

8. Medullary - undifferentiated cancer. The main quality is the superiority of parenchyma over stroma. The tumor is soft. It consists of atypical cells, has a large number of metastases, grows quickly, gives early and numerous metastases.

3. Tumors of exo- and endocrine glands and epithelial coverings (organ-specific) are characterized by the fact that they develop from cells of a certain organ and retain morphological and sometimes functional features.

Liver. Benign organ-specific tumor from hepatocytes - adenoma from liver cells (hepatoadenoma, hepatoma). Malignant hepatocellular (hepatocellular) cancer. The tumor consists of atypical

hepatocytes that form irregularly arranged trabeculae of an irregular shape. The stroma is weakly expressed.

Kidneys Benign organ-specific tumors - various adenomas, malignant - renal cell cancer.

Skin. Benign tumors: syringoadenoma (from the epithelium of sweat gland ducts); hydroadenoma (from the epithelium of the secretory departments of sweat glands); trichoepithelioma (from hair follicles). Malignant tumors: basal cell cancer, sweat gland cancer, sebaceous gland cancer, hair follicle cancer.

Mammary gland. Benign tumors: fibroadenoma (pericanalicular, intracanalicular). Organ-specific breast cancer includes: non-infiltrating intralobular and intraductal cancer: Paget's disease (nipple and areola cancer).

Uterus. Organ-specific epithelial tumors of the uterus are destructive (malignant) cystic drift and chorionepithelioma (chorionic carcinoma). Insertion of the bladder is manifested by the ingrowth of chorionic villi into the veins of the uterus and pelvis. Chorionepithelioma develops from the epithelium of the villi of the chorion and consists of light Langhans cells and syncytium cells. There is no stroma in the tumor. The function of blood vessels is performed by cavities formed by tumor cells. Gives hematogenous metastases.

Tumors of salivary glands and organs of the oral cavity. Adenomas of the salivary glands are quite common. They consist of epithelial structures in combination with fibrous, mucous, cartilaginous, as well as bone-like structures. They are benign, can recur, turn into cancer.

Odontoma develops from the enamel organ, consists of dense tooth tissues (mainly dentine). There are soft and hard odontomas. Benign flow. Relapses are possible.

Ameloblastoma develops from the epithelium of the enamel organ. A tumor of a soft consistency, consisting of delicate connective tissue and passages lined with cubical and prismatic epithelium, may have a cystic structure. It proceeds benignly, tends to give relapses. Localization - jaw bones.

4. General doctrine about tumors.

A tumor, neoplasm, blastoma is a pathological process characterized by uncontrollable cell proliferation. The growth and reproduction of cells in a tumor differs from the growth and reproduction of cells in other processes (inflammation, regeneration, hyperplasia, organization). Tumor cells are characterized by a state of anaplasia or cataplasia - partial loss of cell differentiation factors (due to biochemical rearrangement of DNA).

Most tumors have an organoid structure and consist of parenchyma and stroma. Tumors that consist of one tissue are called histoid. Parenchyma for them is specifically functioning tumor cells, and stroma - fibrous structures, amorphous substance, lymphatic and blood vessels, nerves.

If the tumor parenchyma cells are sufficiently differentiated and resemble the parent tissue, the tumor is called homologous, mature, benign. If the parenchyma cells of the tumor are poorly differentiated

and do not resemble the parent tissue, then such a tumor is called heterotypic, immature, malignant.
The morphological structure of tumors differs from normal tissues. The set of features that distinguish them is called atypical. Tissue and cellular atypism are distinguished: biochemical; antigenic atypism.
Tissue atypism consists of: 1. incorrect quantitative ratio of tumor stroma and parenchyma; 2. the presence in the tumor of various sizes and number of vessels; 3. incorrect, chaotic fiber direction; 4. weakening of the collagenization process; 5. discomplexation and formation of irregular structures.
Cellular atypism consists of: 1. cellular polymorphism, violation of the nuclear cytoplasmic index; 2. increase in the number and size of nuclei; 3. the presence of various cellular inclusions (protein grains, glycogen); 4. the presence of pathological figures of mitoses (multipolar, asymmetric, hyper- and hypochromic, abortive); 5. unusual number of chromosomes, chromosomal aberration; 6. presence of multinucleated giant cells; 7. close topographic contacts between the nuclear membrane and the mitochondrial membrane, between the membrane and the endoplasmic reticulum; 8. loss of membrane-forming and covering properties of the epithelium.
The atypicality of ultrastructures is manifested in an increase in the number of ribosomes, which can lie freely in the form of rosettes and chains, the shape, size and location of mitochondria are changed. Abnormal mitochondria appear. The cytoplasm is poor, the nucleus is large with a diffuse or marginal arrangement of chromatin. Membrane contacts of the nucleus, mitochondria and endoplasmic reticulum appear in large numbers. Hybrid cells appear.
According to the nature of growth, tumors are distinguished as expansively growing and infiltrating. Expansively growing tumors retain syncytial connections between tumor sites. With such growth, the tumor has a clear border that separates it from the tissues of the body.
Expansive growth is characteristic of benign, mature tumors. Appositional growth of a tumor occurs through neoplastic transformation of normal cells into tumor cells.
Infiltratively growing tumors (invasive growth) are characterized by deep penetration of tumor cells into the underlying tissue. This growth is a consequence of weak syncytial connections between tumor cells and is characteristic of malignant, immature tumors.
Exophytic growth of tumors - external growth or into the cavity of the organ (characteristic of mature tumors). Endophytic growth - the growth of a tumor deep into an organ or into its wall (more typical for immature tumors).
Clinically, tumors are divided into benign, malignant and tumors with local destructive growth. Signs of immature, malignant tumors: 1. sharply expressed anaplasia and atypism; 2. a lower degree of differentiation compared to mature tumors; 3. weak syncytial connections between tumor cells; 4. fast, infiltrating, invasive, endophytic growth; 5. tumors can recur and metastasize.
Metastasis of a tumor is manifested in the fact that tumor cells enter other places through embolism and begin to multiply there, forming daughter nodes (metastases). There are hematogenous

(characteristic of sarcomas), lymphogenous (characteristic of cancers), and mixed metastases. Most often, in a metastasis, the tumor has the same structure as in the main node. However, tumor cells in metastases can change, and then it is difficult to establish the nature and localization of the primary node based on the histological structure of the metastasis. Metastases, as a rule, grow faster than the primary node. The time of development of metastasis can be different.

Recurrence of a tumor is the appearance of a tumor in the same place where it was removed surgically or by radiation. It is possible from cancer cells that remained in the wound, as well as from nearby metastases. In addition, it should be taken into account that when removing a tumor, not the cause is eliminated, but the result.

The effect of a tumor on the body can be local and general. Local influence: compression or destruction of an organ or its part. The general effect is characteristic of malignant tumors: metabolic disorders, the development of cachexia, some tumors are hormonally active.

1. Theoretical questions

Questions for self-control

1. Morphological features of epithelial tumors without specific localization: benign (papilloma, adenoma) and malignant (cancer).
2. Histological variants of cancer.
3. Tumors of exo- and endocrine glands and epithelial covers.
4. Nomenclature of epithelial tumors.
5. General doctrine about tumors.
6. Features of tumor growth, types of atypism, features of mature and immature tumors, structure of epithelial tumors.

2 Practical tasks

1. Prepare an abstract on the topic: "Morphological features of epithelial tumors"

2. Make a graph of the logical structure "Classification of epithelial tumors".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

7 Pathomorphology: nats. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.

8 The basics of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.

9 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

Additional:

1. Pathological anatomy: a textbook. Trans. from Russian 4th edition / Strukov A.I., Serov V.V. - Kharkiv.: Fakt, 2004. - 864 p.
2. Skrypnikov P.M., Skrypnikova T.P., Shynkevich V.I., Tovma V.V. Individual and age-related clinical and morphological features of the mucous membrane of the oral cavity: study. manual - Poltava: LLC "ASMI", 2016. - 102 p.

Electronic information resources

17 http://moz.gov.ua- Ministry of Health of Ukraine
18 www.ama-assn.org– American Medical Association /American Medical Association
19 www.who.int- World Health Organization
20 www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
21 http://bma.org.uk– British Medical Association
22 www.gmc-uk.org- General Medical Council (GMC)
23 www.bundesaerztekammer.de– German Medical Association
24 http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
http://www.webpathology.com/- Web Pathology

Topic #4:"Features of childhood tumors. Embryonic tumors. Germinogenic tumors. Teratomas and teratoblastomas. "Adult-type" tumors.

Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:

The student should know:

1. The classification of pdeviation in children.

1 Etiology, pathogenesis, pathological anatomy of the most common of them.

2. Prognosis, complications of these diseases.

:The student should be able to:

1. To interpret the modern classification of childhood tumors.

1 To characterize the etiology of pdeviation in children.

2 To characterize the features of tumor growth in children.

3 To characterize the specificity of metastasis of malignant tumors in children.

4 Explain the peculiarities of tumor growth in children compared to adults.

5 Explain the morphological features of dysontogenetic tumors.

2. Determine the morphological features of teratoma and teratoblastoma.

Topic content:

1. Features of malignant tumor growth in children.

The study of childhood tumors made it possible to clarify a number of features that distinguish these tumors from adults. The following differences are distinguished: - a special frequency of certain types of tumors in children, which differ from such types of adults; - dependence of tumor growth on

the age of children; - the dependence of the occurrence of tumors in children on exogenous influences; - dependence of the occurrence of tumors on postnatal influences; - relationship between tumors and developmental defects and chromosomal syndromes; - the importance of heredity and family predisposition.

A special place is occupied by a completely unusual phenomenon with malignant tumors in children, which contradicts the idea of tumor progression. It consists in the fact that malignant tumors in children are capable of so-called maturation with the transformation of neuroblastoma into ganglioneuroma and even ganglioneurofibroma, teratoblastoma into teratoma. This phenomenon has yet to be explained, but it is noteworthy that it is observed in tumors that develop from embryonic tissues that are delayed in development compared to other tissues of the body - the tumor carrier, or from undifferentiated stem cells that are preserved in childhood in the form of a cambium .

Metastasis also has its own characteristics. Thus, most malignant tumors of the central nervous system do not metastasize beyond the skull, but only have a tendency to spread through adjacent tissues and natural channels - the course of the meninges, along the cerebrospinal fluid. In rare cases, extracranial metastases can be observed, and not only when this was preceded by surgery. Despite the fact that most malignant tumors in children are sarcomas, they primarily metastasize to regional lymph nodes, for example, rhabdomyoblastomas of the urogenital tract metastasize to pelvic lymph nodes, as well as Williams tumor, neuroblastoma, and malignant lymphoma. Thus, 1/3 or 1/2 of soft tissue sarcomas of children's type spread lymphogenically, which contradicts the classic idea of their hematogenous metastasis. Along with this, embryonic hematoblastomas often metastasize not to the regional lymph nodes, but to the lungs. Hematogenous metastases in the lungs are also observed in medulloblastomas, retinoblastomas, embryonal nephroblastomas, teratoblastomas, and mixed teratomas. Bone metastases are relatively rare in children, they can be found in neuroblastomas, which, as a rule, do not metastasize to the lungs. Embryonic neuroblastoma is characterized by sprouting of large venous trunks with the formation of thrombosis and thromboembolism in the small circle of blood circulation. Thus, malignant tumors in children are distinguished by a significant peculiarity of metastasis, the features of which must be known to a pediatric oncologist and a pathologist. retinoblastomas, embryonal nephroblastomas, teratoblastomas and mixed teratomas. Bone metastases are relatively rare in children, they can be found in neuroblastomas, which, as a rule, do not metastasize to the lungs. Embryonic neuroblastoma is characterized by sprouting of large venous trunks with the formation of thrombosis and thromboembolism in the small circle of blood circulation. Thus, malignant tumors in children are distinguished by a significant peculiarity of metastasis, the features of which must be known to a pediatric oncologist and a pathologist. retinoblastomas, embryonal nephroblastomas, teratoblastomas and mixed teratomas. Bone metastases are relatively rare in children, they can be found in neuroblastomas, which, as a rule, do not

metastasize to the lungs. Embryonic neuroblastoma is characterized by sprouting of large venous trunks with the formation of thrombosis and thromboembolism in the small circle of blood circulation. Thus, malignant tumors in children are distinguished by a significant peculiarity of metastasis, the features of which must be known to a pediatric oncologist and a pathologist. Embryonic neuroblastoma is characterized by sprouting of large venous trunks with the formation of thrombosis and thromboembolism in the small circle of blood circulation. Thus, malignant tumors in children are distinguished by a significant peculiarity of metastasis, the features of which must be known to a pediatric oncologist and a pathologist. Embryonic neuroblastoma is characterized by sprouting of large venous trunks with the formation of thrombosis and thromboembolism in the small circle of blood circulation. Thus, malignant tumors in children are distinguished by a significant peculiarity of metastasis, the features of which must be known to a pediatric oncologist and a pathologist.

2. Classification of childhood tumors.

Classifying tumors in children is much more difficult than in adults, because it is difficult and sometimes impossible to apply a single histogenetic principle. Thus, organoid and organosmoid teratomas and teratoblastomas consist of tissue elements of all three germ layers, which makes it impossible to use the histogenetic principle. Some histoid teratomas, progonomas, choristomas and so-called mixed tumors can be classified according to their belonging to one or another germ layer, thereby establishing their origin from tissue elements of the early prenatal period, or if it is impossible to establish their belonging to one or another layer, by topography tumors It is appropriate to divide tumors in children into two main types:

1 tumors that develop from embryonic tissues, derivatives of individual or all germ leaves;

2 tumors that develop from differentiated mature tissues.

Teratomas and some hemartomas and hemartoblastomas should be classified by localization, because it determines the nature of the course of oncological diseases.

Thus, the classification of typical tumors in children requires the use of three principles: origin from one or another germ layer, histogenesis (from mature tissues), localization. In turn, all tumors should be divided into benign and malignant, using the term "blastoma" to determine malignancy.

TUMORS OF THE NERVOUS SYSTEM

1 medulloblastoma, astrocytoma, oligodendroma, chorionic papilloma, craniopharyngioma;

2 meningeal tumors: microglioma, melanoma, retinoblastoma;

3 neuroblastoma, ganglioneuroma, ganglioneuroblastoma.

Teratomas. TERATBLASTOMA

Teratomas: sacrococcal and retroperitoneal teratomas, intrathoracic teratomas, germinogenic tumors of the ovaries and testicles, endodermal sinus tumors, testicular teratomas, testicular

tumors.

KIDNEY TUMORS

- nephroblastoma;
- mesoblastic nephroma.

TUMORS OF THE ENDOCRINE GLANDS

- tumors of the thyroid gland;
- tumors of the islet tissue of the pancreas;
- tumors of the cortical substance of the adrenal glands.

TUMORS OF SOFT TISSUES

- embryonal rhabdomyosarcomas;
- fibrosarcoma;
- fibromatosis

TUMORS OF VASCULAR TISSUE

- hemangiomas;
- hemangiosarcoma;
- tumors from the lymph nodes.

TUMORS OF BONE TISSUE

- malignant (osteosarcoma, chondrosarcoma, Ewing's sarcoma)
- benign (osteoidosteoma, osteoidochondroma, chondromyxoid fibroma).

3. Dysontogenetic tumors: hamartomas and hamartoblastomas.

Hamartoma is a tumor from embryonic tissue that has delayed its differentiation compared to the tissues of the tumor carrier, which develops from excessively disproportionately developed tissue complexes. Tohamartin children include angiomas, nevi, embryonic tumors of internal organs, embryonic tumorsmesodermal and mesenchymalhistogenesis - benign and malignant. Hamartomas andhamartoblastomaof vascular origin: two are observed in childrenbasictypes - capillary and cavernous hemangioma. Capillary hemangioma is a benign tumor that has the appearance of a reddish lobular nodule, localized mainly in the skin and liver. It consists of capillaries with 2-3 layersendothelium, tumor withproliferatingcapillaries, with an infiltrative nature of growth,often relapses, metastases does not give. Cavernous hemangioma -benigna tumor, more often a vascular malformation. It has the appearance of a blue-purple spongy node on the cut. It consists of vascular cavities, separatedconnective tissuepartitions,fulfilledby blood Angiosarcoma -malignanttumor, dense, hilly,infiltratesin the surrounding tissues, with focihemorrhages, necrosis and cysts with blood content. Consists ofheavierpolymorphic spindle-shaped cells, among which there are cavities and slits resembling blood vessels, filled with plasma and blood.

Hamartomas andstriatum hamartoblastomamuscle tissue:rhabdomyoma- benign tumor from embryonic muscle cells. Localized inheartsand muscles of the limbs in the form of a node up to 10-15 cm, brown in color. It consists of cells that resemble embryonic cellsembryoblasts, with a large amount of glycogen in the cytoplasm. Rhabdomyoblastoma is a malignant tumor, from embryonic rudiments of muscle tissue that have split off. It is made of densely or loosely arranged cells with bipolar processes and muscle fibers with a longitudinal line. Localization: pelvic organs. It metastasizes to regional lymph nodes and hematogenously to the lungs.

Nephroblastoma is a malignant kidney tumor that grows for a long timeexpansively,within the capsule, squeezing the kidney tissue. Pinkish-white in section, cangryappearance with focihemorrhagesLater, the tumor grows into the surrounding tissues and metastasizes hematogenously in the lungs.

4. Teratomas and teratoblastomas - morphological manifestations.

Teratomas have a certain localization typical for them: ovaries, testicles, sacrococcygeal region, mediastinum, retroperitonealspace,pharynx, base of the skull. They reach large sizes, in fetuses they complicate the course of childbirth. They consist of structures resembling organs and various tissues. Malignant teratoblastomas are less common when growth predominates alongside mature tissuecarcinoma-likesolid or capillary structures. Sacral-coccygeal teratoblastomas growquickly,metastasize to inguinal and pelvic lymph nodes, lungs and liver. Abdominal and mesenteric teratomas, which appear from birth or at the age of 1-3 years, reach large sizes, are more common in girls, are located closer to the diaphragm, are usually benign.

1. Theoretical questions

Questions for self-control

1. **Modern classification of childhood tumors.**
2. **To characterize the etiology of pdeviation in children.**
3. **To characterize the features of tumor growth in children.**
4. **To characterize the specificity of metastasis of malignant tumors in children.**
5. **Explain the peculiarities of tumor growth in children compared to adults.**
6. **Explain the morphological features of dysontogenetic tumors.**
7. Determine the morphological features of teratoma and teratoblastoma.

2 Practical tasks

1. Prepare an essay on the topic: "Morphological features of tumors in children"

2. Make a graph of the logical structure "Classification of childhood tumors".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

10 Pathomorphology: nats. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.

11 The basics of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.

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Additional:

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Electronic information resources

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

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27 www.who.int- World Health Organization

28 www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine

29 http://bma.org.uk– British Medical Association

30 www.gmc-uk.org- General Medical Council (GMC)

31 www.bundesaerztekammer.de– German Medical Association

32 http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory

http://www.webpathology.com/- Web Pathology

Topic #5: "Systemic vasculitis: periarteritis nodosa, Takayasu's arteritis, temporal (giant cell) arteritis, Wegener's granulomatosis, thromboangiitis obliterans, Kawasaki disease, Schoenlein-Henoch purpura, Raynaud's disease and syndrome. Sjögren's syndrome"

Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:

The student should know:

1. criteria for morphological assessment of vasculitis;
2. topography and prevalence of changes in the vascular wall in systemic vasculitis;
3. principles of classification of systemic vasculitis;
4. macro- and microscopic assessment of systemic vasculitis;
5. course, complications and outcome of the disease on systemic vasculitis.

:The student should be able to:

1. to conduct a macro- and microscopic examination of the vascular wall in systemic

vasculitis;

2. systematize the main morphological features of each type of vasculitis.

Topic content:

Vasculitis

Vasculitis - diseases characterized by inflammation and necrosis of the vessel wall; the process can be local or systemic. Local vasculitis occurs in the foci of inflammation as a result of the transition of the process to the vessel wall from adjacent tissues (eg, purulent-necrotic vasculitis in phlegmon). Systemic vasculitis, which can be the basis of independent diseases (primary vasculitis) or a manifestation of any other disease (secondary vasculitis), is characterized by a widespread weighing of vessels.

SYSTEMIC VASCULITIS

Systemic vasculitis is of primary importance among human diseases with damage to blood vessels. Morphological evaluation criteria:

3 the type of inflammatory reaction that determines the nature of vasculitis;
4 depth of damage to the vascular wall;
5 topography and prevalence of changes in the vessel wall;
6 the nature of organ pathology in connection with damage to blood vessels.

Depending on the type of inflammatory reaction, the preference of alterative-exudative or productive changes, vasculitis is divided into necrotic (destructive), destructive-productive, productive, and granulomatous. Taking into account the depth of damage to the vascular wall: i.e. involvement in the inflammatory process of its inner, middle, outer membrane, endovasculitis, mesovasculitis, perivasculitis are distinguished, and in the case of combined damage to the membranes - panvasculitis. The vast majority of vasculitis in the system is characterized by damage to all membranes of the vessel wall with the transition to sclerosis and calcinosis, which leads in some cases to sharp stenosis and even obliteration of vessels, in others - to the development of an aneurysm.

The topography and spread of changes in the vascular system in systemic vasculitis is diverse: vessels of all sizes and types are involved in the pathological process: aorta (aortitis), arteries (arteritis), arterioles (arteriolith), capillaries (capillaritis), veins (phlebitis), lymphatic vessels (lymphangitis). However, with various types of vasculitis, vessels of a certain caliber are affected mainly: the aorta and its large branches; large, medium and small arteries (elastic-muscular and muscular type), small arteries and vessels of the microcirculatory channel, veins.

Changes in organs and tissues in connection with the development of vasculitis are of a secondary nature and are manifested by heart attacks, post-infarction large-focaland ischemic small-cell sclerosis, atrophy of parenchymal elements, gangrene, hemorrhages. In addition to local, there may be general changes associated with vasculitis of vessels that supply blood flow to another organ. Thus, with the development of the process in the renal arteries, renal hypertension occurs, in the vessels of the lungs - hypertension of the small circle of blood circulation and a symptom of pulmonary heart failure; skin vessels - hemorrhagic diathesis.

Etiology and pathogenesis. The etiology of the vast majority of primary systemic vasculitis is unknown. The pathogenesis of systemic vasculitis, both primary and secondary, is associated with immune hypersensitivity reactions that occur in response to exposure to various antigens. Depending on the preference of one or another mechanism of hypersensitivity, systemic vasculitis is divided into three groups:

4 immediate type hypersensitivity vasculitis;
5 vasculitis: delayed-type hypersensitivity;
6 hypersensitivity vasculitis of mixed type.

With the leading role of hypersensitivity of the immediate type (immune complex damage to the vascular wall), alterative (fibrinoid changes up to necrosis) and exudative (infiltration of the wall by polymorphonuclear leukocytes, macrophages) processes prevail, destructive (necrotic) vasculitis develops, often necrotic arteritis (nodular periarteritis, syndrome Wegener, allergic granulomatosis, vasculitis in rheumatic diseases ("hypersensitivity angiitis").

With the predominance of hypersensitivity of the delayed type, cellular reactions in the form of lymphohistiocytic infiltrates with the formation of granulomas are of primary importance. Productive vasculitis occurs, including granulomatous arteritis (Takayasu's, Horton's diseases).

Vasculitis caused by hypersensitivity of the immediate type, which are characterized by the

destructive nature of the changes, mostly have an acute course, and vasculitis caused by hypersensitivity of the delayed and mixed type (productive, granulomatous) are subacute and chronic.

The classification of systemic vasculitis takes into account the following criteria: etiology, pathogenesis, nosological affiliation, predominant nature and prevalence of the inflammatory reaction, morphological type of affected vessels, cerebrospinal localization, which determines the interest of certain organs (organopathology), clinical manifestations of the disease. At the same time, the nosological principle should be followed, based on which vasculitis is divided into primary and secondary.

Classification of systemic vasculitis

A. Primary vasculitis.

I. With a predominant lesion of the aorta and its large branches and a giant cell granulomatous reaction: nonspecific aortoarteritis (Takayasu's disease), temporal arteritis (Horton's disease).

II. With predominant damage to medium and small arteries
caliber and destructive-productive reaction:
1) nodular periarteritis;
2) allergic granulomatosis;
3) systemic necrotizing vasculitis;
4) Wegener's granulomatosis;
5) lymphatic syndrome with damage to the skin and mucous membranes.

III. With predominant damage to small-caliber arteries, microcirculatory vessels and veins: obliterative thromboangiitis (Buerger's disease).

IV. With damage to arteries of different calibers - mixed (unclassified) form.

B. Secondary vasculitis.

V. For infectious diseases:
1) syphilitic;
2) tuberculosis;
3) rickettsial, including typhoid fever;
4) septic;
5) others.

VI. With systemic diseases of the connective tissue: 1) rheumatic; 2) rheumatoid; 3) lupus.

VII. Hypersensitivity vasculitis with:
1) serum sickness;
2) Schönlein-Henoch purpura;
3) essential mixed cryoglobulinemia;
4) malignant neoplasms.

Among the primary systemic vasculitis, nonspecific aortoarteritis, nodular periarteritis, Wegener's granulomatosis, and obliterating thromboangiitis are the most important.

Nonspecific aortoarteritis.

The basis of non-specific aortoarteritis (Takayasu's disease) is inflammation of the arteries of the elastic type - the aorta and the proximal parts of the branches that depart from it, the trunk of the pulmonary artery.

Etiology and pathogenesis. The etiology of the disease is unknown, but there is a connection with various infectious diseases (rickettsiosis, rheumatism). The role of occupational injuries (intoxication by pesticides, lead compounds, welding aerosols) is also noted. Pathogenesis is associated with immunological mechanisms.

Pathological anatomy. Most often lesions occur in the area of the aortic arch and brachiocephalic arteries (74%), less often - in the abdominal (42%) and thoracic (18%) parts of the aorta, in the bifurcation area (18%) and in the ascending part of the arch (9%). Any branches of the aorta can be involved in the pathological process, including the coronary arteries of the heart. When the process spreads, inflammatory changes are also found in the walls of small-caliber arteries. At the same time, the vessels acquire a characteristic appearance: their walls are thickened, rigid, whitish in color. The intima has thickenings that narrow the lumen of the vessel, where wall or obturating thrombi are found. There is pronounced sclerosis in the adventitia and perivascular tissue; there are aneurysmal protrusions of the wall. Lesions can be segmental or diffuse. Depending on the appearance, stenotic,

Microscopic examination reveals damage to all layers of the vascular wall - panarteritis with

giant cell reaction. There is a change in the phases of the inflammatory reaction, which ends with sclerosis of the wall of the corresponding vessel, which allows us to talk about the stages of nonspecific aortoarteritis. The early (acute) stage is characterized by destruction of the internal elastic membrane and infiltration of all layers of the wall by lymphoid and plasma cells, giant cells are rare. The intima is thickened due to the proliferation of the endothelium and wall thrombi; significant changes occur in the media and adventitia. In the late (subacute) stage, the changes described above are replaced by a productive reaction with the formation of granulomas from macrophages, epithelioid, giant and plasma cells, and lymphocytes. In the final (sclerotic) stage, sclerosis of the vessel wall develops, in which there are remnants of the internal elastic membrane. There is organization of thrombotic masses, vascularization of the middle membrane and stenosis of the lumen up to complete obliteration.

Nodular periarteritis

Nodular periarteritis (synonyms - classic nodular periarteritis, Kussmaul-Meyer's disease) is a rheumatic disease characterized by systemic damage to the connective tissue of arteries of mainly small and medium caliber.

Etiology and pathogenesis. The etiology of the disease is unknown. The main role in the pathogenesis is played by the immune complex mechanism of damage to the vessel wall, which ends in fibrinoid necrosis.

Pathological anatomy. Among the small and medium-sized arteries, the most frequently affected are renal (90-100%), coronary arteries of the heart (88-90%), mesenteric arteries (5.7-60%), hepatic and brain arteries (46%). Arteritis of the striated muscles, stomach, pancreas, adrenal glands, and peripheral nerves is less common. Sometimes large-caliber arteries (carotid, subclavian, femoral, etc.) are involved in the process. The basis of the disease is vasculitis, and inflammation in the vessel wall consists of successive changes in alteration (segmental or circular fibrinoid necrosis of the middle membrane), exudative and proliferative cellular reactions in the outer membrane. The inflammation ends with sclerosis with the formation of nodular thickenings of the arterial wall (nodular periarteritis). Depending on the phase of the process detected by the morphologist,

The course of nodular periarteritis can be acute, subacute and chronic wave-like, which determines the different nature of changes in organs. With an acute and subacute course, foci of ischemia, heart attacks, and hemorrhages appear in the internal organs; with a chronic wave-like course - sclerotic changes in combination with dystrophic-necrotic and hemorrhagic ones, which leads to functional insufficiency of certain organs and systems. Subacute (extracapillary productive) or chronic (mesangial) glomerulonephritis often occurs in the kidneys, which leads to nephrosclerosis and kidney failure.

Wegener's granulomatosis

Wegener's granulomatosis is a systemic necrotizing vasculitis with granulomatosis and predominant damage to small and medium-sized arteries and veins, as well as vessels of the microcirculatory channel of the respiratory tract, lungs, and kidneys.

Etiology and pathogenesis. The development of the disease is associated with a hypothetical antigen, the nature of which has not yet been established. Assumptions are made about the significance of microbial and viral antigens, as well as drugs with antigenic and haptenic features. A number of provocative factors - hypothermia, insolation, vaccination, etc. may be a prerequisite for the disease. Pathogenesis is closely related to immunological mechanisms, and there is evidence in favor of the immunological nature of vascular damage.

Pathological anatomy. The morphological basis of the disease consists of:

1) systemic necrotizing vasculitis with granulomatous reaction;

2) necrotized granulomatosis mainly of the upper respiratory tract with successive involvement of the trachea, bronchi and lung tissue;

3) glomerulonephritis.

Vascular changes in Wegener's granulomatosis consist of three phases: alterative (necrotic), exudative, and productive with a pronounced granulomatous reaction. Sclerosis and hyalinosis of vessels occur with the development of aneurysm or stenosis up to complete obliteration of the lumen of the vessel. In arteries of medium caliber (muscular type) endarteritis occurs more often, and in arteries of small caliber - panarteritis. Vessels of the microcirculatory channel are constantly affected; then destructive and destructive-productive arteriolitis, capillaritis and venulitis appear. Damage to these same vessels is the basis of the formation of granulomas, which then connect with each other, forming areas of granulomatous tissue that are subject to necrosis.

Necrotizing granulomatosis is first detected in the upper respiratory tract, accompanied by nasopharyngitis, saddle deformity, nose, sinusitis, frontitis, ethmoiditis, angina, stomatitis,

laryngitis, otitis. Purulent inflammation with the development of ulcers and bleeding is pathognomonic. In some cases, these symptoms are one of the manifestations of the disease - a localized form of Wegener's granulomatosis. With progression, a generalized form of the disease develops, in which necrotizing granulomatosis is found in the trachea, bronchi, and lungs, where ulcerative-necrotic processes and foci of bronchopneumonia occur. In addition to the respiratory tract, granulomas can also be found in the kidneys, skin, joint tissues, liver, heart, and other organs. As a result, granulomatous lesions lead to sclerosis and deformation of organs.

Gromeluronephritis is a rather characteristic sign of Wegener's granulomatosis. Most often, it is a manifestation of mesangioproliferative or mesangiocapillary form - with fibrinoid necrosis of capillary loops and glomerular arterioles and extracapillary reaction (formation of characteristic "crescents").

In most cases, simultaneous damage to the upper respiratory tract, lungs and kidneys is observed.

Obliterating thromboangiitis

Obliterating thromboangiitis (Winiwarter-Burger's disease) is a systemic vasculitis, in which mainly small arteries and veins of the lower extremities are affected, which leads to occlusion of these vessels.

Etiology and pathogenesis. The causes of the disease and the mechanisms of its development are unknown. But smoking is absolutely important. Men under the age of 40 are more often ill.

Pathological anatomy. In this disease, damage to the veins of the lower extremities prevails, primarily productive endo-, meso- and periphlebitis develops. In the arteries of the lower extremities, which are less affected than the veins, similar processes develop - productive endo-, meso- and periarteritis. The vessels take on the appearance of thick fibrous cords with segmental thickening of the walls.

Acute, subacute and chronic stages of the disease are distinguished. The acute stage is characterized by the development of alterative-proliferative thrombovasculitis. Alterative changes are joined by infiltration of the vessel wall and perivascular tissue by polymorphonuclear leukocytes, which cause the destruction of the internal elastic membrane, and sometimes even the formation of microabscesses. In the subacute stage, a productive tissue reaction prevails. Lymphohistocytic infiltrates, signs of excessive vascularization and early formation of thrombi are found in the vessel wall. Typical is the formation of granulomas, which are found mostly in the middle shell and around necrotized fragments of the internal elastic membrane, as well as in thrombotic masses. Granulomas resemble either oleogranulomas or tuberculous granulomas. In the chronic stage, signs of the organization of blood clots dominate, which leads to complete obliteration of the vessel. The organization of blood clots can be accompanied by their drainage and calcification.

Control materials for the final stage of the lesson: => control questions:

1 *To characterize the criteria for the morphological assessment of systemic vasculitis.*

2 *Give the classification of systemic vasculitis.*

3 *Describe the changes in organs and tissues in systemic vasculitis?*

4 *What is characteristic of nonspecific aortoarteritis?*

5 *Morphological signs of periarteritis?*

1 *Morphological features of Wegener's granulomatosis and obliterating thromboangiitis.*

Control of the student's final level of knowledge is carried out with the help of tests (10 tests) and structural control of practical skills (assessment of knowledge and the ability to analyze macro- and microscopic changes in cells, tissues, organs during pathological processes).

1. Theoretical questions

Questions for self-control

1. Define and classify the concept of vasculitis.
2. Name the criteria for the morphological assessment of systemic vasculitis.
3. Name the types of inflammatory reactions in vasculitis.
4. Classification of vasculitis according to the depth of damage to the vascular wall and their localization.
5. Changes in organs and tissues in vasculitis.
6. Etiology and pathogenesis of systemic vasculitis.
7. Classification of systemic vasculitis.
8. Pathomorphology of nonspecific aortoarteritis.
9. Pathomorphology of Wegener's granulomatosis.
10. Pathomorphology of obliterating thromboangiitis.

2 Practical tasks

1. Prepare an essay on the topic: "Changes in organs and tissues in vasculitis"

2. Make a graph of the logical structure "Classification of systemic vasculitis".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

13 Pathomorphology: nats. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.

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36 www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine

37 http://bma.org.uk– British Medical Association

38 www.gmc-uk.org- General Medical Council (GMC)

39 www.bundesaerztekammer.de– German Medical Association

40 http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory

http://www.webpathology.com/- Web Pathology

Topic #6: "Cerebrovascular diseases. Postresuscitation encephalopathy and brain death syndrome. Neurodegenerative (neurodystrophic) (Alzheimer's disease) and demyelinating diseases (multiple sclerosis). Neuritis (neuropathy)"

Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and

anatomical analysis of sectional observations.

Basic concepts:

The student should know:

1. the place of cerebrovascular diseases in the structure of general morbidity;
2. morphological classification, morphological characteristics of the most important cerebrovascular diseases.

:The student should be able to:

1. to explain the main etiological and pathogenetic mechanisms of the occurrence and development of cerebrovascular diseases;
2. interpret pathomorphological changes in the brain with further diagnosis of a specific disease.
3. The student should have an idea of general pathological processes that develop in the brain with vascular pathology.

Topic content:

*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 importance. 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).

1. Theoretical questions

Questions for self-control

1. General characteristics, classification, background diseases and risk factors of cerebrovascular disease.
2. Infarct (ischemic stroke) of the brain: morphological characteristics.
3. Morphogenesis, morphological characteristics of selective necrosis of neurons (ischemic encephalopathy).
4. Morphological characteristics, consequences of hemorrhagic stroke.
5. Morphological characteristics, complications of spontaneous intracranial hemorrhage.
6. Morphological characteristics, complications of spontaneous subarachnoid hemorrhage.

2 Practical tasks

1. Prepare an essay on the topic: "Morphological features of strokes"

2. Make a graph of the logical structure "Classification of cerebrovascular diseases".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

16 Pathomorphology: nats. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.

17 The basics of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.

18 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

Additional:

1. Skrypnikov P.M., Skrypnikova T.P., Shynkevich V.I., Tovma V.V. Individual and age-related clinical and morphological features of the mucous membrane of the oral cavity: study. manual - Poltava: LLC "ASMI", 2016. - 102 p.

Electronic information resources

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

42 www.ama-assn.org– American Medical Association /American Medical Association

43 www.who.int- World Health Organization

44 www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine

45 http://bma.org.uk– British Medical Association
46 www.gmc-uk.org- General Medical Council (GMC)
47 www.bundesaerztekammer.de– German Medical Association
48 http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
http://www.webpathology.com/- Web Pathology

Topic #7: "Hypothalamic-pituitary disorders. Adrenal gland pathology. Pathology of the thyroid gland. Pathology of the endocrine apparatus of the pancreas"

Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:

The student should know:

1. principles of classification of pituitary diseases,
2. principles of classification of thyroid gland diseases,
3. principles of classification of diseases of the adrenal glands,
4. types of diabetes,
5. macro- and microscopic characteristics of endocrine gland diseases;

:The student should be able to:

1. define diseases of endocrine glands;
2. explain the morphological features of diseases of the endocrine glands;

Topic content:

Diseases of the endocrine system can be congenital or acquired in origin. They mostly arise as a result of pathological changes in the central nervous system, hypothalamic-pituitary regulation disorders, the development of autoimmune or tumor processes. Manifestations of such changes can be dysfunction of one or more glands - hypo-, hyper- or dysfunction. Structural rearrangement of endocrine glands is manifested by dystrophic, atrophic or dysplastic (hypo- and hyperplastic) and sclerotic processes, as well as the development of tumors.

Pituitary

Pituitary disorders occur with tumors of the gland, autoimmune damage, inflammation, necrosis (ischemic infarction) or develop as a result of damage to the hypothalamus or other parts of the central nervous system.

In this regard, in some cases, we can talk about cerebro(hypothalamus)-pituitary diseases. Among them, a significant place is occupied by: 1) acromegaly; 2) pituitary dwarfism; 3) cerebral-pituitary cachexia; 4) Itsenko-Cushing's disease; 5) adiposogenital dystrophy; 6) diabetes insipidus; 7) tumors of the pituitary gland.

Acromegaly. The cause of the development of this disease are hypothalamic-pituitary

disorders or somatotropic (eosinophilic) adenoma, less often – adenocarcinoma of the anterior lobe of the pituitary gland. An excessive amount of somatotropic hormone stimulates the growth of tissues, mainly of mesenchymal origin (connective, cartilage, bone), as well as parenchyma and stroma of internal organs (heart, liver, kidneys), etc. An especially noticeable increase in the size of the nose, lips, ears, eyebrows, lower jaw, bones and feet. The growth of bone tissue is quite often associated with its reconstruction, restoration of endochondral osteogenesis. If the disease occurs in childhood or youth, gigantism develops. Acromegaly is accompanied by changes in other endocrine glands: goiter, atrophy of the islet apparatus of the pancreas, hyperplasia of the thymus gland and pineal gland, cortex of adrenal glands, atrophy of gonads. Such morphological changes have characteristic clinical manifestations of the disease.

Pituitary dwarfism (pituitary dwarfism) occurs with congenital underdevelopment of the pituitary gland or with its destruction in childhood (inflammation, necrosis). In patients, there is a general underdevelopment with a proportional body structure, but the genitals, as a rule, are underdeveloped.

Cerebral-pituitary cachexia (Simonds' disease). The disease is characterized by progressive cachexia, atrophy of internal organs, and decreased function of the gonads. It is observed in young women and often after childbirth. In the anterior lobe of the pituitary gland, centers of necrosis are found, as a result of vascular embolism, or scars after necrosis. In some cases, the destruction of the anterior lobe of the pituitary gland is associated with a syphilitic, tubercular or tumor process. In this disease, in addition to damage to the pituitary gland, dystrophic or inflammatory changes in the diencephalon are observed. Sometimes they prevail over pituitary pathology; then cerebral cachexia develops.

Itsenko-Cushing's disease. This disease can be the result of hypothalamic disorders or the development of an adrenocorticotropic (basophilic) adenoma, less often - adenocarcinoma of the anterior lobe of the pituitary gland. As a result of hypersecretion of ACTH, there is bilateral hyperplasia of the adrenal cortex with excess production of glucocorticosteroids, which play a leading role in the pathogenesis of the disease. The disease is more often observed in women, its manifestation is progressive obesity of the upper type (face and trunk), arterial hypertension, steroid diabetes and secondary ovarian dysfunction. Osteoporosis with spontaneous bone fractures, hypertrichosis and hirsutism is also observed; purple-blue stretch marks (stretch marks) on the skin of the thighs and abdomen. Quite often, the disease is combined with nephrolithiasis and chronic pyelonephritis.

Adiposogenital dystrophy (from Latin adiposus - fatty and genetalis - sexual) or Babinsky-Ferlich disease. At the heart of the disease are pathological changes in the pituitary gland and hypothalamus, which arise as a result of a tumor or neuroinfection. The disease is characterized by progressive obesity, underdevelopment of the genitals and decreased function of the gonads.

Adiposogenital dystrophy is sometimes associated with hypothyroidism, decreased function of the adrenal cortex, and diabetes insipidus.

Diabetes insipidus. The disease occurs when the posterior lobe of the pituitary gland is damaged (tumor, inflammation, trauma). Along with damage to the posterior lobe of the pituitary gland, changes in the diencephalon are constantly observed. The disease is manifested by non-sugar enuresis, which is associated with the elimination of the function of the antidiuretic hormone and the ability of the kidneys to concentrate urine, which is accompanied by the excretion of a significant amount of urine (polyuria) and increased thirst; severe consequences of diabetes insipidus are associated with the loss of water in the body and a violation of mineral metabolism.

Pituitary tumors. In most cases, they are hormonally active.

Adrenal glands

In the cortex of the adrenal glands, mineralocorticosteroids (aldosterone), glucocorticosteroids and sex hormones are formed, the secretion of which is controlled by adenocorticotropic and gonadotropic hormones of the anterior lobe of the pituitary gland, respectively. An increase in the tropical effects of the pituitary gland or the development of a hormonally active tumor of the adrenal cortex is accompanied by their hyperfunction, and a decrease in these effects or the destruction of the gland cortex is accompanied by hypofunction. The secretion of hormones of the medulla of the adrenal glands (adrenaline, norepinephrine) is stimulated by the sympathetic nervous system. Their hypofunction is well compensated by chromaffin tissue; hyperfunction is associated with a tumor (phaeochromocytoma).

Addison's disease (named after the English doctor T. Addison, who first described this disease in 1849), or bronze disease. The disease is caused by bilateral damage mainly to the cortical substance of the adrenal glands and exclusion (acorticism) or decrease (hypoadrenocorticism) of hormone production by the glands. Quite often, the cause of bronze disease is tumor metastases in both glands, their autoimmune damage (primary Addison's disease), general amyloidosis (epinephric amyloidosis), hemorrhages, necrosis due to vascular thrombosis, as well as tuberculosis. In some cases, the disease is caused by a disturbance in the hypothalamic-pituitary system (decrease in the secretion of ACTH or corticotropin-releasing factor) or hereditary processes. In Addison's disease, there is hyperpigmentation of the skin (melanoderma) and mucous membranes in connection with the hyperproduction of ACTH and melanocyte-stimulating hormone, myocardial atrophy, and a decrease in the lumen of the aorta and main vessels. There is also adaptive hyperplasia of cells of the islet apparatus of the pancreas (hypoglycemia), atrophy of the mucous membrane of the stomach, especially the lining cells; along with the mentioned changes, hyperplasia of lymphoid tissue and thymus gland is found. Death in Addison's disease occurs from acute adrenal insufficiency, cachexia (suprarenal cachexia), or cardiovascular failure. There is also adaptive hyperplasia of cells of the islet

apparatus of the pancreas (hypoglycemia), atrophy of the mucous membrane of the stomach, especially the lining cells; along with the mentioned changes, hyperplasia of lymphoid tissue and thymus gland is found. Death in Addison's disease occurs from acute adrenal insufficiency, cachexia (suprarenal cachexia), or cardiovascular failure. There is also adaptive hyperplasia of cells of the islet apparatus of the pancreas (hypoglycemia), atrophy of the mucous membrane of the stomach, especially the lining cells; along with the mentioned changes, hyperplasia of lymphoid tissue and thymus gland is found. Death in Addison's disease occurs from acute adrenal insufficiency, cachexia (suprarenal cachexia), or cardiovascular failure.

Adrenal gland tumors are hormonally active in origin.

Thyroid

Diseases of the thyroid gland include goiter (goiter), thyroiditis and tumors. These diseases are accompanied by hyperthyroidism (thyrotoxicosis) or hypothyroidism (myxedema).

Goiter (goiter) is a pathological enlargement of the thyroid gland. The classification of goitre takes into account, on the one hand, morphological features, on the other - epidemiology, causes, functional and clinical features. Depending on the morphological features and appearance of the glands, diffuse, nodular, and diffuse-nodular (mixed) goiters are distinguished; by histological structure - colloidal and parenchymal goiter. A colloidal goitre is made up of follicles of various sizes filled with colloid. In some cases, follicles are large, cyst-like, their epithelium is flattened (macrofollicular colloidal goiter); in others - small (microfollicular colloidal goiter); in the third, there are large follicles along with small ones (macro-microfollicular colloidal goiter). Epithelial growth in the form of papillae is possible in colloidal goiter (proliferative colloidal goiter). After some time, blood circulation disorders occur in the goitre tissue, necrosis and calcification appear, connective tissue grows, sometimes with the formation of bone. A colloidal goiter usually has the appearance of a dense knot. Parenchymal goiter is characterized by the proliferation of the epithelium of follicles, which grows in the form of solid structures with the formation of small follicle-like structures without or with a small amount of colloid. Quite often, this goiter is diffuse, has the appearance of grayish-pink fleshy tissue. A combination of colloidal and parenchymal goiter is possible. Depending on the epidemiology, functional and clinical features, endemic, sporadic and diffuse toxic (thyrotoxic) goiters are distinguished (Based's disease, Graves' disease). sometimes with bone formation. A colloidal goiter usually has the appearance of a dense knot. Parenchymal goiter is characterized by the proliferation of the epithelium of follicles, which grows in the form of solid structures with the formation of small follicle-like structures without or with a small amount of colloid. Quite often, this goiter is diffuse, has the appearance of grayish-pink fleshy tissue. A combination of colloidal and parenchymal goiter is possible. Depending on the epidemiology, functional and clinical features, endemic, sporadic and diffuse toxic (thyrotoxic) goiters are distinguished (Based's disease, Graves'

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Endemic goiter develops in people who live in mountainous areas (some regions of the Urals, Siberia, Central Asia; in Europe - Carpathians, Alps). The reason for the development of goiter is mostly iodine deficiency in drinking water. At the same time, the thyroid gland increases, has the structure of a colloidal or parenchymal goiter; gland function is reduced. If goiter develops in early childhood, then its manifestations are general physical and mental underdevelopment - endemic cretinism.

Sporadic goiter appears in youth or adulthood. It can have a diffuse, nodular or mixed colloid or parenchymal structure. This goiter does not affect the general state of the body, but when it increases significantly, it squeezes the neighboring organs (esophagus, trachea, pharynx), disrupts their function (retroesophageal, retrotracheal goiter). In some cases, the so-called basedovification of goiter (moderate papillary proliferation of the epithelium of the follicles and accumulation of leukocyte infiltrates in the stroma of the gland) may occur. Sporadic goiter becomes the basis of diffuse toxic goiter.

Diffuse toxic goiter (Basedov's disease, Graves' disease) is the most striking manifestation of hyperthyroidism syndrome, which is why it is also called thyrotoxic goiter. The reason for its development is autoimmunization: antibodies stimulate cellular receptors of thyrocytes. This makes it possible to attribute diffuse toxic goiter to "receptor antibody diseases". Morphological features of diffuse toxic goiter are found only during 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 that branch in the middle of the follicles; vacuolization and change in tinctorial properties of the colloid (poorly accepts paints) in connection with its dilution and depletion of iodine; lymphoplasmacytic infiltration of the stroma; the formation of lymphatic follicles with germinal centers in them. Visceral changes are also 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 stroma, as well as intracellular swelling of muscle fibers are observed - thyrotoxic heart, the consequence of which is diffuse intermediate sclerosis. Serous edema is also observed in the liver, as a result of which thyrotoxic fibrosis of the liver develops. Dystrophic changes in nerve cells, perivascular cellular infiltrates are found in the medulla oblongata. An increase in the thymus gland, hyperplasia of lymphoid tissue, and atrophy of the adrenal cortex are often found. Death in diffuse toxic goiter occurs from heart failure or exhaustion.

Thyroiditis is a group of diseases, among which Hashimoto's thyroiditis, or Hashimoto's disease, is a true autoimmune disease. Autoimmunization is associated with the appearance of autoantibodies to microsomal antigen and surface antigens of thyrocytes, as well as thyroglobulin. Autoimmune process determined by DR histocompatibility antigens leads to diffuse infiltration of gland tissue by lymphocytes and plasmatic cells, formation of lymphoid follicles in it. As a result of the influence of mainly immune effector cells, the parenchyma of the gland dies and is replaced by connective tissue. With a long course, morphological changes in the thyroid gland resemble Riedel's thyroid (goiter).

Riedel's thyroid (Riedel's goiter) is characterized by the primary growth of coarse fibrous connective tissue in the gland, which leads to atrophy of the follicular epithelium (fibrous goiter). The gland becomes hard ("iron", "stone" goiter). Fibrous tissue spreads to tissues adjacent to the gland and imitates a malignant tumor.

Thyroid tumors are mostly epithelial, both benign and malignant.

Parathyroid glands

An important practical significance belongs to the syndrome of hyperfunction of the parathyroid glands - hyperparathyroidism, the morphological manifestation of which is hyperplasia or a tumor (adenoma) of these glands; possible hyperparathyroidism and autoimmune genesis. There is a distinction between primary and secondary hyperplasia of the parathyroid glands. Primary hyperplasia, more often a gland adenoma, leads to parathyroid osteodystrophy. Secondary hyperplasia occurs as a reactive, compensatory phenomenon in connection with the accumulation of lime in the body during primary destruction of bones (metastases of malignant tumors, myeloma, rickets) and kidney diseases (chronic renal failure).

At the heart of parathyroid osteodystrophy or fibrous osteodystrophy are disorders of calcium and phosphorus metabolism in connection with excessive secretion of parathyroid hormone by adenoma of the glands. Under the influence of this hormone, mineral salts are mobilized from the bones; resorption processes prevail over new formation of bone tissue; during this process, mainly osteoid tissue is formed, deep bone remodeling takes place. Hypoparathyroidism is mostly the result of autoimmunity, which leads to the death of the glands. Sometimes it occurs after accidental surgical removal of glands, accompanied by tetany.

Pancreas

Violations of the incretory function of the islet apparatus of the pancreas can be manifestations of an increase or decrease in the function of the cells from which the islets are built. A decrease in the function of ß-cells is more often observed, which is accompanied by the development of diabetes; less often, in connection with the development of a tumor (adenoma) from ß-cells (ß-insuloma), a hypoglycemic syndrome occurs. With adenoma of G - islet cells (synonyms: G-insuloma, gastrinoma, or ulcerative adenoma), the characteristic Allison-Zollinger syndrome develops, in which multiple ulcers of the gastric mucosa, hypersecretion of gastric juice, and diarrhea appear.

Diabetes.

Diabetes mellitus (sugar disease) is a disease caused by a relative or absolute lack of insulin. Classification. The following forms of diabetes are distinguished: spontaneous, secondary, diabetes of pregnant women and latent (subclinical). Among the spontaneous form of diabetes, diabetes of the first type (insulin-dependent) and diabetes of the second type (non-insulin-dependent) are distinguished. Secondary diabetes develops in diseases of the pancreas (pancreatitis), diseases of the endocrine system (acromegaly, Itsenko-Cushing syndrome, pheochromocytoma), complex genetic syndromes (Louis-Bar ataxia-telangiectasia, myotonic dystrophy, etc.), when using certain medications (drug diabetes). Gestational diabetes occurs when a pregnant woman begins to have a violation of glucose tolerance, and the so-called latent (subclinical) diabetes – when glucose tolerance is impaired in seemingly healthy people. Only spontaneous diabetes is considered as an independent disease. Among the etiological and pathogenetic factors (risk factors) in diabetes, the following are distinguished: 1) genetically determined disorders of the function and quantity of ß-lithins (decreased insulin synthesis, impaired transformation of proinsulin into insulin, and abnormal insulin synthesis); 2) environmental factors that disrupt the integrity and functioning of ß-cells (viruses, autoimmune reactions; nutrition that leads to obesity; increased activity of the adrenergic nervous system). With different types of spontaneous diabetes, the risk factors are not equivalent. So, for type 1 diabetes, which is quite common in young people (juvenile diabetes), characteristic connection with viral infectious diseases (quite high titers of antibodies to Coxsackie viruses, epidemic parotitis), genetic

predisposition (association with certain histocompatibility antigens DW3, DW4, B8, B15, etc.), autoimmunization (presence of antibodies to ß-cells) . In diabetes of the second type, which affects adults (elderly) people (adult diabetes), metabolic anti-insular factors and a decrease in the receptor activity of cells (ß-cells of pancreatic islets, insulin-dependent tissue cells), which are inherited in an autosomal dominant type, are of primary importance. However, there is no association of this type of diabetes with certain histocompatibility antigens. Insular insufficiency determines a violation of glycogen synthesis, an increase in the content of sugar in the blood (hyperglycemia), and its appearance in the urine (glucosuria). In such conditions, a significant part of sugar (glucose) is formed due to the transformation of fats and proteins, hyperlipidemia, acetone- and ketonemia occur; underoxidized "ballast" substances accumulate in the blood, acidosis develops. In diabetes, in connection with impaired metabolism and autoimmunization, damage to blood vessels and the successive development of diabetic macro- and microangiopathy are associated, which should be considered as an integrative component of diabetes and one of the characteristic clinical and morphological manifestations of this disease. Pathological anatomy. In diabetes, changes in the islet apparatus of the pancreas, liver, kidneys, and vascular bed are observed, first of all. The pancreas is reduced in size, sclerosis and lipomatosis occur. Most of the islets are subject to atrophy and hyalinosis, individual islets compensatory hypertrophy. In some cases, the pancreas looks unchanged, only with the help of special methods of histochemical research, degranulation of ß-cells is found. The liver is moderately enlarged, glycogen in hepatocytes is not detected, fatty dystrophy is detected. The vascular bed changes due to its reaction to hidden and real metabolic disorders, as well as immune complexes circulating in the blood. At the same time, diabetic micro- and macroangiopathy develops. Diabetic macroangiopathy is manifested by atherosclerosis of elastic and muscular arteries. In diabetic microangiopathy, there is plasmarrhagic damage to the basement membrane of the microcirculatory channel with a joint reaction of the endothelium and perithelium, which culminates in sclerosis and hyalinosis. At the same time, lipohyaline, characteristic only of diabetes, appears. In some cases, the proliferation of endothelium and perithelium is combined with lymphohistiocytic infiltration of the wall of microvessels, which allows us to talk about vasculitis. Microangiopathies in diabetes are widespread. Stereotypical changes in microvessels are observed in the kidneys, retina of the eye, skeletal muscles, skin, mucous membranes of the gastrointestinal tract, pancreas, brain, etc. bodies The most characteristic and relatively specific morphological changes of diabetic microangiopathy are found in the kidneys in the form of diabetic glomerulosclerosis. The basis of such changes is the proliferation of mesangial cells in response to the clogging of the mesangium with "ballast" metabolic products and immune complexes, as well as their increased formation of a membrane-like substance. The consequence of such changes is the hyalinosis of the mesangium and the death of glomeruli. Diabetic glomerulosclerosis can be diffuse, nodular or mixed. It manifests

itself clinically in the form of Kimmelstiel-Wilson syndrome: high proteinuria, edema, arterial hypertension. The so-called exudative manifestations of diabetic nephropathy are possible - the formation of "fibrin caps" on the capillary loops of the glomeruli and "capsular drop". Such glomerular changes are complemented by peculiar changes in the epithelium of the narrow segment of the nephron, where glucose is polymerized into glycogen (the so-called glycogenic infiltration of the epithelium). The epithelium becomes high, with light, translucent cytoplasm, in which glycogen is detected using specific staining methods. Peculiar morphological changes in diabetic angiopathy are observed in the lungs: lipogranulomas, which are made of macrophages, appear in the walls of arteries, especially of the muscular type. lipophages and giant cells of foreign bodies. Diabetes is characterized by lipid infiltration of cells of the histiomacrophage system (spleen, liver, lymph nodes) and skin (xanthomatosis of the skin). Complications of diabetes are various. Possible development of diabetic coma. Quite frequent complications caused by macro- and microangiopathies (gangrene of limbs, myocardial infarction, blindness) and especially diabetic nephropathy (renal failure – acute with papillonecrosis, chronic – with glomerulosclerosis). In patients with diabetes, infectious diseases are possible, especially purulent ones (pyoderma, furunculosis, sepsis), often exacerbation of tuberculosis with generalization of the process and the prevalence of exudative changes. Death in diabetes occurs from complications. Diabetic coma is rare. More often, patients die from gangrene of the limbs (septicopyemia),

Gonads

Dyshormonal, inflammatory and tumor diseases develop in the ovaries and testicles.

1. Theoretical questions

Questions for self-control

1. Define acromegaly.
2. What is pituitary dwarfism?
3. What lesion leads to the development of Symonds disease?
4. What is the difference between Itsenko-Cushing disease and Itsenko-Cushing syndrome?
5. What are the manifestations of Babinski-Frehlich disease?
6. What damage is associated with the occurrence of diabetes insipidus?
7. Describe the changes in the body in Addison's disease.
8. What are the causes of endemic goiter?
9. What are the causes of sporadic goiter?
10. What are the causes of Based's goiter?
11. What thyroid diseases do you know?
12. What diseases of the parathyroid glands do you know?
13. Describe macroangiopathies in diabetes.
14. Describe microangiopathies in diabetes.
15. Describe the adiposogenital syndrome.
16. Give a description of vascular changes in diabetes.
17. What do the vessels of the fundus look like in diabetes?
18. What is characteristic of Kimelstiel-Wilson syndrome?
19. Morphological signs of Addison's disease?

2 Practical tasks

1. Prepare an essay on the topic: "Morphological manifestations of diabetes"

2. Make a graph of the logical structure "Classification of endocrine diseases".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

19 Pathomorphology: nats. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.

20 The basics of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.

21 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

Additional:

1. Pathological anatomy: a textbook. Trans. from Russian 4th edition / Strukov A.I., Serov V.V. - Kharkiv.: Fakt, 2004. - 864 p.
2. Skrypnikov P.M., Skrypnikova T.P., Shynkevich V.I., Tovma V.V. Individual and age-related clinical and morphological features of the mucous membrane of the oral cavity: study. manual - Poltava: LLC "ASMI", 2016. - 102 p.

Electronic information resources

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

50 www.ama-assn.org– American Medical Association /American Medical Association

51 www.who.int- World Health Organization

52 www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine

53 http://bma.org.uk– British Medical Association

54 www.gmc-uk.org- General Medical Council (GMC)

55 www.bundesaerztekammer.de– German Medical Association

56 http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory

http://www.webpathology.com/- Web Pathology

Topic #8: "Clinical and morphological features of the organs of the maxillofacial system and the oral cavity"

Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:

The student should know:

1. Classification and essence of changes associated with the pathology of the organs of the

maxillofacial system and the oral cavity.

2. Etiology, pathogenesis, pathological anatomy of these pathological conditions.
3. Results, complications of diseases associated with the pathology of the organs of the maxillofacial system and the oral cavity.

:The student should be able to:

1. Classify the pathological processes of the organs of the maxillofacial system and the oral cavity.
2. To characterize the etiology, pathogenesis and morphological essence of the pathology of the organs of the dento-maxillofacial system and the oral cavity.

Topic content:

General information about the pathomorphological structuremaxillofacial system and oral cavity. The oral cavity is the first part of the digestive tract. Its main function is the entry of food into the digestive tract and the beginning of its digestion due to salivation and pushing the food ball into the throat. It is also: secondary respiratory tract, place of sound modification for speech, chemosensory department. The mobility of the lips is also of great importance for the function of speech, singing, and coughing. The oral cavity is limited by the lips in front, the cheeks on the sides, the floor of the oral cavity from below, the oropharynx from behind and the palate from above. The oropharynx begins at the junction of the hard and soft palate and below behind the grooved papillae of the tongue. The bone base of the oral cavity is represented by the upper and lower jaw bones. The oral cavity includes the lips, gums, retromolar triangle, teeth, hard palate, mucous membranes of the cheeks, movable tongue and floor of the mouth. The large salivary glands are closely related to the structures of the oral cavity, although they are not part of the oral cavity. The tongue is part of the oral cavity; its anatomy is described in more detail below. The tonsils, soft palate, root of the tongue and the back wall of the pharynx are anatomically part of the oropharynx.

Mucous membrane of the oral cavity (SOPR). The oral cavity, except for the teeth, is lined with a mucous membrane. The structure of the SOPR varies in different areas of the oral cavity. There are masticatory (hard palate and gums), lining (mucous cheeks, lips, floor of the mouth, lower/ventral surface of the tongue, soft palate) and specialized (dorsal surface of the tongue) SOPR. The masticatory type is covered with a keratinized epithelium, and the lining type is normally covered with a non-keratinized epithelium. The specialized mucous membrane of the tongue forms outgrowths - papillae of the tongue. The functional commonality of SOPR departments is due to embryonic development. In general terms, the stratified squamous epithelium of the oral cavity and the epithelium of the salivary glands develop from the oral cavity of the embryo, in the formation of which the first, second and third gill arches play an important role, each of which is supplied with an artery, a nerve, cartilaginous and muscular components, which explains the complexity of the blood supply and innervation of the tissues and organs of the oral cavity covered by the SOPR. The SOPR of the oral cavity includes two layers: the epithelium and the lamina propria, the latter of which passes without a sharp border into the submucosal base, which is absent in certain areas. The thickness of the epithelial layer in different parts of the oral cavity ranges from 200 microns to 500 microns.
The epithelium located closer to the oral cavity is of ectodermal origin, and closer to the pharynx is endodermal. The mucous membrane of the lips, cheeks, tongue, hard and soft palate with small

salivary glands, epithelium of large salivary glands, gums, tooth enamel are of ectodermal origin. Epithelium SOPR - multilayered flat non-keratinized (about 30% of the area) with areas of keratinization in areas of increased functional load: dorsal surface of the tongue, hard palate, gingival sections. Keratinized epithelium covers the surface of the masticatory mucosa (hard palate, gums), separate areas of the lining mucosa (cheeks along the line of teeth closure) and specialized mucosa (dorsal surface of the tongue). Epithelium consists of several layers. The basal layer is formed by cubic or prismatic cells that lie on the basement membrane, have an oval nucleus with one or two nucleoli, basophilic cytoplasm with well-developed organelles. 20% of the volume of the cytoplasm is occupied by intermediate keratin filaments. Basal cells are cambial, actively dividing; together with the basement membrane, they ensure the connection of the epithelium with the underlying connective tissue. The layer of spinous cells is represented by several layers of large cells of irregular shape with cytoplasmic outgrowths in the form of spines (spinous processes), connected to each other by desmosomes; organelles are well developed; 30% of the volume of the cytoplasm is occupied by tonofilaments. The granular layer is formed by flattened cells, their nuclei are also flat, with condensed chromatin, two types of granules are found in the cytoplasm: keratohyaline (form the matrix of the corneal substance) and lamellar (provide the barrier function and waterproofing of the epithelium). The stratum corneum is formed by flat hexagonal horny scales that do not contain nuclei and organelles and are filled with keratin filaments. The scales have high mechanical strength and resistance to the action of chemicals. Nonkeratinized epithelium covers the SOPR in the area of the floor of the oral cavity, the ventral surface of the tongue, cheeks (except the line of closure of the teeth), most of the mucous membrane of the lips. It is represented by three layers: 1) a basal layer, similar in structure to the corresponding layer of the keratinized epithelium; 2) a spiny layer, characterized by numerous diffusely located tonofilaments and the chemical composition of cytokeratins; 3) a layer of flat cells formed by flattened cells containing cytokeratin filaments and nuclei with faint chromatin granules. According to modern concepts, during the keratinization of the SOPR epithelium, successive processes of differentiation take place, according to associated with the synthesis of specific proteins. Cytokeratins - proteins of the intermediate filaments of the cytoskeleton are a marker of epitheliocyte differentiation. One of their values is diagnostic, when determining the origin of epithelial tumors. Differentiation processes of keratinized and non-keratinized epithelium in the oral cavity occur in a similar sequence. The differences are mainly related to the number of synthesized cationic proteins, which act as regulators of morphological processes. In particular, the involvement of these proteins in the mechanism of destruction of nuclei in the surface layer of epitheliocytes has been clarified. So, the keratinized epithelium of the SOPR is represented by several cell layers, starting from the outside: 1. An anucleate keratinized scale with a dense zone adjacent to the plasmolemma, filaments and a keratin matrix. 2. Cells of the granular layer with a large number of cationic proteins. 3. Cells of the spinous layer. Cytoplasm is less basophilic, tonofilaments are found in it. 4. The cells of the basal layer are sharply basophilic, they divide. The basement membrane of the SOPR is located between the epithelium and the connective tissue of the own lamina of the mucous membrane. At the light-optical level, it has the appearance of a structureless strip that does not stain with hematoxylin and eosin and gives an intense Schick reaction. At the ultrastructural level, a light fine-grained layer adjacent to the outer cell membrane (light plate) is revealed, a layer formed by a fine-grained or fibrillar substance (dense plate) lies deeper. Epitheliocytes are attached to the basement membrane by half-desmosomes, from which thin anchor filaments sink deep into the light plate. The light plate is formed by glycoproteins. A dense plate lies deeper, which contains type IV collagen and entactin, which binds to laminin. Anchor fibrils are formed by collagen type VII, and fibrils associated with them - collagen types I and III. In addition, the basement membrane includes type V collagen and fibronectin. Due to its structure, the basement membrane can retain a number of molecules with a high mass, for example, antigen + antibody complexes, as well as microorganisms. The SOPR epithelium contains cells that differ in their origin from the epithelium itself, but are closely related to it functionally. These include: 1) leukocytes, which are constantly found in the epithelium. Most often, in a smear-imprint from the surface of the CSF there are individual segmented neutrophils, usually degeneratively changed; 2) T-lymphocytes of various subpopulations, which are

the main effectors of the immune response in the epithelium; 3) cells with a process structure, are described below. Melanocytes are of neuronal origin. The body of these cells is contained in the basal layer, and the processes reach the spinous, without forming intercellular connections with the surrounding epitheliocytes. Their main function is the production of melanin, the nature of which is purely individual and determined genetically. Langerhans cells (CL) are localized in the basal and spinous layers of the epithelium; their long branched processes reach the granular layer and are located between epitheliocytes without forming intercellular connections with them. Together with tissue macrophages, committed bone marrow progenitors and circulating monocytes constitute a mononuclear-phagocytic system. CL are able to capture antigens that have penetrated the epithelium. The influence of Langerhans cells on the proliferation and differentiation of epitheliocytes has been established. The number of CL is different in different areas: the epithelium of the lips, cheeks and soft palate contains about 500 cells per 1 mm2 of the epithelium, while the epithelium of the hard palate and gums contains less - 150-200 cells per 1 mm2. Women have more CL cells than men. The number of KL increases in smokers. Merkel cells are derivatives of the neural crest, associated with afferent nerve fibers and perform a receptor function. The cell body lies in the basal layer, and the processes are connected by desmosomes with epitheliocytes of the basal and spinous layers. Organelles are moderately developed. Merkel cells contain granules with neurotransmitters: vasoactive intestinal peptide, histidine-isoleucine peptide, calcitonin gene-related peptide, substance R. Often these cells form clusters (especially in the gums). In the SOPR, there is a constant interaction of epitheliocytes with non-epithelial cells, which forms a single system of connected elements. Thus, epitheliocytes produce a number of cytokines and chemokines that regulate the activity of KL and leukocytes, providing a microenvironment for local immune processes. On the other hand, KL produce IL-1, which activates lymphocytes and enhances the expression of melanocyte-stimulating hormone receptors on melanocytes. Thus, for example, a system of network interactions between the epithelium itself and cells of the immune system is formed. The own (connective tissue) plate, on which the layer of epithelium lies, forms numerous projections, or papillae, that penetrate the layer of epithelium in the surface sections. Connective tissue papillae are represented by loose connective tissue, they contain blood vessels that feed the epithelium. In accordance, the recesses of the epithelial layer between the connective tissue papillae are morphologically outlined as epithelial ridges. It is believed that these mutual protrusions increase the contact area between the epithelium and its own plate and contribute to a better exchange of substances between them. In addition, they provide a stronger attachment of the epithelial layer to its own lamina of the mucous membrane. The height of the connective tissue papillae and epithelial ridges is different in different parts of the SOPR: higher papillae, more densely located, are found in those parts of the SOPR where the mechanical load is higher (clear, hard palate). The own plate of the SOPR is divided into two layers: papillary - woven into the epithelium, formed by loose fibrous connective tissue, which was already discussed above, and reticular - formed by dense fibrous, irregular connective tissue. The main substance of the lamina propria is formed by complex hydrated complexes of proteoglycans and glycoproteins and has the histological name "amorphous substance". An amorphous substance is a gel-like substance, the main microenvironment for cellular elements and fibrous structures. Here, connective tissue fibers are formed and complex metabolic processes take place. The volume of the amorphous substance is different in different departments of the mucous membrane. Most of it is in the mucous membrane of the lips and cheeks. The amorphous substance contains three main types of fibers: 1) collagen fibers (formed by type III collagen); 2) reticular fibers (formed by type III collagen); 3) elastic fibers (formed by elastin and glycoproteins). Elastic fibers include elastic, oxytalan and elaunin fibers. Cellular elements of the own plate of the SOPR are represented by several main types. Fibroblasts are the main cellular elements of connective tissue, they are large process cells with basophilic cytoplasm and an oval nucleus with finely dispersed chromatin. Organelles are well developed in the cytoplasm. Produce components of intercellular substance and participate in intracellular and extracellular destruction of intercellular substance. Fibrocytes are more differentiated elongated cells, with short, wide processes and a small number of organelles; have weak synthetic activity. Sedentary macrophages are migratory cells with an elongated or appendage shape, have a powerful lysosomal

apparatus. Macrophagocytes combine the functions of effector cells in innate and adaptive immune reactions, as a result, they can be involved in inflammation and antigen presentation and, activated by cytokines, exhibit nonspecific cytotoxicity against damaged cells through direct contact and at the expense of cytolytic factors, as well as mediate healing. The leading role of macrophages at rest is manifested as "cleaner cells", the main function of which is to clean the interstitial space from foreign cellular material. The functions of macrophages vary depending on their anatomical location and phenotype. Tissue basophils are large cells containing granules with histamine and heparin. Often located perivascularly, the number of these cells decreases deep into the lamina propria. Plasma cells are the final forms of B-cell differentiation. The main function of plasma cells is the production of antibodies. Their content increases with infectious and allergic diseases. A small number of plasma cells and a small number of T-lymphocytes at different stages of differentiation may be present in the own plate of the CSF. In certain areas of the lamina propria, mainly where the submucosal layer is developed, small cells of B- and T-lymphocytes may be contained in the form of lymphoid clusters (in the mucous membrane of the cheeks and lips - up to 1%-5% of the total volume). Submucous base. The lamina propria of the mucous membrane of the oral cavity gradually passes into the submucosal base, which consists of loose fibrous connective tissue and contains a cluster of fat cells, the end sections of small mucous and salivary (more often mixed) glands, blood vessels and nerve elements. The submucosal base is expressed on the ventral surface of the tongue, cheeks, lips, oral surface of the soft palate, days of the oral cavity. The submucosal base of the SOPR performs a supporting function, ensuring the mobility or flexibility of the mucous membrane and attaching it to the underlying muscles or periosteum. The submucous base is absent on the dorsal and lateral surfaces of the tongue, in the gums, partially on the hard palate: in these areas, the mucous membrane is fused with the intermuscular connective tissue (tongue) or with the periosteum (gum, hard palate). Therefore, the structure of the SOPR determines the following functions: - protective, due to the constant renewal of the multilayered epithelium (physiological regeneration); - barrier for microorganisms and many substances; - participation in immune reactions; - permeability to a range of nutrients, certain microorganisms, and medications. alve or periosteum. The submucous base is absent on the dorsal and lateral surfaces of the tongue, in the gums, partially on the hard palate: in these areas, the mucous membrane is fused with the intermuscular connective tissue (tongue) or with the periosteum (gum, hard palate). Therefore, the structure of the SOPR determines the following functions: - protective, due to the constant renewal of the multilayered epithelium (physiological regeneration); - barrier for microorganisms and many substances; - participation in immune reactions; - permeability to a range of nutrients, certain microorganisms, and medications. alve or periosteum. The submucous base is absent on the dorsal and lateral surfaces of the tongue, in the gums, partially on the hard palate: in these areas, the mucous membrane is fused with the intermuscular connective tissue (tongue) or with the periosteum (gum, hard palate). Therefore, the structure of the SOPR determines the following functions: - protective, due to the constant renewal of the multilayered epithelium (physiological regeneration); - barrier for microorganisms and many substances; - participation in immune reactions; - permeability to a range of nutrients, certain microorganisms, and medications. due to the constant renewal of the multilayered epithelium (physiological regeneration); - barrier for microorganisms and many substances; - participation in immune reactions; - permeability to a range of nutrients, certain microorganisms, and medications. due to the constant renewal of the multilayered epithelium (physiological regeneration); - barrier for microorganisms and many substances; - participation in immune reactions; - permeability to a range of nutrients, certain microorganisms, and medications.

lipsIncludes: the skin and red border of the lips, the mucous membrane of the lips and the transitional fold. The longer upper lip and the shorter lower lip are connected by labial commissures, forming the corners of the mouth. The lips are separated from the cheeks by nasolabial folds. The skin part of the lips has a structure typical of skin, covered with a multi-layered keratinized epidermis. There are hair, sebaceous and sweat glands. The red border of the lips is separated by a white line from the outer skin part and has an inner shiny pale red wet surface and an outer dry shiny red surface, which are separated by a red line (or Klein's zone), where the transition of the skin part to the mucous membrane takes

place. In another way, the red border of the lips is called vermilion, that is, it is the entire red outer surface of the lips. Vermilion has both dry and wet surfaces. internal, the mucous surface of each lip is connected along the central line with clear folds of the mucous membrane - frenulums, of which the upper one is more developed. Therefore, the part of the lips between the white and red lines inclusively is called the red border (vermilion). The characteristics of the red border are: configuration, relief, color, moisture. The red border of the lips is covered by the epidermis, but the stratum corneum is thinner here than on the skin. It has a well-defined granular layer. Located under the epidermis, the lamina propria is a direct extension of the dermis. It forms numerous papillae here that penetrate deep into the epidermis layer. These papillae have many capillary plexuses that shine through the surface layers and give them color. The wet surface of the red border (wet vermilion) is moistened by small mucous glands, it is covered with a multi-layered flat epithelium, is not keratinized, does not contain sebaceous glands, therefore it looks richer in color. Whitish nodules, 1-3 mm in size, are often visible on the red border of the lips, especially the upper one: sebaceous glands (or Fordyce's granules). Fordyce's granules can rarely be observed on the mucous membrane of the lips, in other parts of the gastrointestinal tract (as well as on the genitals, areolas of the mammary glands) and belong to the normal variant, although they can cause a cosmetic defect. The reason for the appearance of Fordyce's granules is considered to be increased production of sebaceous glands and narrowing of their ducts, which is partly related to the influence of sex hormones. In the early periods of a person's mature age, Fordyce's granules can gradually smooth out and disappear, which is associated with a physiological decrease in the secretion of sebaceous glands. The mucous membrane of the lips is covered with a multi-layered flat non-keratinized epithelium, located on its own plate (lamina propria). In the submucosa there are numerous tubuloacinar labial glands, mainly mucous, with shallow ducts that open into the lining of the oral cavity. The bodies of these glands are easy to detect visually and by palpation, so the patient himself can detect them and fix excessive attention on it. With their acute or chronic injury, the development of retention cysts (ranulae) is possible. The base of the lips is made up of the fibers of the striated circular muscle of the mouth. Intermuscular connective tissue is soldered with bundles of collagen fibers of the submucous base. This prevents the formation of wrinkles of the mucous membrane. Determine the configuration, relief, color and moisture of the red border of the lips. The color provides information about the patient's age and state of health: pallor, dryness, soreness, swelling, loss of sensitivity, the presence of lesions, etc.

CheeksCheeks have a muscular-mucosal structure and are bounded from above and below by the upper and lower oral cavity, in front by the labial commissure (corners of the mouth), behind by retromolar triangles and intermaxillary ligaments. The cheeks are covered by a mucous membrane, lined with non-keratinized epithelium, rich in glycogen, except for the line of closing the teeth, where thickening has developed due to parakeratosis. In the mucous membrane of the cheek, three zones can be conditionally distinguished: the upper (maxillary), lower (mandibular) and middle - along the line of closing the teeth. The underlying own plate of the mucous membrane forms a thick papillary layer and is able to stretch, adapting to the movements of the underlying muscles. The submucous base is rich in fibrous elastic structures that promote muscle mobility, and contains numerous small glands, mainly mucous, and islands of adipose tissue. It grows tightly with the intermuscular connective tissue of the buccal muscle. The last circumstance determines the smoothness and elasticity of the mucous membrane of the cheek. During the examination of the mucous membrane of the cheeks, a pale pink color is normally determined. A small ridge or line of normal or pale color is determined along the line of teeth closure - the so-called occlusal or white line (linea alba). Different amounts of sebaceous glands can also be located in the area of this line. In the distal parts of the cheeks, especially in front of the third upper molar, there are often quite specific protrusions of the mucous membrane, formed by a large number of end sections of mixed buccal salivary glands (mainly mucous), which lie in the submucous sections and are often immersed in the muscle layer. The last circumstance determines the smoothness and elasticity of the mucous membrane of the cheek. During the examination of the mucous membrane of the cheeks, a pale pink color is normally determined. A small ridge or line of normal or pale color is determined along the line of teeth closure - the so-called

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At the level of the second upper molar, the duct of the parotid salivary gland (Stenon's duct, ductus parotideus) opens, forming a papilla (papilla parotidea). It has the appearance of a triangular pink papule. Its size and degree of expression are individual. Along the transitional folds, i.e., at the transition of the mucous membrane of the cheek to the gums, blood vessels are clearly visible, which are defined as "arterial pattern".

Gums. The gingiva is a part of the ENT that covers the alveolar processes of the jaws. Healthy gums are covered with a masticatory type of mucous membrane, the epithelium of which is keratinized mainly by parakeratosis, does not have a submucous base, glands, only its own plate, which is tightly welded to the periosteum of the jaws. In the area of the neck of the teeth, the fibers of the circular ligament of the tooth are woven into the own plate of the gums, which also contributes to the tight attachment of the gums to the surface of the tooth.

The gingival margin, or the marginal part of the gums, the interdental papilla and the alveolar, attached part of the gums are distinguished. The gingival surface normally has a microgranular appearance, pale pink or coral pink in Europeans and varying degrees of pigmentation in people of other races. The gingival margin adheres tightly to the tooth tissue and is thinned in this area in the form of a "knife blade". The gingival margin is located at the level of the enamel-cement junction. The "serrated" configuration of the gums corresponds to the interdental papillae and the lingual and buccal depressions along the necks of the teeth. During the examination, it is necessary to pay attention to the difference in the color of the gums. So, the alveolar part of the gums has a richer color, near the teeth - pale. A change in the color of the gums can be caused by intoxication with salts of heavy metals, pathology of internal organs, sometimes smoking tobacco.

On the lingual, buccal part of the gums, yellowish-white small papules - milia, which are described below, are sometimes identified. In early literary sources, they were referred to as Sores' glands. The marginal edge of the gums, adhering to the surface of the teeth, forms a gingival or crevicular groove. Its depth is 1-1.5 mm, sometimes up to 3 mm. Normally, the furrow contains a little crevicular fluid, and when it is easily probed, there is no bleeding. The epithelium that lines the gingival groove is not keratinized, in the area of its bottom it passes to the surface of the tooth and connects with the enamel cuticle, which is called "epithelial attachment". The epithelium of the gingival sulcus is a direct continuation of the multilayered epithelium of the gums, but they are different in structure and origin.

The own plate in the area of epithelial attachment does not form papillae, therefore, the border between the epithelium and the connective tissue has the appearance of a straight line. It is believed that the reduced epithelium of the enamel organ, which covers the entire enamel before the tooth erupts, is involved in the formation of epithelial attachment. When the crown of the tooth begins to erupt, the reduced epithelium merges with the gingival epithelium, turning into an epithelial attachment. Over time, the remnants of the epithelium of the enamel organ, the components of the epithelial attachment, are gradually replaced by the epithelium of the gums. Epithelial attachment plays an important role in the biological protection of the periodontal ligament against the penetration of infection and other harmful agents of the external environment. The alveolar part of the gums, the part that covers the body of the jaws, continues vestibularly, into the transitional folds of the lips, cheeks, or, from the oral surface - respectively,

Tongue and floor of mouth. The tongue is a muscular organ, almost completely covered with a mucous membrane, which is tightly joined with intermuscular connective tissue. It occupies most of the oral cavity and oropharynx. Feels the taste, helps in chewing, swallowing, articulation (speech), cleaning the oral cavity. Five pairs of cranial nerves provide complex innervation of this multifunctional organ. The back of the tongue (dorsum) is covered with a specialized mucous membrane capable of perceiving taste, has a non-shiny, rough or velvety villous surface, because it is abundantly covered with papillae: thread-like (papillae filiformis), mushroom-like (papillae fungiformis) and leaf-like (papillae foliatae). Among them, only filamentous ones are covered with keratinized epithelium, and it is they that do not contain taste receptors and mainly determine the color of the back of the tongue - it is often a little lighter or colored after eating colored food, compared to other departments of the gastrointestinal tract. Leaf-shaped papillae are pale pink in color, mushroom-shaped - bright pink, thread-like - whitish-gray or brown. In the front and middle thirds of the back of the tongue there are thread-like and mushroom-like papillae, and leaf-like papillae prevail on the lateral surfaces. The middle part of the tongue is separated from its distal part or root by grooved papillae (papillae circumvallatae), located in a line in the form of a V-letter, at the top of which a blind hole (foramen caecum) is located, although not everyone has it, but farther from the line - a massive cluster lymphoid tissue - lingual tonsil, which is part of the lympho-epithelial ring together with other tonsils. Thread-like papillae provide tactile sensitivity of the tongue. Keratinization on filiform papillae increases with an increase in body temperature, indigestion, liver diseases, and insufficient oral hygiene. In the case of pathology, a sharp increase in the keratinization of the filiform papillae, combined with a simultaneous violation of the exfoliation of the keratinized masses, allows the papillae to dramatically lengthen, become pigmented and take on the appearance of hair (black hairy tongue). With atrophy of filiform papillae, a pattern of desquamative glossitis is observed in certain areas of the tongue. There are fewer mushroom-shaped papillae, they have a narrow base and a wider rounded top. Their height reaches 2 mm. The epithelium does not keratinize, due to which the papillae have a bright pink or red color. Clinically, mushroom-shaped papillae have the appearance of red dots scattered on the surface of the tongue. Leaf-shaped papillae are located on the lateral surfaces of the tongue in the form of 3-8 parallel folds 2-5 mm long, separated by narrow grooves. They are better expressed in newborns and in early childhood. The epithelium of leaf-shaped papillae has the highest density of taste receptors. Grooved papillae have a characteristic appearance, clear localization and number of 7-12, also contain a large number of taste buds. They slightly protrude above the surface of the mucous membrane, and most of them are immersed in the thickness of the tongue. Each papilla is surrounded by a ridge of the mucous membrane and a groove. Small salivary glands of the tongue open into this groove, the bodies of which are located in the intermuscular connective tissue. Taste receptors allow you to recognize four tastes: sweet, sour, bitter and salty. All other nuances of taste are provided by the perception of smells. The tongue contains salivary glands of three types: mixed in the front part of the tongue, mucous in the area of the tongue root, where the tongue tonsil is located, protein on the border of the body and the root of the tongue in the area of grooved papillae. The mucous membrane of the lower (ventral) surface of the tongue belongs to the lining type and is covered with a multi-layered flat non-keratinized epithelium. Histologically, the own plate forms short papillae and contains a large number of elastic fibers. There

is a submucosal layer that is adjacent to the muscles of the tongue. On the ventral surface of the tongue, it is anatomically possible to distinguish the frenulum of the tongue, located along the middle line, on both sides of which stretch fringed folds (plica fimbriata) converging at the apex. In this fold there is an opening of the serous-mucous anterior lingual gland (or Blandin-Nuna gland, glandula lingualis anterior), which is embedded in the muscles of the tip of the tongue. A pronounced venous network is located along the fringed folds, its appearance and norm depend on age and the pathology of the cardiovascular system, but even the thickening of the vascular pattern and the brownish color of this plexus is not a pathology for the tongue, but only an indicator of the patient's general state of health.

Bottomlimits the oral cavity from below. Its shape is often compared to a quadrangular pyramid with the base located at the back. Conventionally, the floor is divided into 3 zones: the front floor of the oral cavity, located in front of the frenulum of the tongue, and two areas between the lateral surfaces of the tongue and the lower jaw. Sublingual papillae (carunculae sublingualis) are clearly visible on both sides of the frenulum in the front part of the floor of the oral cavity when the tip of the tongue is raised. They open the excretory ducts of the submandibular salivary glands (Wharton's ducts), which pass along the medial edge of the hypoglossal glands. The sublingual glands have several small ducts that open directly at the bottom of the oral cavity.

Individual differences concern the torus, which can be located on the lower jaw in its lateral parts from the lingual surface, that is, visible during the examination of the floor of the oral cavity.

At the bottom of the oral cavity there is a pronounced vascular network, its appearance and rate are determined by age and the state of the cardiovascular system.

Palate. It resembles the shape of a horseshoe and forms a domed roof of the oral cavity. The palate is divided into hard and soft. The hard one belongs to the oral cavity, the soft one to the oropharynx and separates the nasopharynx. The hard palate is concave, and this concavity is mainly occupied by the tongue at rest. The hard palate is divided into primary and secondary palate. The primary is separated from the secondary by a small depression behind the central incisors - the incisive fossa, where the incisal opening opens. The front two thirds of the hard palate are formed by the incisor bone (premaxilla) and the palatine processes of the upper jaws. The horizontal plate of the palatine bone forms the posterior third. The secondary palate is represented by a median raised line - this is the median or palatal suture (raphe palati). Transverse or transverse folds (rugae palatine) on the front third of the palate serve to form a food ball. The hard palate is lined with a chewing type of mucous membrane. In the area of the suture, the mucosa is tightly attached to the periosteum and there is no submucous base. In other parts, the submucosa contains thick collagen bundles that extend from the mucosa to the bone and lie in its own plate, and the submucosa base contains adipose tissue in the front part of the palate and mucous and salivary glands in the back part. The excretory ducts of the glands open in the form of punctate openings. Depending on the features of the submucosa, the hard palate is clinically divided into 4 zones: the zone of the palatal suture; glandular zone - approximately in the front third; fat - back 2/3 of the palate; marginal zone, where the transition of the palate to the gums occurs. Hard palate torus, like the torus of the lower jaw (as exostoses are mentioned in domestic textbooks), these are benign bone growths of the jaws. They are observed in 3%-56% of adults and are more common in women, Asians and representatives of northern ethnic groups. Along with Fordyce's glands, the torus provide individual characteristics of the norm. They usually appear after the age of 20 and continue to grow throughout life. The torus of the hard palate is located along its midline, and the torus of the lower jaw is located on the lingual surface of the front part of the lower jaw, primarily in the area of the premolars. Toruses are asymptomatic, but sometimes interfere with placement of the prosthesis or cause discomfort during eating. If necessary, treatment is carried out by surgical excision, but toruses can recur, although their malignant transformation has not been described. The soft palate is the beginning of the oropharynx, the movable posterior third of the palate. It creates an incomplete membrane between the mouth and pharynx, has a median seam and is a continuous continuation of the roof of the oral cavity and the nasal mucosa of the lower floor. On the border of the hard and soft palate, on the sides of the median seam, two depressions (foveola palatina) can often be identified. These formations can be important as a reference point for determining the

limits of the prosthetic bed. In a relaxed state, the front surface of the soft palate is concave, and the back is convex. The front aponeurotic part is attached to the back border of the hard palate, and the back muscular part is suspended between the mouth and the pharynx and is called the "palatine sail" (velopharynx). The sail is elongated due to the middle free process - the tongue (uvula) and double bilateral folds:

Pharynxis the space between the oral cavity and the oropharynx, bounded above by the edge of the soft palate, below by the root of the tongue, and on the sides by the palatine brackets. During swallowing, the soft palate first tenses up to compress the lump of food between the tongue and pharynx, and then rises to block the nasal passages during swallowing. The soft palate on the side of the oral cavity and the tongue are covered with a lining nonkeratinized mucous membrane, similar to the one described above. In this area, the submucosal base is well defined. The back surface of the soft palate faces the nasopharynx and is lined with multi-row ciliated epithelium. The soft palate is intensively supplied with blood, due to which the mucous membrane has a bright pink color. There are lymph nodes in the soft palate.

The division of ENTs into departments corresponds to their different morphology, which causes limited pathological processes - cheilitis, glossitis, palatinitis, gingivitis, periodontitis. Thus, hyperkeratosis develops more often in areas where the epithelium of the cornea is keratinized and normal: the red border of the lips, the corners of the mouth, the line of closing the teeth, the back and lateral surfaces of the tongue.

1. Theoretical questions

Questions for self-control

2 Practical tasks

1. Prepare an essay on the topic: "Clinical and morphological features of the organs of the dento-jaw system"

2. Make a graph of the logical structure "Clinical-morphological features of the organs of the maxillofacial system and the oral cavity".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

22 Pathomorphology: nats. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.

23 The basics of pathology according to Robbins: trans. 10th Eng. ed.: in 2 volumes, T. I / Vinay Kumar, Abdul K. Abbas, John K. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.

24 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

25 Skrypnikov P.M., Skrypnikova T.P., Shynkevich V.I., Tovma V.V. Individual and age-related clinical and morphological features of the mucous membrane of the oral cavity: study. manual - Poltava: LLC "ASMI", 2016. - 102 p.

Electronic information resources

57 http://moz.gov.ua- Ministry of Health of Ukraine
58 www.ama-assn.org– American Medical Association /American Medical Association
59 www.who.int- World Health Organization
60 www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
61 http://bma.org.uk– British Medical Association
62 www.gmc-uk.org- General Medical Council (GMC)
63 www.bundesaerztekammer.de– German Medical Association
64 http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
http://www.webpathology.com/- Web Pathology

Topic #9: "Dental manifestations of other diseases"
Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:
The student should know:
1. Classification and essence of changes associated with diseases that are dental manifestations of other diseases.
2. Etiology, pathogenesis, pathological anatomy of these pathological conditions.
3. Results, complications of diseases associated with dental symptoms.
:The student should be able to:
1. Classify the pathological processes of diseases that are dental manifestations of other diseases.
2. To characterize the etiology, pathogenesis and morphological essence of the pathology of diseases that are dental manifestations of other diseases.

Topic content:

In some diseases, lesions typically develop in areas of the oral cavity, on the mucous membrane of the oral cavity, where there is a submucosal layer. The classification according to pathogenesis divides all diseases and changes in the SOPR into two main groups: primary, or intrinsic, and secondary, that is, symptomatic. Primary/intrinsic diseases include diseases that arise as a result of the direct impact of a pathogenic stimulus on the ENT (these are primarily dental diseases). The secondary/symptomatic ones include symptom complexes that manifest themselves on the SOPR in diseases of various organs and systems of the body. They are presented in the table.
Secondary symptomatic dental manifestations of other diseases
Nosological units and their etiology
Symptom complexes, dental manifestations
Exogenous infections (Class I)
1.1. Bacterial
Scarlet fever
Scarlet fever. "Raspberry Tongue"
Diphtheria
Diphtheria sore throat, possible gingivitis
Tularemia

Islet necrosis of the mucous membrane of the pharynx and tonsils
Typhoid
"Funginous tongue" (dry, brown, with cracks)
Syphilis
Hard chancre, papules, erythema, gum depending on the stage
Gonorrhea
Lilac-red erythema, erosion
Miliary-ulcerative tuberculosis
Papules, ulcers with pitted edges, at their bottom "Trel's grains"
Tuberculous lupus
Lupus, with diascopy - the symptom of "apple jelly"
Leprosy
Pink infiltrates, ulcers with a bumpy bottom and a gray coating
1.2. Viral
Flu
Granular rash of the "dew" type on the mucous membrane of the palate. Ulcerative-membranous elements, blisters
OX
Hairy leukoplakia, Kaposi's sarcoma, nonspecific ulceration of the SOPR, symptom complexes of autoinfectious stomatitis and periodontal syndrome are often observed
Botkin's disease
Yellowness of the sclera and mucous membrane of the hard palate. Catarrhal gingivitis
Murrain
Vesicles, erosions on the lips. Vesicles on the skin around the mouth and nail bed
Mononucleosis
Catarrhal-ulcerative gingivitis and tonsillitis
Measles
Filatova-Koplyk spots. Measles rash on the skin of the face and body
Varicella
Vesicles, erosions on the SOPR. Vesicular rash on the skin
Shingles
Bubbles and dark crusts on the mucous membrane of the cheeks and the skin of the face, located unilaterally along the course of the nerve branches
2. Dermatostomatitis with an autoimmune component (Class XII)
Benign non-acantholytic pemphigus itself is a SOPR
On the SOPR - one or more blisters, erosions are possible. Symptom
Pemphigus:
a) vulgar
Blisters are short-lived, burst easily; erosion, Nikolsky's symptom is positive
Pemphigus: b) vegetative

Necrotic films, erosions with vegetation on the bottom, Nikolsky's symptom is positive
Pemphigoid
Blisters, papules, Nikolsky's symptom is negative
Red lichen planus, pemphigoid form
A papular rash of pearl color, blisters in various areas of the SOPR

Lupus erythematosus
There is a "butterfly" on the skin of the face, erythema, atrophy in the center, on the periphery of the scales
Behcet's disease
Recurrent aphthae on SOPR can be combined with conjunctivitis, retinitis and atrophy of the optic nerve
Chronic recurrent aphthous stomatitis
Aphthous-ulcer syndrome
3. diseases of the blood and hematopoietic organs (Class III)
Acute leukemia
Hemorrhagic syndrome on the SOPR and in the subcutaneous tissue. Ulcerative-necrotic gingivitis, stomatitis
Chronic leukemia
Hypertrophy of gums, lymphoid tissue. Aphthae, candidiasis are possible
Radiation sickness is acute
Ulcerative gingivitis, bleeding gums, loss of taste
Radiation sickness is chronic
Post-radiation periodontitis
Agranulocytosis
Agranulocytic angina, periodontal syndrome with gum ulceration
Waukesha's disease
Cherry-red hypertrophic gingivitis, stomatitis
Werlhof's disease
Hemorrhagic syndrome and spontaneous bleeding from the gums and nose
Addison-Birmer anemia
Gunter-Miller glossitis
Iron deficiency anemia
Creeping atrophy of the filiform papillae of the tongue from the tip to the root of the tongue
4. endocrine diseases, digestive disorders and metabolic disorders (Class IV)
Diabetes
Periodontal symptom complex, the smell of acetone is possible
Itsenko-Cushing syndrome
The SOPR is swollen. Imprints of teeth on the mucous membrane of the cheeks, lateral surfaces of the tongue
Addison's syndrome
There are numerous black-gray spots in various areas of the swollen SCC
Hypovitaminosis A
Mild keratosis SOPR, dry skin
Hypovitaminosis S
Hemorrhagic syndrome. Periodontal symptom complex
Hypovitaminosis B2
Zaida ariboflavinova
Hypovitaminosis B12
Gunter-Miller glossitis
Hypovitaminosis RR
The tongue is the color of the "cardinal mantle"

Hypovitaminosis K
Catarrhal gingivitis, bleeding gums
Hypovitaminosis B6
Paresthetic syndrome in the tongue and other parts of the ENT
5. diseases of the nervous system (Class VI)
Glossodynia
Periodically, a burning sensation is felt in the tongue, other areas of the digestive system, possibly the skin (Geda's zone)
Glossalgia
Pain in the tongue
Melkerson-Rosenthal syndrome
Triad: paresis of the facial nerve, swelling of the lip (unilateral), folded tongue
Collagenosis
Microstomia
Scleroderma
"Face of Dante". The skin of the face is initially purplish-red, over time it acquires an ivory shade
6. exogenous drug intoxication (Class XIX)
Mercury gingivostomatitis
Hypersalivation. Gray-dirty border on the gingival margin, possible erosions
Bismuth gingivostomatitis
Along the gingival margin and around the erosion, there is a border of purple color
Lead gingivostomatitis
There is a black border along the gingival margin and on the interdental papillae
Aurostomatitis
Aphthous rash on the tongue
Diphenine hypertrophic gingivitis
Hypertrophy of the gingival margin and interdental papillae affecting the crowns of the teeth
For local use of tablets (antibiotics):
a) "penicillin tongue"
The tongue is slightly swollen, the back is bluish-red, in some places atrophy of the filiform papillae
b) "tetracycline tongue"
The back of the tongue, sometimes the tongue is covered with brick-red spots, blisters and erosions are possible
c) "levomycetin glossitis"
The tongue is swollen, covered with bright red spots. The skin is often ash-colored
7. Manifestation of allergic reactions (Class XIX)
7.1. immediate type
Anaphylaxis
General symptoms of anaphylaxis
Angioedema
Swelling of the lower third of the face, lower lip, tongue, throat
7.2. Slow type
Contact allergic cheilitis
Lips are bright red, dry, sometimes with a blistering rash
Catarrhal and hemorrhagic gingivitis
Against the background of bright red gums, a hemorrhagic rash is noticeable

Papular stomatitis
Against the background of the bright red mucous membrane of the cheeks, there are visible papules reminiscent of ChPL
Vesiculobullous stomatitis
Against the background of the bright red mucous membrane, especially the cheeks, vesicles and blisters are present, erosions are possible
Allergic glossitis
The back of the tongue is often devoid of thread-like papillae, smooth, shiny and dry
Erythema multiforme (BE) a) infectious-allergic form
Erythema, blisters, papules on the mucous membrane, often in combination with skin lesions - papules
b) toxic-allergic form
Erythema, blisters, papules on the SOPR, the skin of the extremities
Stevens-Johnson syndrome
Erosive-membranous stomatitis, conjunctivitis, vesiculopapular rash on the skin of the extremities
Lyell's syndrome
Generalized vesicular-erosive damage of the ENT and skin

miliums Milia are divided into primary and secondary. Congenital (primary) milia of newborns make up the vast majority. But they can occur in connection with some genodermatoses or sporadically, without any apparent reason. Secondary milia can be associated with an underlying skin disease, medication, or trauma. The typical appearance of a milium is a dome-shaped papule with a diameter of 1–3 mm and a smooth surface. Can be single or multiple. Congenital milia predominate on the face, where the nose is most often involved. Benign secondary (acquired) milia in children and adults are formed on the forehead, cheeks, eyelids and genitals. Milia can be clustered and form plaques in the form of an erythematous raised spot with numerous "spikes" of milia. The size of these formations can reach several centimeters. Such multiple eruptive milia can be selectively localized on the face, upper body, proximal parts of the limbs and in the groin. The classification of milia is known (Berk and Bayliss, 2008). Congenital: • benign primary milia of children and adults; • milia in the form of plaques; • nodular grouped milia; • multiple eruptive milia; • pigmentless nevus with milia; • genodermatosis-associated. Secondary (acquired) milia: • associated with diseases; • related to medications; • associated with trauma. Congenital milia are observed in almost half of healthy newborns and are usually present at birth, although their appearance may be delayed in premature babies. They usually disappear spontaneously within a week. Benign acquired milia of children and adults also disappear spontaneously; however, like other acquired milia, they tend to persist without treatment. Multiple eruptive milia are described in the literature as acquired and widespread milia that occur more often suddenly over several weeks or months. Multiple eruptive milia can be associated with genodermatosis or be inherited in an autosomal dominant pattern without other visible abnormalities. However, in most cases, they arise spontaneously. Genodermatosis-associated milia have been reported in basal cell nevus syndrome, Rombo syndrome, Brooke-Spiegler syndrome, congenital pachyonychia type 2, and atrichia with papular lesions. In children, traumatic milia most often occur after cuts or burns. Milia have been reported after skin grafting. Milia can accompany blistering skin diseases. Epidermolysis bullosa congenita and porphyria cutaneous tarda are classic examples. Rare reports of milia have been associated with the use of topical corticosteroids. Histopathological studies suggest that primary

milia arise from the lower parts of the funnel-shaped sebaceous sac of downy hair, while secondary ones are more often formed from eccrine ducts. Epidemiology of milia: the frequency of congenital - in 40%-50% of healthy newborns. Babies born prematurely are less likely to be affected, as the onset of milia may be delayed. Racial bias is not observed. Gender: in general, milia occur with equal frequency in men and women. But milia in the form of plaques are more common in women. They can occur at any age, and most often - during the newborn period. Milia in the form of plaques are most common among middle-aged women. The prognosis of congenital benign milia is spontaneous disappearance without scarring. Acquired forms may require treatment. Epstein's pearls are a special case of pediatric milia (milia), which are also benign, keratin cysts and usually appear as tiny white papules on the face of a newborn.
In some diseases, lesion elements typically develop in areas where there is a submucosal layer, or, conversely, on the attached mucosa. In many diseases (vemiculosis, leukoplakia, lichen planus, Manganotti cheilitis, limited hyperkeratosis, recurrent herpetic stomatitis), the assessment of the state of epithelial cells is of diagnostic importance. And in some cases (pemphigus, malignancy), changes in epitheliocytes are pathognomonic.
In case of hyperkeratoses, the clinic uses an index assessment of the state of epithelial cells in pathology, in fact, these are methods of cytology. Keratinization index (IK) is determined by counting the percentage of anucleated cells in a cytological preparation (N.F. Danylevsky, 1997). The degree of destruction of epithelial cells (Matveeva L.A., 1977), which is based on the identification of five classes of destruction, differs depending on changes in the structural integrity of cellular elements. The nuclear-cytoplasmic ratio (NCR) of 100 epithelial cells makes it possible to assess the stages of differentiation of each epitheliocyte.
The nature of the elements of the lesion in pathological conditions has a diagnostic value: a blister is a hollow structure formed inside the epithelium, the main element of herpes viral diseases. A blister is also a hollow formation of various sizes, it is formed inside the epithelium or subepithelially. Blisters are observed in erythema multiforme (BE), drug-induced lesions, pemphigus, vesicular-vascular syndrome, etc. In the thickness of the epithelium of the red border of the lips and the skin of the lips, the formation of a hollow pustule filled with purulent exudate is possible, and subepithelially - ranulas - cysts of the small labial gland.

1. Theoretical questions
Questions for self-control
2 Practical tasks
1. Prepare an essay on the topic: "Dental manifestations of other diseases"
2. Make a graph of the logical structure "Dental manifestations of other diseases".

3. Test tasks for self-control:

4. Individual tasks
1. Make an outline on this topic

5. List of recommended literature:
Main:
1 Pathomorphology: national. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.

2 Fundamentals of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.
3 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

Additional:
1. Skrypnikov P.M., Skrypnikova T.P., Shynkevich V.I., Tovma V.V. Individual and age-related clinical and morphological features of the mucous membrane of the oral cavity: study. manual - Poltava: LLC "ASMI", 2016. - 102 p.

Electronic information resources

1 http://moz.gov.ua – Ministry of Health of Ukraine
2 www.ama-assn.org – 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 - Pathology Laboratory
http://www.webpathology.com/ - Web Pathology

Topic #10: "Precancerous changes and tumors of the lips, tongue, soft tissues of the oral cavity"
Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:
The student should know:
1. Clinic, diagnosis, treatment of soft tissue tumors of SCD and PR.
2. Classification and essence of changes associated with tumors of soft tissues of SCD and PR.
3. Etiology, pathogenesis, pathological anatomy of these pathological conditions.
4. Results of treatment, complications of diseases associated with tumors of soft tissues (ShLD) and PR.
5. Types and clinic of the most common benign tumors of soft tissues of SCD and PR.
6. To master the method of examination of patients with the specified pathology, using auxiliary diagnostic methods.
7. To learn the concept of oncovigilance.
8. To consider the indications for the choice of treatment methods and the method of its implementation in benign soft tissue formations of SCD.
9. To study the principles of ambulatory surgery for benign soft tissue tumors of SCD and PR.
:The student should be able to:
1. To classify the pathological processes associated with tumors of soft tissues of SCD and PR.
2. To characterize the etiology, pathogenesis and morphological essence of the pathology associated with soft tissue tumors of SCD and PR.

3. To master the essence of tumor-like processes, etiological features, to get acquainted with all types of tumor-like formations of soft tissues (soft tissue tumors of SLD) and PR, their statistics and age characteristics.
4. To use in practice the knowledge about the clinic, diagnosis, differential diagnosis and treatment of tumor-like diseases of PR and SLD.
5. Master the clear differentiation between tumors and tumor-like formations in etiopathogenetic, diagnostic, therapeutic and preventive aspects, study the classifications of tumor-like diseases of SCD and PR.
6. Based on the classification, learn to formulate a diagnosis with tumor-like processes in SCD and PR.

Topic content:

To classify the pathological processes associated with tumors of soft tissues of SCD and PR.
1 To characterize the etiology, pathogenesis and morphological essence of the pathology associated with soft tissue tumors of SCD and PR.
2 To learn the essence of tumor-like processes, etiological features, to get acquainted with all types of tumor-like formations of soft tissues, tumors of soft tissues (SLD) and PR, their statistics and age characteristics.
3 Use in practice knowledge about the clinic, diagnosis, differential diagnosis and treatment of tumor-like diseases of PR and SLD.
4 To master the clear differentiation between tumors and tumor-like formations in the etiopathogenetic, diagnostic, therapeutic and preventive aspects, study the classifications of tumor-like diseases of SCD and PR.

1. Theoretical questions
Questions for self-control
1. How is the clinical examination of SCD and PR performed?
2. What additional methods of examination are used to establish the final diagnosis of neoplasms?
3. Scheme of formulating a clinical diagnosis for neoplasms of SCD and PR.
4. Basic methods of cancer treatment.
5. Definition of the concept of "tumor" and "tumor-like formation".
6. Patterns of tumor-like processes.
7. Repetition of the basic principles of classification of tumor processes and mastering the classification of tumor-like processes.
8. The difference between benign tumors and tumor-like formations.
9. Possible reasons for the development of tumor-like formations of soft tissues of SCD and PR.
10. Definition of the concept of "cyst".
11. Classifications of benign neoplasms in SLD and PR, principles of their construction.
12. Frequency of certain types of neoplasms of SCD and PR.
13. How dangerous is the localization of tumors in SCD?
14. How is the clinical diagnosis of tumors in SCD formulated?
15. What are the methods of diagnosis in SCD oncology?
16. Papilloma: definition, clinic, diagnosis, differential diagnosis, treatment, prevention
17. Soft tissue fibromyoma of SCD: forms, clinic, diagnosis, differential diagnosis, treatment, prevention.

18. Lipoma: typical locations, clinic, diagnosis, differential diagnosis, treatment, prevention.
19. Rhabdomyoma, myoblastoma: origin, clinic, diagnosis, differential diagnosis, treatment, prevention.
20. Myxoma: origin, clinic, diagnosis, differential diagnosis, treatment, prevention.
21. Tumors of the tongue, features of their clinic and treatment, prevention.
22. Papillomatosis: clinic, diagnosis, differential diagnosis, treatment.
23. Gum fibromatosis: causes, classification, clinic, diagnosis, differential diagnosis, treatment, prognosis.
24. Neurofibromatosis: clinic, diagnosis, treatment.
25. Dermoid cyst: causes, clinic, diagnosis, differential diagnosis, treatment.
26. Classification of cysts, causes of their occurrence, clinic, diagnosis, differential diagnosis.

2 Practical tasks
1. Prepare an abstract on the topic: "Precancerous changes and tumors of the lips, tongue, and soft tissues of the oral cavity."
2. Make a graph of the logical structure ""Tumors of the lips, tongue, soft tissues of the oral cavity".

3. Test tasks for self-control:

4. Individual tasks
1. Make an outline on this topic

5. List of recommended literature:
Main:
1 Pathomorphology: national. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.
2 Fundamentals of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.
3 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

Additional:
1. Pathological anatomy: a textbook. Trans. from Russian 4th edition / Strukov A.I., Serov V.V. - Kharkiv.: Fakt, 2004. - 864 p.
2. Skrypnikov P.M., Skrypnikova T.P., Shynkevich V.I., Tovma V.V. Individual and age-related clinical and morphological features of the mucous membrane of the oral cavity: study. manual - Poltava: LLC "ASMI", 2016. - 102 p.
3. Havrilyuk O.M., Servetnyk M.I., Vovk V.I. Under the editorship of Pospisil Yu.O. Methodological recommendations for students of the 3rd year of medical and stomatological faculties. Module 1 "General pathology". Content module 4 "Tumors". - Lviv: PP "Aral", 2016. - 60 p.
4. Tomashova S.A., Hrytsyna I.V. Servetnyk M.I., Vovk V.I., Bisyarin Y.V., Kuzyk Y.I., Vovk V.V. Under the editorship of Pospisil Yu.O. Pathomorphology workbook. Section "Special pathology". Methodological developments for students of the Faculty of Dentistry. - Lviv, 2011. -

118 p.

Electronic information resources

1 http://moz.gov.ua – Ministry of Health of Ukraine
2 www.ama-assn.org – 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 - Pathology Laboratory
http://www.webpathology.com/ - Web Pathology

Topic No. 11: "Disorders of the face, neck and oral cavity"

Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:
The student should know:
1. Classification and essence of changes associated with congenital defects of SCD.
2. Etiology, pathogenesis, pathological anatomy of congenital defects of SCD.
3. The essence of congenital defects, in particular - congenital defects of SCD.
4. Embryogenesis of SCD in terms of the development of possible pathological disorders in the specified process.
5. Statistics of birth defects of the cervical spine, neck and RP.
6. How to formulate a diagnosis for congenital defects of SCD on the basis of their classification.
7. Treatment methods for non-union of the upper lip.
8. Principles of treatment of nonunion of the palate.
9. Methods of surgical intervention for defects in the development of the frenulum of the tongue and the upper lip, and the small hairline
:The student should be able to:
1. Classify congenital defects of SCD, neck and RP.
2. To characterize the etiology, pathogenesis and morphological essence of congenital defects of SCD.

Topic content:

Defects of SCD, neck and RP have the following division:
1. Non-union of tissues of the maxillofacial area: - lips; - coloboma; - macrostoma; - cellular process; - the palate.
2. Non-union of SCD tissues, combined with other defects (syndromes).
3. Orofacial dysplasia of soft tissues and bones of the face.

4. Absence of an organ or its part and (or) tissue.
5. Defects in the development of individual organs (auricle, nose).
6. Malformations of the frenulum of the lips and tongue.
7. Atresia of the nasal and auditory passages, microstoma.
8. Noritsia.

Statistics. At the beginning of the 20th century children with birth defects in Ukraine were born in a ratio of 1:2000; in 1960-1970 - 1:1000; in 2000-2002 - 1:800-1:900. In most European countries, this ratio is now 1:600. The tendency to a significant decrease in the number of such children is not expected, since environmental, economic, social and other conditions affecting the health of parents and their children are not improving.

The etiology of SCD, neck and RP defects is multifactorial in nature. The conducted clinical and geneological studies showed that in 85% of patients, the defects of SCD were sporadic in nature, and in 15% - familial.

Non-union of the lip and palate has a polyetiological nature, in which both genetic and teratogenic factors are involved. The genes responsible for these deformities have now been identified, namely the folate receptor gene and the genes encoding familial growth transforming factor or retinoic acid receptor. But despite the success of genetics in revealing the causes of the formation of nonunion, it is still impossible to predict the birth of a child with defects of the maxillofacial area. The occurrence of this defect is determined by exo- and endogenous factors. Exogenous should include physical (radioactive radiation, ionizing radiation; mechanical effect on the fetus - intrauterine pressure, chronic trauma, uterine tumors, increased body temperature of a pregnant woman), chemical (occupational harm), biological (influenza, epidemic parotitis, rubella viruses; hypoxia fetus), violation of the ecological balance, which has a teratogenic effect on the body of the fetus (atmospheric pollution, intensive development of the chemical industry, the presence of pesticides in food products). Endogenous factors include diseases of the parents (chronic diseases of the genital organs - trichomoniasis, infectious diseases of the mother, toxoplasmosis, hypovitaminosis, hypervitaminosis-A), the age of the mother (with age, the probability of having a child with a nonunion increases), stressful situations that increase the likelihood of the formation of intrauterine malformations of the fetus, bad habits of the parents (smoking, drug use, alcohol), use of pharmacological drugs during pregnancy only in the first trimester - 6-11 weeks. All these factors to some extent create prerequisites for the development of anomalies of the maxillofacial area. In 30% of children, congenital non-union of the lip and palate is hereditary. Pathogenesis. According to His, Dursy, during the genesis of a human embryo on the 15th-20th day in its main part, primary oral recesses, or naso-oral fossa, appear. It has a pentagonal shape and is surrounded by five protrusions: from above - a frontal process, from the sides - two maxillary processes, and from below - two mandibular processes (the so-called first gill arch). They consist of ecto- and mesodermal layers. The hard and soft palate, the outer part of the upper lip are formed from the maxillary processes. The frontal process is divided into intranasal, which form the middle part of the upper lip and the intermaxillary bone with incisors. If during this period under the influence of some factors there is a delay in the growth of the facial area, then the fusion of processes may not occur. The presence of congenital non-union of the upper lip and palate affects the child's weak body from the very beginning. Children with congenital non-unions of the lip and palate have a high morbidity rate, which, according to most researchers, increases with age and causes their unfavorable somatic condition. Such children are 4-5 times more likely to suffer from diseases of the ENT organs (100% of children have hearing loss), 25% have disorders of the cardiovascular system, 20% - organs of vision, 15% - urinary system, 10% - musculoskeletal system. The following types of congenital

non-union of the lip are distinguished: 1) by the depth of non-union (apparent, hidden); 2) depending on the localization of nonunion in the transverse plane (lateral - 99% and medial - 1%). Lateral, in turn, divided depending on the side of non-union into unilateral - 82% (left-sided more often than right-sided) and bilateral - 17%; bilateral non-union of the lip can be symmetrical and asymmetrical; 3) by the length of non-union in the sagittal plane of the lips (partial, full); 4) isolated and combined (with non-irrigation of the cellular process of the upper jaw, palate). The clinical picture of congenital nonunions of the lip has characteristic features and usually does not cause difficulties during diagnosis. In the presence of isolated hidden non-unions of the lip, the defect and deformation of the soft tissues is the least, even sometimes it is only a retraction of the skin in the projection of the column of the upper lip. Non-union is partial in the presence of the floor of the nasal passage. In the case of through defects of the upper lip, the clinical picture is aggravated. Unilateral isolated nonunions of the upper lip appear as a defect of the upper lip on one or the other side. In this case, the red border consists of two fragments, on the larger fragment it rises to 1/3-1/2 the height of the upper lip, and sometimes more. The column of the upper lip on the unfused side is also divided into two parts, the height of the column on the large fragment is always reduced, and on the small - almost normal. The wing of the nose is flattened from the side of non-union (the muscles of the upper lip on a small fragment are woven into the base of the wing of the nose, which leads to its fixation in a pathological position), the tip is also flattened and, together with the septum, is shifted to the healthy side, the bottom of the nasal passage is missing - its " replaces" the defect. If nonunion of the upper lip is combined with nonunion of the cellular process, then a large fragment of the latter (due to the growth of the blade) moves forward and up (as if it turns out), the frenum of the upper lip is always short. In this case, the small fragment is turned medially downward and looks underdeveloped. Bilateral non-union of the lip is the most severe defect of the maxillofacial area both in terms of the clinical picture and in relation to the provision of medical care. The biomechanism of its development is significantly different from unilateral ones. In the presence of such a defect, the upper lip is divided into three fragments, the soft tissues on the lateral fragments are quite pronounced, the height of the columns is slightly reduced, the red border and the muscle layer are usually well defined. The medial fragment is represented by the intermaxillary bone, soft tissues of the prolabium (the central part of the upper lip is limited by columns), a red border and a septum of the nose. The timing and type of surgical intervention and the need for orthodontic treatment depend on its position (presence of a protrusion of the intermaxillary bone, its rotation, displacement to one side), its relationship with the lateral fragments and the severity of soft tissues on it. There is always a shortage of soft tissues on the medial fragment (both the red border and the skin part of the upper lip), especially in height. The oral cavity is shallow. With this type of non-union, significant deformation of the nose occurs. The septum of the nose is always very short, the medial legs of the wing cartilages are underdeveloped. Sometimes the septum of the nose is drawn to the soft tissues of the lip, the tip of the nose is bifurcated, the wings are stretched and the nose looks like a sheep. The somatic condition of such children depends on the type and degree of nonunion, the presence of concomitant diseases. In the case of isolated nonunions of the lip, the child can eat well, so it develops normally physically, gains weight, which is very important, especially in the 1st year of life. In the case of through (unilateral and bilateral) nonunion of the upper lip, combined with nonunion of the palate, the functions of sucking and breathing are impaired. This leads to the development of various inflammatory processes of the upper respiratory tract and ENT organs, anemia and hypotrophy. So, for example, 30% of such children have contraindications to surgical treatment of a general somatic nature. During a detailed examination, thymomegaly, otitis media, chronic kidney diseases, etc. are revealed. Only additional research

methods can detect concomitant diseases, which is very important for preparing the child for surgery. There are many methods of cheiloplasty, both unilateral and bilateral nonunions. Depending on the fabric cut, they are divided into 2 - linear and rectangular. But they all pursue the same goal of restoring the anatomical integrity of the elements of the lip (red border, columns, nasal passage, muscles, oral cavity) and its functional capacity. The stages of the operation include: dissection of the tissues of the skin part of the lip fragments using one of the methods, mobilization of the muscles of the base of the wing of the nose, and the mucous membrane of the red border, the border of the mouth, suturing the edges of the wound in layers, taking into account the new retrotransposition of tissues. The optimal term of cheiloplasty for congenital unilateral nonunions of the upper lip in the absence of general contraindications is 3-6 months. With bilateral nonunions - 6-12 months. But they all pursue the same goal of restoring the anatomical integrity of the elements of the lip (red border, columns, nasal passage, muscles, oral cavity) and its functional capacity. The stages of the operation include: dissection of the tissues of the skin part of the lip fragments using one of the methods, mobilization of the muscles of the base of the wing of the nose, and the mucous membrane of the red border, the border of the mouth, suturing the edges of the wound in layers, taking into account the new retrotransposition of tissues. The optimal term of cheiloplasty for congenital unilateral nonunions of the upper lip in the absence of general contraindications is 3-6 months. With bilateral nonunions - 6-12 months. But they all pursue the same goal of restoring the anatomical integrity of the elements of the lip (red border, columns, nasal passage, muscles, oral cavity) and its functional capacity. The stages of the operation include: dissection of the tissues of the skin part of the lip fragments using one of the methods, mobilization of the muscles of the base of the wing of the nose, and the mucous membrane of the red border, the border of the mouth, suturing the edges of the wound in layers, taking into account the new retrotransposition of tissues. The optimal term of cheiloplasty for congenital unilateral nonunions of the upper lip in the absence of general contraindications is 3-6 months. With bilateral nonunions - 6-12 months. dissection of the tissues of the skin part of the lip fragments using one of the methods, mobilization of the muscles of the base of the wing of the nose, and the mucous membrane of the red border, the border of the mouth, suturing the edges of the wound in layers, taking into account the new retrotransposition of tissues. The optimal term of cheiloplasty for congenital unilateral nonunions of the upper lip in the absence of general contraindications is 3-6 months. With bilateral nonunions - 6-12 months. dissection of the tissues of the skin part of the lip fragments using one of the methods, mobilization of the muscles of the base of the wing of the nose, and the mucous membrane of the red border, the border of the mouth, suturing the edges of the wound in layers, taking into account the new retrotransposition of tissues. The optimal term of cheiloplasty for congenital unilateral nonunions of the upper lip in the absence of general contraindications is 3-6 months. With bilateral nonunions - 6-12 months.

Cleft palate is a cleft palate that results from the failure of the two halves of the palate to fuse during embryonic development. Only part of the palate may be affected (for example, only the soft palate or uvula), or the cleft may run along the entire length of the palate. Congenital non-union of the palate is classified as follows: 1. Explicit (through) and hidden (non-through). 2. Complete and incomplete (partial). 3. Unilateral, bilateral and intermediate. 4. Hard and (or) soft palate. 5. Combinations with non-union of the lip and (or) cellular process. Complaints In the case of congenital non-union of the palate, the child's parents complain of a defect in the tissues of the palate and food getting into the nose during feeding, which causes coughing and choking. At an older age, the child "mumbles", pronounces words indistinctly. Clinic. In the case of medial isolated non-unions during the examination of the oral cavity, the cellular process is intact, the hard and soft

palate consist of two fragments, the blade is located in the middle. In the presence of a hidden isolated non-union of the hard and soft palate, the following is revealed: on the hard palate, there is an area of tissue retraction in the middle, which shines through with a bluish color, and during palpation, the absence of bone tissue in this area is determined; on a soft one, such tightening of tissues is clearly visible during the child's pronunciation of the vowel "A" or a cry. Often, isolated non-unions are accompanied by defects in the development of the musculoskeletal system and other systems. That is why such children need a thorough examination. In the presence of one-sided through non-union, the cellular process, hard and soft palate consist of two fragments, one of them is bigger, the other is smaller; the ploughshare is always fused with the larger one throughout or partially (by 1/3, 2/3 of the length). The oral cavity is connected to the nasal cavity from the non-union side. Usually, the small fragment is underdeveloped, shorter, sinks inward, and the larger one is turned outward. In the case of bilateral through-and-through non-unions, the cellular process consists of three fragments - two lateral and medial. The latter is represented by the intermaxillary (incisor) bone, a part of the cellular process with incisors and a blade lying between the lateral fragments in the middle. In the remaining cases, the intermaxillary bone protrudes forward - this is the so-called protrusion, which can be congenital or acquired as a result of improper feeding of the child. A significant protrusion of the intermaxillary bone requires orthodontic preoperative treatment. The lateral fragments are represented by the cellular process and horizontal plates of the palatine bone (in most cases, they are shifted medially and backward). The oral cavity is connected to the nasal cavity. In the presence of all types of nonunions, fragments of the soft palate are usually short and may be asymmetric. The middle part of the pharynx is wide. In older children, hypertrophied tonsils and adenoid vegetations on the back wall of the pharynx are identified. Treatment of children with congenital through and isolated non-union of the palate is complex and includes surgical, orthodontic, speech therapy and psychological rehabilitation. Depending on the type of nonunion of the palate, the somatic condition of the child, the terms and sequence of this or that type of treatment are determined, but at each stage they interact and have their own patterns. Tasks of uranostaphyloplasty: 1. closing the connection of the nasal cavity with the oral cavity, 2. narrowing of the middle part of the pharynx, 3. lengthening of the soft palate. The basic technique of gentle uranostaphyloplasty involves the following stages: 1. Cutting out mucous-oxidizing flaps. 2. Exfoliation of these flaps to the border of the hard and soft palate. 3. Separating them from the rear edge of the horizontal plates of the palatine bone. 4. Osteotomy of the back wall of the palatine opening and removal of the vascular-nerve bundle. 5. Removal from the hook of the pterygoid process of the sphenoid bone of the tendon of the tensor muscle of the palatine curtain. Cutting of mucous-oxidizing flaps. 2. Exfoliation of these flaps to the border of the hard and soft palate. 3. Separating them from the rear edge of the horizontal plates of the palatine bone. 4. Osteotomy of the back wall of the palatine opening and removal of the vascular-nerve bundle. 5. Removal from the hook of the pterygoid process of the sphenoid bone of the tendon of the tensor muscle of the palatine curtain. Cutting of mucous-oxidizing flaps. 2. Exfoliation of these flaps to the border of the hard and soft palate. 3. Separating them from the rear edge of the horizontal plates of the palatine bone. 4. Osteotomy of the back wall of the palatine opening and removal of the vascular-nerve bundle. 5. Removal from the hook of the pterygoid process of the sphenoid bone of the tendon of the tensor muscle of the palatine curtain.

The defect in the development of the frenulum of the tongue is manifested by a decrease in length and an atypical place of attachment of the "legs", as well as a change in its thickness. It can be represented both by a fold of the mucous membrane (a thin frenulum) and by strong cords with interweaving of connective tissue and muscle fibers. Normally, the frenulum of the tongue is

attached 1-1.5 cm below its tip. The second point of attachment is in the area of the floor of the oral cavity along the median line behind the sublingual papillae. More often, abnormalities of the frenulum of the tongue are manifested by its attachment in an atypical place, significant prominence of the tongue, and a decrease in length up to its fusion with the floor of the oral cavity. All this limits movements or leads to immobility (contracture) of the tongue and its unnatural position. In the case of a short frenulum of the tongue, there are 2 options for attaching it: 1) to the tip of the tongue and the tissues of the floor of the oral cavity in front of the sublingual papillae; 2) to the tip of the tongue and the cellular process. Complaints of parents are different and depend on the age of the child: 1. From the first days of the child's life - for violation of the act of sucking. One feeding lasts 50-60 minutes, the child gets tired, cries, falls asleep at the breast, swallows a lot of air. Often, babies with a short tongue frenulum are transferred to artificial feeding because they refuse to suckle. 2. At the age of 6-9 months. - on a noticeable lag in the growth of the frontal part of the lower jaw in the presence of connective tissue and muscle elements in the frenulum of the tongue. 3. At the age of 5-6 - for pronunciation disorders (more often children do not pronounce the letters "r" and "l"). 4. At the age of 7-9 years - on the incorrect location of the front teeth on the lower jaw, malocclusion, and in the case of attachment of the frenulum to the gingival margin of the lower jaw, there are complaints of inflammation of the gums in the area of its front teeth, bleeding from the gums during brushing and eating. Sometimes it is possible to break the frenulum (in case of excessive tongue movements), then there will be complaints about the presence of short-term bleeding and pain at the place of the frenulum rupture. Clinic. In babies, the frenulum of the tongue is represented only by a mucous membrane, so it is thin and short. In older children, during the examination of the oral cavity, the tongue is of normal size, its movements are limited. The child cannot touch the palate with the tip of the tongue and lick the upper lip, and when trying to do this, the tip of the tongue may split. The frenulum of the tongue is often short, represented by a dense connective tissue cord or a duplication of the mucous membrane, its attachment points are shifted outward. Sometimes it is practically absent, that is, the tongue is attached to the bottom of the tissues of the oral cavity, which causes its contracture. With age, deformation of the frontal part of the lower jaw, improper arrangement of teeth in this area, distal bite is revealed. The phenomena of local periodontitis in the area of the named group of teeth are often expressed - swollen gums that bleed easily and lag behind the necks of the teeth, periodontal pockets with plaque and an unpleasant smell, etc. Treatment. Newborns with a short frenulum of the tongue, which leads to a violation of the sucking function, in the first months of life perform a frenulotomy - transverse crossing of the fold of the mucous membrane in the condition of a thin frenulum. Sometimes this manipulation is performed with application anesthesia. In the presence of a dense, wide mass in early infancy, frenulum plasticity is performed according to O.O. Limberg (Z-plasty) or Diefenbach (V-plasty) under general anesthesia. Children with a short, thin frenulum of the tongue at the age of 3-6 years are initially shown myogymnastics, which helps to stretch the frenulum and increase the mobility of the tongue. In the case of ineffectiveness of conservative treatment, such children are shown plastic surgery of the frenulum of the tongue with mandatory myogymnastics in the postoperative period. Frenulotomy is performed using the same technique as in newborns, but with suturing the wound in the longitudinal direction. The choice of pain relief method depends on the age of the child, his somatic health and the level of psycho-emotional lability. It is generally indicated for children under 5 years of age with a vulnerable psyche, chronic somatic diseases, intolerance to local anesthetics, etc. In other cases, infiltration anesthesia is used. Children with a short, thin frenulum of the tongue at the age of 3-6 years are initially shown myogymnastics, which helps to stretch the frenulum and increase the mobility of the tongue. In the case of ineffectiveness

of conservative treatment, such children are shown plastic surgery of the frenulum of the tongue with mandatory myogymnastics in the postoperative period. Frenulotomy is performed using the same technique as in newborns, but with suturing the wound in the longitudinal direction. The choice of pain relief method depends on the age of the child, his somatic health and the level of psycho-emotional lability. It is generally indicated for children under 5 years of age with a vulnerable psyche, chronic somatic diseases, intolerance to local anesthetics, etc. In other cases, infiltration anesthesia is used. Children with a short, thin frenulum of the tongue at the age of 3-6 years are initially shown myogymnastics, which helps to stretch the frenulum and increase the mobility of the tongue. In the case of ineffectiveness of conservative treatment, such children are shown plastic surgery of the frenulum of the tongue with mandatory myogymnastics in the postoperative period. Frenulotomy is performed using the same technique as in newborns, but with suturing the wound in the longitudinal direction. The choice of pain relief method depends on the age of the child, his somatic health and the level of psycho-emotional lability. It is generally indicated for children under 5 years of age with a vulnerable psyche, chronic somatic diseases, intolerance to local anesthetics, etc. In other cases, infiltration anesthesia is used. which helps to stretch the frenulum and increase the mobility of the tongue. In the case of ineffectiveness of conservative treatment, such children are shown plastic surgery of the frenulum of the tongue with mandatory myogymnastics in the postoperative period. Frenulotomy is performed using the same technique as in newborns, but with suturing the wound in the longitudinal direction. The choice of pain relief method depends on the age of the child, his somatic health and the level of psycho-emotional lability. It is generally indicated for children under 5 years of age with a vulnerable psyche, chronic somatic diseases, intolerance to local anesthetics, etc. In other cases, infiltration anesthesia is used. which helps to stretch the frenulum and increase the mobility of the tongue. In the case of ineffectiveness of conservative treatment, such children are shown plastic surgery of the frenulum of the tongue with mandatory myogymnastics in the postoperative period. Frenulotomy is performed using the same technique as in newborns, but with suturing the wound in the longitudinal direction. The choice of pain relief method depends on the age of the child, his somatic health and the level of psycho-emotional lability. It is generally indicated for children under 5 years of age with a vulnerable psyche, chronic somatic diseases, intolerance to local anesthetics, etc. In other cases, infiltration anesthesia is used. lingual myogymnastics in the postoperative period. Frenulotomy is performed using the same technique as in newborns, but with suturing the wound in the longitudinal direction. The choice of pain relief method depends on the age of the child, his somatic health and the level of psycho-emotional lability. It is generally indicated for children under 5 years of age with a vulnerable psyche, chronic somatic diseases, intolerance to local anesthetics, etc. In other cases, infiltration anesthesia is used. lingual myogymnastics in the postoperative period. Frenulotomy is performed using the same technique as in newborns, but with suturing the wound in the longitudinal direction. The choice of pain relief method depends on the age of the child, his somatic health and the level of psycho-emotional lability. It is generally indicated for children under 5 years of age with a vulnerable psyche, chronic somatic diseases, intolerance to local anesthetics, etc. In other cases, infiltration anesthesia is used. chronic somatic diseases, intolerance of local anesthetics, etc. In other cases, infiltration anesthesia is used. chronic somatic diseases, intolerance of local anesthetics, etc. In other cases, infiltration anesthesia is used.

Defects in the development of frenulums of the lips are manifested by a decrease in their length, an atypical place of attachment of its stem, and an increase in the number of frenulums. There are two forms of defects of the frenum of the lips depending on the attachment of its stem: to the interdental papilla without interweaving of the fibers of the frenulum into the interalveolar seam (impermeable

form) and attachment of the frenulum of the lip, in which case its fibers are woven into the median seam (permeable form). In the case of a short frenulum of the upper or lower lip, there are mostly no complaints from children and their parents. A short frenulum of the lip is more often detected by an orthodontist, to whom they turn with complaints about the presence of a gap between the central incisors (more often on the upper jaw). The rest of the children turn to a dental therapist with complaints of bleeding from the gums during tooth brushing, lagging of the dento-gingival papillae from the necks of the incisors, looseness and soreness of the gums, bad breath, sometimes - on the mobility of the teeth. Clinic. The frenum of the upper or lower lip is short, which causes the central part of the red border to retract. The leg of the bridle is attached to the papilla between the central incisors, which may be accompanied by a diastema. If the fibers of the bridle are woven into the median seam, there is always a diastema. In such cases, the absence of bone tissue is determined in the form of a narrow "dark" band between the roots of the central incisors. In the case of the development of local periodontitis phenomena (mostly on the lower jaw) in the area of the frontal teeth, the gums are swollen, hyperemic, the papillae lag behind the necks of the incisors. If treatment is not carried out in such a condition, over time, dento-gingival pockets are formed, and in the future, pathological mobility of the teeth and anomalies in their position may appear. In children 10-12 years old, it is possible to detect a distal bite or anomalies in the position of the incisors - their inclination towards the tongue and rotation along the axis. Treatment. Surgical intervention in the presence of a short frenulum of the upper lip is more often performed during the period of variable bite after the eruption of the central and lateral incisors. However, in case of the development of local periodontitis phenomena or in case of permanent trauma to the frenulum while eating, the intervention is performed after 2 years, when all temporary teeth have erupted. There are the following methods of treating malformations of the frenulum of the lips: - transverse crossing - frenulotomy - performed in the case of a short frenulum without deviation of its "leg" attachment; - plasty of the bridle with triangular flaps according to O.O. Limberg - is used very rarely, because after Z-plasty, the frenum practically disappears, which should not be the case; - moving the frenum with a V-shaped Dieffenbach dissection is the most common method of intervention, the essence of which is to move the "leg" of the frenum, which is fixed to the cellular process or cellular part. If the "leg" of the frenulum of the upper or lower lip is attached to the interdental papilla and its fibers are woven into the middle seam with the formation of a diastema, the surgical intervention is performed as follows: the frenulum is moved up with a V-shaped incision; with a scalpel, the tissues of the incisor papilla are cut to the bone, moving to the palate and trying not to injure the area where the vascular-nerve bundle exits (this can lead to bleeding, which is best stopped by electrocoagulation); with a curettage spoon or with the help of a drill with a spherical bur, the remaining connective tissue fibers are carefully selected from the median seam. The V-shaped flap is fixed in a new position. After such an intervention, the orthodontist eliminates the diastema with the help of a mouth guard, plate or bracket system. In the case of a short frenulum of the lower lip, the same operations are performed as described above for the treatment of a short frenulum of the upper lip.

The consequences of short frenulums of the lips are the appearance of diastemas (more often on the upper jaw), limited local periodontitis (more often on the lower jaw), deformation of the tooth rows and the frontal area of the cellular process and the cellular part.

Small oral cavity in children is more often acquired and formed after surgical interventions for congenital non-unions of the upper lip (usually bilateral), burns, tumor processes and traumatic injuries of the soft tissues of the upper lip as a result of cicatricial changes. In isolated cases, it can be congenital (in so-called syndromic children) and caused by the presence of buccal cords, short

frenulums of the lips or several signs at the same time. Complaints Children with a small hairline, as well as with short frenulums of the lips and tongue, turn to an orthodontist or a dental therapist with complaints about the presence of maxillofacial deformity, exposure of the necks and roots of the teeth in the place of attachment of ligaments and ligaments, inflammation of the mucous membrane in the place of the largest tightening them, bad breath, mobility of teeth, etc. In the case of cicatricial reduction in the size of the oral cavity (most often the upper one), complaints will be about the immobility of the lip, impaired pronunciation of sounds, later - about the lag in the growth of the upper jaw and the formation of an incorrect bite. Clinic. In the case of congenital shallow oral cavity, additional cords and labial ligaments are found, which pull the gingival margin away from the necks of the roots of the teeth, gingival pathological pockets, varying degrees of tooth mobility and gingival inflammation. In their area, if a small scar was formed after surgical interventions on the soft tissues of the lips, cheeks or after burn or post-traumatic scarring, the upper lip is welded to the jaw, which limits its mobility. Over time, scarred tissues press on the cellular process, causing deformation of the jaws in the sagittal and transverse directions. For the treatment of shallow upper hairline, which is due to the presence of strings and ligaments of the mucous membrane, use Z-plasty according to O.O. Limberg, G.V. Kruchynskyi and A.S. Artyushkevich, V-like plastic surgery according to Diefenbach, etc. The essence of the techniques is to disperse these threads and connections. Significant difficulties arise in the case of cicatricial reduction of the depth of the hairline after surgical interventions, injuries and burns. Under such conditions, the upper lip seems to be soldered to the cellular process and soft tissues to restore the depth of the hairline. For a successful intervention, regardless of the method chosen by the surgeon to deepen the incision, it is essential to isolate two wound surfaces (on the upper lip and cellular process) or one (more often on the upper lip). This is a guarantee that in the future relapse, i.e. the growth of the tissues of the lip and cellular process, will not occur. To close the tissue defect on the upper lip, Z-plasty is used according to O.O. Limberg, transfer of rectangular flaps, pedicle flaps from adjacent areas of the lip, etc. The wound is closed tightly. The wound surface on the cellular process can be closed with a flap of the mucous membrane on the leg from the upper lip or cheek. This method is used in the case of a (small) tissue defect limited in area. It is possible to isolate a large-sized wound surface with the help of free transplantation of a flap of the mucous membrane (more often from the cheek). The fixation of the flap, which is carried out with a mouthpiece with a pellet under the upper lip, is of particular importance in the postoperative period. Previously, a thin layer of sterile foam (through a napkin) is applied to the transplanted area, which will ensure uniform pressure and grafting of the flap.

1. Theoretical questions

Questions for self-control

1. What is the difference between "nonunion" and "cleft"?
2. Classification of nonunions of the upper lip.
3. Causes of birth defects of SCD.
4. Anatomical and functional disorders in a child with non-union of the upper lip.
5. Tasks of cheiloplasty.
6. Early and medium terms of cheiloplasty.
7. What are the types of non-unions of the palate and what is their essence?
8. Anatomical and functional disorders in a child with non-union of the palate?
9. Average terms of surgical treatment of nonunions of the hard and soft palate?
10. Basic principles of the most common methods of uranostaphyloplasty.

11. The main clinical manifestations of congenital defects of the frenulum of the tongue and upper lip.
12. Methods of treatment of congenital defects of tongue frenulum and upper lip.
13. Determination of the depth of the cutting.
14. Embryogenesis of SHLD?
15. Define the terms "hereditary disease", "hereditary predisposition", "teratogenic developmental defect".
16. Define the medical terms "congenital defect", "anomaly", "deformation".

2 Practical tasks
1. Prepare an essay on the topic: "Defects of the face, neck and oral cavity"
2. Make a graph of the logical structure of "Disorders of development of the face, neck and organs of the oral cavity".

3. Test tasks for self-control:

4. Individual tasks
1. Make an outline on this topic

5. List of recommended literature:
Main:
1 Pathomorphology: national. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.
2 Fundamentals of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.
3 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

Additional:
1. Pathological anatomy: a textbook. Trans. from Russian 4th edition / Strukov A.I., Serov V.V. - Kharkiv.: Fakt, 2004. - 864 p.
2. Skrypnikov P.M., Skrypnikova T.P., Shynkevich V.I., Tovma V.V. Individual and age-related clinical and morphological features of the mucous membrane of the oral cavity: study. manual - Poltava: LLC "ASMI", 2016. - 102 p.

Electronic information resources

1 http://moz.gov.ua – Ministry of Health of Ukraine
2 www.ama-assn.org – 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 - Pathology Laboratory

Topic #12: "Diseases of the female and male reproductive system. Pathology of pregnancy, postpartum period and placenta. Diseases of the mammary gland"
Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:
The student should know:
1. principles of classification of diseases of genital organs and mammary glands,
2. the essence and mechanisms of their development,
3. morphological signs of the specified diseases, their consequences and prognosis;
4. pathological processes observed in diseases of the genital organs and mammary glands;
5. diseases of the cervix: classification, clinical and morphological characteristics;
6. diseases of the uterus and endometrium: morphological characteristics;
7. mammary gland diseases: classification, clinical and morphological characteristics;
8. diseases of the male genital organs: classification, clinical and morphological characteristics; complications, consequences.
:The student should be able to:
1. interpret the morphological manifestations of diseases of the male genital organs;
2. interpret the morphological manifestations of diseases of the female genital organs;
3. interpret the morphological manifestations of diseases of the mammary gland;
4. to conduct a macro- and microscopic examination of various diseases of the male and female genital organs and mammary gland;
5. systematize the main signs specific for each individual type of disease.

Topic content:

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 prostatopathy) is observed in 95% of men over the age of 70. At the same time, the gland is enlarged, soft and 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 are distinguished.
Glandular hyperplasia is characterized by an increase in the number of glandular elements, while the number and size of the lobes 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; formation of retention cysts is possible.

Adenoma of the 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, compensation becomes insufficient, an excess of urine accumulates in the bladder, 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 is a fairly common 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 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. When examined histologically, the mucous membrane corresponds to a protracted proliferation phase, 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 of atypia appear - 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 condition of the uterus.

Endocervicosis - an 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 glandular structures that develop from the cambial elements of the prismatic epithelium of the cervical canal (it can differentiate into both glandular and flat 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.

Cervical adenomatosis refers to a process when glandular formations lined by one layer of cuboidal epithelium grow under the covering epithelium of its vaginal part.

Cervical polyps arise 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 a precancerous process.

Benign dysplasia of the mammary gland (synonyms: mastopathy, fibrocystic disease) is characterized by a violation of the differentiation of the epithelium, its atypia, a change in histostructure, but without penetration through the basement 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.

The non-proliferative form is characterized by the 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 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.

The proliferative form is characterized by the growth of epithelium and myoepithelium or joint growth 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 in the size of the particles in connection with the proliferation of the epithelium of the glands. The growth of ductal or lobular epithelium leads to the formation of structures of solid, adenomatous and cribriform type, and at the same time connective tissue grows. 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 breast dysplasia, cancer often develops,

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, Escherichia coli and others. The endometrium is thickened, covered with a gray-yellow purulent film. When the inflammatory process spreads to the vessels of the myometrium, purulent metritis and thrombophlebitis occur. 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.

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 is an 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; upon penetration of spermatozoa into the stroma of the testicle, peculiar spermatozoal granulomas are formed. The consequence of chronic orchitis is unfavorable (infertility).

Prostatitis is 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, according to the course, should be 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 parenchymal 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.), in which lymphohistiocytic infiltration of the stroma of the gland, growth of granulation and scar tissue prevail; 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 breast cancer. 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 is preceded by precancerous conditions, such as endocervicosis and severe dysplasia of the epithelium of the vaginal part of the cervix. 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 into the wall of the neck, adjacent tissue and grows into the wall of the 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, endometrioid adenocarcinoma of the cervix is also distinguished.

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. It can develop both from normal components of the ovary (covering mesothelium, ovum and its derivatives, granulosa cells), rudimentary formations of it (duct of the primary kidney or Wolff's duct), as well as embryonic remnants. However, the vast majority of malignant ovarian tumors are the result of malignancy of benign epithelial serous or mucinous tumors. Ovarian cancer has the appearance of a lumpy node of various sizes, that is, it is a malignant serous and pseudomucinous tumor (see Tumors of exocrine glands and epithelial coverings).
Tumor metastases are lymphatic and hematogenous, occur in lymph nodes, peritoneum, and 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 up to several centimeters in diameter; 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 a mushroom-shaped nodule with ulceration on its surface - a cancerous ulcer.
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 cancer ranks second among oncological diseases in men and is 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 strands of connective tissue intertwining with each other, between them is cancerous tissue of 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, and seminal vesicles. Cancer metastases are observed both in pelvic, iliac and inguinal lymph nodes, and 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.

1. Theoretical questions
Questions for self-control
1. determination of diseases of genital organs and mammary glands;
2. peculiarities of the classification of the specified diseases;

3. etiology of genital diseases;
4. pathophysiological foundations of the development of the specified diseases;
5. possible consequences of each disease;
6. Complications of genital diseases.

2 Practical tasks
1. Prepare an abstract on the topic: "Morphological features of breast tumors"
2. Make a graph of the logical structure "Classification of diseases of the genital organs".

3. Test tasks for self-control:

4. Individual tasks
1. Make an outline on this topic

5. List of recommended literature:
Main:
1 Pathomorphology: national. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.
2 Fundamentals of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.
3 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

Electronic information resources

1 http://moz.gov.ua – Ministry of Health of Ukraine
2 www.ama-assn.org – 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 - Pathology Laboratory
http://www.webpathology.com/ - Web Pathology

Topic #13: "Pre- and perinatal pathology"

Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:

The student should know:

1. classification of diseases of pregnancy, postpartum period;

2. classification of litter pathology;
3. principles of diagnosis of pathology of pregnancy, postpartum period and litter;
4. characteristic morphological features of any of the forms of the aforementioned pathology.

:The student should be able to:

1. distinguish separate forms of the aforementioned pathology (by macro- and microscopic preparations);
2. to determine the features of the pathomorphology of various types of pathology of pregnancy, the postpartum period and litter.

Topic content:

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.

Gestosis (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.

Among the manifestations of toxicosis of pregnant women, eclampsia is the most clinically significant and dangerous, it 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.

Death occurs from liver or kidney failure, as well as from DIC and hemorrhages in vital organs.

Ectopic pregnancy - the 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, as a rule, is observed in one tube. 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 is 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.

A placental polyp is formed in the mucous membrane of the uterus at the site 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.

Obstetric infection of the uterus is 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. Placenta growth is manifested by the 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 ischemia of the placenta 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 placenta is observed in hemolytic disease, infectious processes, diabetes and nephropathy of the mother. 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 space occurs 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.

A heart attack is a 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 is 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.

Підгостра плацентарна недостатність розвивається протягом декількох днів, викликаючи перервисті порушення функції плаценти. По характеру поразки ця форма близька до попередньої, але зони крововиливів невеликі, для них характерні тромби в міжворсинчастих

просторах різної давності. Порушення матково-плацентарного кровообігу виявляється за наявності незрілості ворсин, але відшарування плаценти не відбувається. Розвивається внутрішньоутробна гіпоксія і гіпотрофія плоду.

Chronic respiratory placental insufficiency is characterized by disturbances in the diffusion of gases 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 is long-term (months) disorders of placental function with 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.

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 found, mainly, 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.

First, 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.

Secondly, the accompanying signs of the delayed development of the villous tree can be used to judge the early action of an infectious agent, for example, with 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-fourthly, the use of appropriate 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.

Mycoplasma infection is characterized by 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, cells with large, hyperchromic nuclei appear in the placenta tissue in the chorionic plate, in the epithelium of the villi, in the basal plate, and in the septal cells. 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.

A purulent bacterial infection is characterized by 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 is 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.

Entanglement or true nodes of the umbilical cord 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.

Oligohydramnios - 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 (strings of Simonart) are dense connective tissue hyalinized strings 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.

1. Theoretical questions

Questions for self-control

1. Give the classification of diseases of pregnancy and the postpartum period.
2. Etiology, pathogenesis and pathology of eclampsia.
3. Ectopic pregnancy, types, course and complications.
4. Morphology of spontaneous abortion.
5. Trophoblastic disease: morphology of invasive cystic drift.
6. Trophoblastic disease: morphology of choroid carcinoma.

7. Pathology of the litter: age-related changes and disorders of placenta implantation processes.
8. Morphology of placental blood circulation disorders.
9. Classification of placental insufficiency.
10. The morphology of litter inflammation.
11. Tumors of the placenta.
12. Pathology of the umbilical cord and membranes.

2 Practical tasks

1. Study and sketch "purulent endometritis", staining with hematoxylin and eosin.

2. To study the macropreparation "bladder drift", describe it and indicate the possible etiology.

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

26 Pathomorphology: nats. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.

27 The basics of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.

28 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

Electronic information resources

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

66 www.ama-assn.org– American Medical Association /American Medical Association

67 www.who.int- World Health Organization

68 www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine

69 http://bma.org.uk– British Medical Association

70 www.gmc-uk.org- General Medical Council (GMC)

71 www.bundesaerztekammer.de– German Medical Association

72 http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory

http://www.webpathology.com/- Web Pathology

Topic #14:"Pathology of changes in diseases related to nutrition. Radiation sickness, drug sickness. Occupational diseases"

Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:

The student should know:

1. principles of classification of occupational diseases;
2. principles of diagnosis of occupational diseases;
3. peculiarities of the development of occupational diseases depending on the cause;
4. characteristic morphological signs of occupational diseases.

:The student should be able to:

1. recognize the characteristic morphology of a number of occupational diseases during microscopic examination;
2. evaluate the research results and describe them in the study album;
3. make drawings after studying micropreparations in accordance with the instructions in the study album;
4. predict possible complications.

Topic content:

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 lack of vitamins, the following most often develop: rickets, scurvy, xerophthalmia, pellagra, deficiency of vitamin B12 and folic acid.

RICKETS

Rickets is a consequence of hypo- or vitamin D deficiency.

Classification. There are several forms of rickets: 1) the classic 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 of rickets in childhood and rickets in adults deserve the most attention.

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 deficient ultraviolet irradiation, 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)2Oz 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, the 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 metaphyseal sections of the long tubular bones, that is, in the places with the most intense 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. The size of the fountains increases sharply, they close late. Thickenings appear at the junctions of the cartilage and bone parts of the ribs (especially visible on the inner surface of the VI, VII and VIII ribs), which are called "rachitic rosaries". 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 rickets in children, disorders of endosteal ossification prevail, not enchondral. The bones, especially of the lower limbs and pelvis, are 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.

Complications in children with rickets are pneumonia, feeding and digestive disorders, as well as purulent infections.

SCURVY

Scurvy (synonyms: scurvy, Barlow's disease) - vitamin C. 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 state 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 deficiency of vitamins C and R. 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 the inhibition of proliferative processes in the areas of the most intensive bone 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 leading in the picture of the disease and are expressed in suppression of bone 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.

Complications are mainly associated 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 is 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 into keratinized, multi-layered flat occurs. 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.

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 with vitamin A is significantly delayed.

PELLAGRA

Pellagra is a chronic disease that occurs when the body lacks nicotinic acid (vitamin PP) and other B vitamins.

Etiology and pathogenesis. Pellagra develops when the body lacks not only 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 is found in both the small and large intestines, cystic expansion of glands, ulceration of follicles with successive epithelization of ulcers. Atrophic changes 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 is the smallest particles of a solid substance, formed during an industrial process, which, falling into the air, remain 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. To the organic - plant (flour, wood, cotton, tobacco dust, etc.) and animal (hair, etc.). 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. Industrial dust particles are divided into visible (more than 10 μm), microscopic (from 0.25 to 10 μm) and ultramicroscopic (less than 0.25 μm), which is detected using an electron microscope. 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 dust of animal and vegetable origin causes chronic rhinitis, laryngitis, tracheitis, bronchitis, pneumonia. During the dissolution of dust particles, chemical compounds are formed that have an irritating, toxic and histo-pathogenic effect. They have the ability to cause the development of connective tissue in the lungs, that is, pneumosclerosis. 25 to 10 μm) and ultramicroscopic (less than 0.25 μm), which is detected using an electron microscope. 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 dust of animal and vegetable origin causes chronic rhinitis, laryngitis, tracheitis, bronchitis, pneumonia. During the dissolution of dust particles, chemical compounds are formed that have an irritating, toxic and histo-pathogenic effect. They have the ability to cause the development of connective tissue in the lungs, that is, pneumosclerosis. 25 to 10 μm) and ultramicroscopic (less than 0.25 μm), which is detected using an electron microscope. 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 dust of animal and vegetable origin causes chronic rhinitis, laryngitis, tracheitis, bronchitis, pneumonia. During the dissolution of dust particles, chemical compounds are formed that have an irritating, toxic and histo-pathogenic effect. They have the ability to cause the development of connective tissue in the lungs, that is, pneumosclerosis. Particles less than 5 μm in size that penetrate deep into the lung parenchyma

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When dust of various composition enters the lungs, lung tissue can react differently.

The reaction of lung tissue can be:

• inert, for example, with common pneumoconiosis-anthracosis of coal miners;

• fibrosing, for example, with massive progressive fibrosis, asbestosis and 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 local accumulations of it, which are invisible until massive pulmonary fibrosis is formed. The accumulation of coal in the lungs, referred to as "pulmonary anthracosis", is typical for residents of industrial cities. It can be observed in almost the majority of cases. Importantly:

• 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 caused by 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 (from Latin - silicon), or chalicosis (from Greek - limestone) 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. Flint dust is found in most industrial productions, in particular in gold, tin and copper mines, in the cutting and grinding of stones, in the production of glass, in metal melting, in the production of pottery and porcelain. In all these productions, the size of the particles is important. Sand usually contains 60% silicon oxide. But its particles are very large, to reach the periphery of the lungs. Only small particles that enter 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 an important 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, "acute" silicosis, may occur in 1-2 years. In some cases, the disease may appear several years after exposure to industrial dust (late silicosis). The risk group for this disease includes the workers of the professions mentioned above. especially its particles with a size of 2-3 nm are 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

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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, we are talking about the leading role in the pathogenesis of silicotic fibrosis of the death of coniophages 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, autoantigens appear, which leads to autoimmunization. The immune complex that occurs when an antigen interacts with an antibody has a pathogenic effect on the connective tissue of the lungs. But no specific antibodies were detected. According to the immunological theory, during the influence of silicon dioxide on tissues and cells, during their disintegration, autoantigens appear, which leads to autoimmunization. The immune complex that occurs when an antigen interacts with an antibody has a pathogenic effect on the connective tissue of the lungs. But no specific antibodies were detected. According to the immunological theory, during the influence of silicon dioxide on tissues and cells, during their disintegration, autoantigens appear, which leads to autoimmunization. The immune complex that occurs when an antigen interacts with an antibody has a pathogenic effect on the connective tissue of the lungs. 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 silicosis nodes in the lungs are absent or there are very few of them, they are often found in 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 bronchiolitis, 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 siliceous 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 silicosis nodes have irregular shapes, they lack a 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. Silicose 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. Silicotic 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 the lymph nodes (bifurcation, 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

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Clinical manifestations of silicosis depend on the prevalence of the impression and its severity. If it is a big impression, then shortness of breath may appear after several years. It is caused by silicoprotein pneumonia. If the impression area is smaller, then the onset of the disease is asymptomatic and the detection of silicosis can be detected during a systematic X-ray examination. On x-ray images, you can see a picture of the so-called "snowstorm", which indicates silicotuberculosis, in which, in addition to silicotic nodules and tubercular changes, so-called silicotuberculosis foci are found. The right half of the heart is often hypertrophied, up to the

development of a typical pulmonary heart. Patients most often die from progressive pulmonary and heart failure.

ASBESTOSIS

The word "asbestos" comes from the Greek word "indestructible". About 6 million tons of this mineral are extracted annually in the world. There are several types of asbestos: serpentine, or white asbestos (the chrysotile variety is most often used in industry) and amphiboles, or blue asbestos, such as crocidolite and ammonite. All of them are pathogenic and have a fibrosing effect. 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. Asbestos has been used for many centuries, as it is fire resistant as an insulating material, bituminous coating, in industrial structures, audio products, as well as in many other potentially dangerous products. The disease is widespread in Canada, which ranks first in the world in terms of asbestos reserves. About 5 million people come into contact with asbestos every day on the construction site alone. Among them is a group of insulation workers, 38% of whom are affected by asbestos. It should be emphasized that exposure to asbestos can be indirect, for example, in spouses and members of their families, people working with asbestos. It is believed that crocidolite, which has thinner fibers, causes the development of mesothelioma of the pleura and peritoneum, as well as carcinoma of the bronchi and gastrointestinal tract. According to most authors, the carcinogenicity of asbestos depends not only on its type, but on the length of the fibers. Yes, fibers larger than 5 microns do not have carcinogenic properties, while fibers less 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, 90 times. Cancer of the esophagus, stomach, and colon is twice as common in patients with asbestosis. 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. Fibers are found not only in the lungs, but also in the peritoneum and other organs. Fibers damage the walls of bronchioles and alveoli, which is accompanied by small hemorrhages, which is the basis for the formation of hemosiderin inside macrophages. Sets consisting of asbestos fibers are sometimes covered with protein, but most often with glycosaminoglycans, on which iron-containing grains of hemosiderin settle, they are called "asbestos bodies". Under an optical microscope, they are reddish or elongated yellowish structures shaped like rings or strings of 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 differentiate asbestosis 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 disease occurs 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, hammering sleepers, 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 line communications, radio telephones, etc.), television, during physiotherapeutic procedures. They are distinguished: - 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 from 1 to 10 m;

• short waves (HF) or high frequency waves (HF) with a wavelength from 10 to 1000 m or more.

Acute deaths among people exposed to massive exposure to radio frequency electromagnetic waves have not been described.

Chronic exposure to low 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) organ, rupture of the eardrums, bleeding from the ears is 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.

Meteosensitivity is the body'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."

Manifestations of weather sensitivity depend 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. However, people who do not feel the weather still have reactions to it, although sometimes they are not aware of it. They must be taken into account. for example, with transport drivers. With a sudden change in weather conditions, it becomes more difficult for them to concentrate. Hence, the number of accidents may increase. As a result of diseases (influenza, tonsillitis, inflammation of the lungs, diseases of the joints, etc.) or fatigue - the body's resistance and reserves decrease. That is why weather sensitivity is noted in 35-70% of patients with various diseases. Yes, every second patient with diseases of the cardiovascular system feels the weather. Significant atmospheric changes can cause overstrain and disruption of adaptation mechanisms. Then 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 is 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 diametrically opposite weather reactions, favorable and unfavorable, are often noted for the same diseases. Meteosensitivity is more often observed in persons 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 the saturation of blood with oxygen (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 an overload of the nervous system, irritating the receptors of the skin. In recent years, a new direction has been gained in the study of the impact of weather conditions on the body, 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 well-being 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 (+6o) 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, changes in solar activity and the Earth's magnetic field (geomagnetic storms) have been of great importance. their effect on the body is revealed 1-2 days before the weather changes, while other weather 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 a depressing effect on the psyche of people, reduced work capacity,

There are three degrees of weather sensitivity. A mild degree is only a subjective malaise. With severe meteosensitivity (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. Mixed type - 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 manifestations of weather sensitivity. This is an unspecified type of reaction: general weakness,

The nature and magnitude of damage caused by the influence of atmospheric pressure depends on the magnitude (amplitude) of atmospheric pressure deviations and, mainly, on the speed of its change.

Decompression sickness most often occurs in divers (during deep-sea diving), pilots, workers in caissons (caisson disease) as a result of saturation of the blood and tissues of the body 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 body tissues with nitrogen or helium in the high pressure zone continues until the pressure of these gases in the inhaled air equalizes with their pressure in the 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 atm. h (when observing the rules for creating increased pressure) does not have an adverse effect on the body.

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 the blood vessels of the brain are damaged, dizziness, stupor, vomiting, weakness, fainting, sometimes paresis and paralysis are observed. When the blood vessels of the lungs are damaged, there are chest pains, a sharp cough. Depending on the severity of the disease, death can occur either a few

minutes after decompression, or within one day to three weeks.

With the rapid onset of death, strongly pronounced cadaveric emaciation. There is widespread 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. Edema, 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, that is, increasing the pressure followed by its 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 doses sometimes 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 and knock out electrons.

Ionizing and non-ionizing electromagnetic radiation

Frequency (Hz)

Type of radiation

Biological effect

1-50

Electric current

?

106-10y

Radio waves

Thermal effect, cataract

9-10

Microwave radiation

opacification of the lens

10p-1014

Infrared radiation

Cataract

1015

Visible light

Retinal burns (laser radiation)

1015-1016

Ultraviolet

Skin burns, ulcers

1018-1022

X-ray and gamma radiation

Acute and long damage:

MALIGNANT TUMORS

Cosmic 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.

The dose of ionizing radiation is measured in the following units:

- x-ray: a 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 of X-ray or gamma radiation.
- sievert (Zv): radiation dose that produces a biological effect is equal to the effect 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 large doses (>10 Gy), death of proliferating cells at medium doses (from 1 to 2 Gy) to the absence of histopathological effect at doses 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-phosphate chains, 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, ulcers of mucous membranes

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

Brain

Edema, necrosis

Necrosis of white matter, gliotic tumors of the brain

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 damage includes increased expression of proto-oncogenes, induction of cytokines such as tumor necrosis factor, and activation of antioxidant defense enzymes such as 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 during various types of DNA damage: its protein product changes to an activated form as a result of post-translational transformation.

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 and, to a lesser extent, cancer of the gastrointestinal tract and urinary tract.

Clinical characteristics of acute radiation syndrome

Stage

Whole body dose

Symptoms

Forecast

Subclinical

<200 REM

Mild nausea and vomiting Lymphocytes<1500/mm3

100% survival

Hematopoietic

200-600 REM

Nausea and vomiting. Petechiae and hemorrhages. A sharp decrease in neutrophils and thrombocytes within 2 weeks Lymphocytes <1000/mm3

Frequent development of infectious complications, bone marrow transplantation is necessary.

Gastrointestinal

600-100 REM

Nausea, vomiting. diarrhea. Bleeding and infection for 1-3 weeks. Severe neutro- and

thrombocytopenia. Lymphocytes <500/mm3.

Shock and death within 10-14 days

CNS lesions

>1000 REM

Constant vomiting and diarrhea. Fainting, drowsiness, convulsions. Absence of lymphocytes.

Death within 14-36 hours

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.

Clinical manifestations of exposure. Acute irradiation of the whole body. Whole body exposure is potentially lethal; clinical manifestations depend on the dose and are described as acute radiation syndrome or radiation sickness. When studying disasters at nuclear plants and nuclear bombing in Japan, it was established that the human dose of X-rays or gamma rays, under the influence of which 50% of those exposed die within 60 days, is 2.5-4.0 Gy (250-400 Rad). Depending on the received dose, 4 syndromes may develop: subclinical or prodromal, hematopoietic, gastrointestinal syndromes or central nervous system damage syndrome (table). Acute symptoms reflect damage to highly sensitive and rapidly proliferating tissues of the body, such as bone marrow and epithelium of the gastrointestinal tract. If the patient survives,

Radiation therapy. External irradiation is used for radiotherapy of malignant tumors in doses from 40 to 70 Gy (4000-7000 Rad), while protecting the surrounding tissues. Even with local irradiation, especially of the lungs and abdomen, acute radiation sickness can develop. Under the influence of radiation, the tumor tissue can shrink sharply, which leads to the occurrence of pain and/or compression of the surrounding tissues. As a result of radiation therapy, patients may develop infertility, secondary malignant tumors, and late complications.

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 for diagnostic purposes, from the moment of implantation to the 9th week of pregnancy, a large number of various developmental disorders are observed, 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 disruption of the 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 constrictive pericarditis. Less often, as a result of damage to the coronary arteries, myocardial ischemia develops and, as a result, cardiosclerosis.

Lungs Lungs are easily damaged by ionizing radiation. Acute pulmonary insufficiency often develops, and in the later term - radiation pneumonia. They develop both intraalveolar 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. Cigarette smoke, in addition to carcinogenic substances, contains two radionuclides: Pb210 and Po210. Sometimes Rn222 is found in mines. 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, vascular damage, and glomerular hyalinization, which eventually leads to hypertension and kidney atrophy. Acute necrosis of the epithelium can be observed in the bladder, then submucosal fibrosis, contractures, bleeding, and ulceration develop.

As a result of exposure to ionizing radiation, there is a delay in the neuropsychological development of children. The risk of childhood leukemia and nerve tissue tumors also increases.

Postnatal period. When exposed to radiation 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. In flies and mice, it was proved that mutations arising 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. Wound healing is often disrupted, and infectious processes develop in them.

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). 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 sensitive 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). 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 and late effects of ultraviolet radiation

Type of radiation

Wavelength (mm)

Acute effects

Late effects

UV-A

320-400

Erythema 8-48 hours. Loss of Langerhans cells. Darkening of the pigment. Inflammation of the dermis.

Tanning Skin cancer.

UV-V

290-320

Erythema 3-24 hours. Apoptosis of keratinocytes. 3appearance of Langerhans cells.

Tanning Solar elastosis. Premature aging. Keratosis. Skin cancer.

UV-C

200-290

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-A, there 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 keratin-neutralizing "sunburn cells" appear in the epidermis.

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 and the synthesis 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 in the p53 gene are often found. 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 a strong pole 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.

1. Theoretical questions

Questions for self-control

1. Define the concept of occupational diseases.
2. Classification of occupational diseases.
3. Clinical and morphological manifestations of diseases associated with insufficient and excessive nutrition.
4. Clinical and morphological manifestations of pneumoconiosis.
5. Clinical and morphological manifestations of atmospheric pressure disorders.
6. Morphological manifestations of diseases under the influence of industrial noise.
7. Morphological manifestations of diseases under the influence of electromagnetic waves and radio frequencies.
8. Vibration disease and its pathomorphology.
9. Morphological manifestations in the human body under the action of electricity.
10. Morphology of injuries from temperature effects, burns, heat stroke, hypothermia.
11. Pathomorphology of acute and chronic radiation sickness.
12. Iatrogenic pathology, medicinal disease, morphological characteristics.

2 Practical tasks

1. Prepare an essay on the topic: "Radiation disease, medical disease. Occupational diseases"
2. Make a graph of the logical structure "Radiation disease, medical disease. Occupational diseases".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

29 Pathomorphology: nats. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.
30 The basics of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.
31 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

Electronic information resources

73 http://moz.gov.ua- Ministry of Health of Ukraine
74 www.ama-assn.org– American Medical Association /American Medical Association
75 www.who.int- World Health Organization
76 www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
77 http://bma.org.uk– British Medical Association
78 www.gmc-uk.org- General Medical Council (GMC)
79 www.bundesaerztekammer.de– German Medical Association
80 http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
http://www.webpathology.com/- Web Pathology

Topic #15: "Diseases caused by protozoa and helminths"

Purpose: as a result of self-study, knowledge of the topic is necessary for the study of diseases at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:

The student should know:

1. Classification and features of the structure of mycoses.
2. Etiology, pathogenesis, pathomorphology of the most common mycoses.
3. Prognosis, complications of these diseases.
4. Clinical and morphological forms of mycoses, pathomorphosis and complications accompanying certain forms of mycoses in both children and adults.

:The student should be able to:

1. To interpret the modern classification of mycoses.
2. To characterize the etiology of mycoses.
3. To characterize the features of the pathomorphosis of mycoses.
4. Classify diseases caused by helminths.
5. To characterize the etiology, pathogenesis and morphological essence of diseases caused by helminths.
6. To characterize the etiology, pathogenesis and morphological essence of diseases caused by simple
7. To characterize the etiology, pathogenesis and morphological essence of diseases caused by helminths.
8. Carry out clinical and laboratory diagnostics with microscopy of certain types of mycoses, helminths, protozoa.

Topic content:

1. Classification of mycoses:

1) Visceral:

- primary
- secondary (opportunistic)
- epidermomycoses: epidermophytes (branoid lichen)

2) Dermatomycosis:

- superficial dermatomycoses: the main changes are emphasized in the epidermis, but the dermis is also affected
- deep dermatomycoses: together with the dermis, the epidermis is affected.

Visceral mycoses O.K. Khmelnytskyi in 1962 divided into:

I. Primary acute infectious visceral mycoses (obligate pathogenic):

- cryptococcosis, blastomycosis, coccidomycosis, histoplasmosis, rhinosporidosis

II. Opportunistic mycoses (facultatively pathogenic):

- candidiasis and mold mycoses (aspergillosis, mucorosis, penicillosis)

Actinomycosis is a chronic disease, the causative agent is the anaerobic radioactive fungus Actinomyces Israeli saprophytes in tonsil crypts and carious teeth. Endogenous infection usually occurs with injuries and microtraumas of the organs of the oral cavity. Drusen causes positive neutrophil taxis, i.e. a focus of purulent inflammation is formed, then macrophages, plasma cells, undifferentiated connective tissue cells proliferate perifocally, xanthoma cells appear, autogenous vessels form, granulomas merge. Areas of purulent inflammation, surrounded by granulation and mature connective tissue, have the appearance of bee honeycombs on the section, and grains of actinomycete drusen are visible in the pus. Actinomycotic infiltrate spreads through cellular and connective tissue layers of organs, forming fistulas. There are two clinical and morphological forms:

I. Destructive - in which tissue destruction with the formation of large abscesses prevails;

P. Destructive-proliferative with growth of connective tissue.

According to localization, the following are distinguished:

- cervicofacial actinomycosis (most often);
- abdominal
- actinomycosis of the lungs and chest cavity;
- bone-articular, muscular, skin;
- actinomycosis of the nervous system.

Candidiasis (candidomycosis)

A yeast-like fungus of the genus Candida saprophytes on the surface of the skin and mucous membranes, parasitises when the resistance of the macroorganism is weakened. Primary candidiasis

(without provoking factors) can develop in young children. Secondary candidiasis - autoinfection (after the provoking effect of any factors: antibiotics, corticosteroid therapy, viral infection, etc.). It is likely that the elimination of endogenous microflora during antibiotic therapy promotes adhesion of fungi to the epithelium without keratinization of the oral cavity, esophagus, and vagina. The fungus is tropene to the glycogen-rich multilayered squamous epithelium of the mucous membranes. When fungi penetrate the underlying tissue, oval or rounded yeast-like cells can transform into pseudomycelial (filamentous) forms, which carry out invasive growth. Defensive reactions, which develop in response to the introduction of candida in the tissue, are controlled by the immune system and are provided by neutrophils. Neutrophils are capable of active phagocytosis of fungi, characteristic stringing of neutrophils on a thread of pseudomycelium and their mass death.

I. Local candidiasis: on the skin or mucous membrane, brown overlays of pseudomycelium threads, desquamated epithelium and neutrophils, there may be foci of mucosal necrosis with demarcation purulent inflammation. When pseudomycelium germinates in the vessel lumen, metastases occur in vascular organs.

Two forms of tissue reactions in visceral candidiasis:

- exudative-necrotic - around the fungi there is a cellular infiltrate of neutrophils strung on a thread of pseudomycelium, which disintegrate, and when weakened - necrotic ones prevail.
- tuberculoid granulomatous reaction - with a prolonged course, a productive reaction prevails. The center of the granuloma usually contains only fragments of the fungus and cellular detritus, surrounded by macrophages, epithelioid and giant polynuclear cells, and on the periphery - lymphocytes. As the granuloma matures, fibroblasts appear in it.

Candida can parasitize in the cytoplasm of macrophages and giant cells (endocytobiosis), so it is impossible to speak with certainty about their complete phagocytosis by macrophages.

Incomplete phagocytosis can contribute to the dissemination of the pathogen and the development of hematogenous forms of candidiasis.

Gastrointestinal candidiasis. The favorite localization of candidiasis, along with the skin, is the upper part of the digestive tract, lesions of the mucous membranes of the stomach and intestines are somewhat less common. Penetration of fungal threads deep into the wall leads to the formation of ulcerative defects, sometimes leading to perforation.

- esophageal candidiasis - characterized by stenosing films
- intestinal candidiasis is characterized by pseudomembranous overlays and the formation of ulcers.
- candidiasis of the stomach can be accompanied by perforation of the stomach. If the fungi are localized at the bottom of the stomach ulcer, they can become the source of a generalized process.

Candidiasis of the urinary tract of the kidneys occurs in an ascending way. Possible candidal urethritis, cystitis, pyelonephritis. Small abscesses, foci of necrosis or granulomas form in the cortical

layer of the kidneys.

Candidiasis of respiratory organs. Characteristic candidal laryngitis, tracheitis, bronchitis, as well as pneumonia, which develop both as a result of aerogenic and hematogenous entry of the pathogen into the lung tissue. Small foci of fibrinous inflammation with necrosis in the center are found in the lungs, cavities are formed after their suppuration. With long-term flow, a productive tissue reaction occurs perifocally, followed by fibrosis.

P. Generalized candidiasis: with hematogenous spread, there are either isolated metastatic foci (in the brain, kidneys, heart, bone marrow, striated muscles), or widespread metastases in many organs. Forms of generalized candidiasis:

- candidal septicemia;
- candidal septic endocarditis;
- chronosepsis;
- chronic granulomatous candidiasis of children.

Cryptococcosis (European blastomycosis, Brusse-Buchke disease, torulosis) - the causative agent is a blastomycete - a yeast-like fungus that has a gelatinous capsule that delays the migration of leukocytes to it, so the early forms of the lesion are characterized by an almost complete absence of an inflammatory reaction.

Later, polymorphic granulomas are formed, consisting of epithelioid and giant polynuclear cells, containing a small amount of the pathogen, including and its degenerative forms.

The primary object of the lesion is the skin and lungs, from where hematogenous metastasis to the central nervous system (into the substance of the brain and meninges) is characteristic, further metastasis leads to visceral lesions.

North American blastomycosis (Gilchrist's blastomycosis) is a chronic mycosis of the skin of granulomatous and ulcerative nature, which is complicated by metastases in internal organs, and disseminated forms may occur. A primary lesion of the lungs (and not the skin) is possible. Blastomyces dermatitides causes abscessation and chronic inflammation with a pronounced giant cell reaction and necrosis in the center, as a result, the area of perifocal inflammation undergoes fibrosis.

Histoplasmosis (Darming's disease, reticuloendothelial cytoplasmosis) deep mycosis, can be in a generalized or localized form. A characteristic lesion of the mononuclear macrophage system: the pathogen is localized in macrophages, giant multinucleated cells, and cells of the macrophage-histiocytic system of the spleen, liver, lymph nodes, and bone marrow.

Three clinical and morphological forms:

1. Benign pulmonary histoplasmosis, both primary, caused by inhalation of fungal spores, and secondary, which develops as a result of lympho-hematogenous spread of the fungus from other organs;

2. Primary histoplasmosis of mucous membranes and skin;

3. Disseminated. Three types of tissue changes are possible:

- intracellular (in histiocytes) localization of the pathogen without a tissue reaction;
- hyperplasia of reticuloendothelial cells containing pathogens, which may be accompanied by perifocal necrosis;
- granulomatous inflammation (epithelioid cell granulomas with single giant cells and a significant admixture of leukocytes).

Coccidiosis is an extremely deep mycosis, which is rare and occurs in two stages:

- primary - the lungs, skin and gastrointestinal tract are affected
- secondary - has a chronic, malignant, progressive course with generalization of the process, which is accompanied by the defeat of lymph nodes, internal organs, the central nervous system and bones.

Tissue forms of Coccidioides immitus are spherical in shape and contain endospores. Morphological changes in the affected tissues are related to the life cycle of the fungus. In response to the rupture of the spherule and the release of endospores, an acute purulent inflammation develops, as they mature, a tubercle-like granuloma with few giant cells forms perifocally in the spherule. After maturation and rupture of the spherule, in response to the release of endospores, macrophage leukocyte infiltration is again observed. In the chronic course of the disease, the growth of granulation tissue with the phenomena of fibrosis is observed, and the causative agent occurs more often in the form of deformed and devastated spherules.

Rhinosporoidosis is characterized by papillomatous growths on the mucous membranes. The tissue reaction to the pathogen is expressed by productive inflammation with the development of granulation tissue: polypous inflammation develops in the mucous membranes of the nasal cavity and nasopharynx, less often localized in the conjunctiva of the eye, mucous membrane of the vulva, urethra, and in the skin.

Aspergillosis.

Aerobic fungi of the genus Aspergillus constantly vegetate in the soil. Can cause damage to the skin and respiratory tract of a person. In the tissues of the host, they appear in the form of uniform septate hyphae (threads), grow radially, fan-like, because they divide dichotomously, but can form mycelium balls. Fungal threads are found among necrotized tissues, and a leukocyte reaction develops around the necrosis zone, and leukocytes are characterized by karyopyknosis and karyorrhexis. With a prolonged course, granulomas are formed - a productive inflammatory reaction develops around the zone of necrosis or abscessation with the accumulation of histiocytes and fibroblasts, giant and a small number of ethelioid cells are present. In the center of the granuloma, only fragments of the cells of the fungus are found.

Visceral forms of aspergillosis are divided into:

- bronchopulmonary (bronchitis, pneumonia);
- cerebral;
- gastrointestinal;
- genitourinary;
- generalized.

The most characteristic pulmonary aspergillosis is distinguished by four types:

1. Non-purulent pulmonary aspergillosis with the formation of gray-brown dense infiltrative foci with a whitish center, where a cluster of fungi is determined among the infiltrate;
2. Purulent pulmonary aspergillosis with focal necrosis and suppuration;
3. Aspergilloma (aspergliosis-lycetoma) of the lung - spherical growth of the fungus, which is formed as a result of its growth on the inner surface of the cavity (bronchoectatic, or abscess cavity), forms shrunken membranes that peel off into this cavity;
4. Tuberculous pulmonary aspergillosis with the formation of granulomas similar to epithelioid tubercles.

Aspergillosis is often associated with chronic lung diseases (bronchitis, bronchiectasis, abscess, lung cancer, fibrous-cavernous tuberculosis), while the wall of the bronchus or cavity is lined with a thin layer of mold.

CNS damage is observed when the process spreads from the air-nasal sinuses and orbit of the eye as a result of hematogenous metastasis.

Generalized aspergillosis develops with hematogenous spread, more often with bronchopulmonary forms and is characterized by single or multiple metastatic foci in internal organs.

Mucormycosis (mucorosis).

Deep chronic mycosis, which, in addition to superficial damage, damages the respiratory organs and is prone to hematogenous generalization of the process, is accompanied by tissue and organ infarctions.

Inflammation is manifested in weak leukocyte and lymphocyte infiltration with a significant content of eosinophils, necrotic processes prevail. Visceral forms of mucorosis quickly end in death, so granulation tissue rarely develops. If this happens, it is characterized by a large number of histiocytes and giant cells. In connection with the growth of fungi in vessels, thromboangiitis and mycotic thrombosis develop, which leads to the development of multiple heart attacks. Moreover, ascending thrombosis is characteristic. The growing ascending thrombosis in the pulmonary arteries reaches the main trunk of the pulmonary artery, the ascending thrombosis of the vessels of the eye socket and additional nasal cavities contributes to the transition of the process to the brain tissue, as a result of which necrosis and hemorrhage are formed.

Echinococcosis is a human and animal disease caused by the larval stage (fin) of Echinococcus from

the class of tapeworms and the family of hookworms. Etiology. Eshinococcus granularis, which causes the hydatid form of echinococcosis, and Eshinococcus milticularis, which causes the alveolar form of echinococcosis, or alveococcosis, are of the greatest importance in human and animal pathology. Hydatid echinococcosis is more common than alveococcosis. Epidemiology and pathogenesis. In the development of hydatid echinococcosis in humans, a major role belongs to the obligate host of sexually mature tapeworm - the dog, in which the parasite lives in the intestines. The alveococcus larva, which differs from the hydatid echinococcus larva, is found in rodents and humans. Humans are likely to become infected when handling rodent skins. Pathological anatomy. With hydatid echinococcosis, bubbles (or one bubble) of one or another size (from a walnut to the head of an adult) appear in the organs. They have a whitish layered chitinous shell and are filled with a transparent colorless liquid. There is no protein in the liquid, but it contains succinic acid. From the inner germinal layer of the bladder membrane, daughter bladders with scolexes arise. These daughter cysts fill the chamber of the mother cyst (unicameral echinococcus). The tissue of the organ in which the single-chamber echinococcus develops undergoes atrophy. At the border with the echinococcus, connective tissue grows, forming a capsule around the bubble. Vessels with thickened walls and foci of cellular infiltration with an admixture of eosinophils are found in the capsule. Giant cells of foreign bodies appear in the areas of the capsule directly adjacent to the chitinous membrane, phagocytizing elements of this shell. More often, the echinococcal cyst is found in the liver, kidneys, lungs, less often - in other organs. With alveococcosis, oncospheres give rise to the development of several bubbles at once, and foci of necrosis appear around them. Cytoplasmic outgrowths are formed in alveococcal bubbles, and bubble growth occurs by budding outward, not inside the mother bubble, as is the case with unicameral echinococcus. As a result, with alveococcosis, more and more bubbles are formed, penetrating the tissue, which leads to its destruction. Therefore, alveococcus is also called multichambered echinococcus. Therefore, the growth of alveococcus has an infiltrating nature and is similar to the growth of a malignant neoplasm. Toxic substances released from the bubbles cause necrosis and a productive reaction in the surrounding tissues. In the granulation tissue, there are many eosinophils and giant cells of foreign bodies, phagocytizing the membranes of dead bubbles. Primary alveococcus is more common in the liver, less often in other organs. In the liver, it occupies a whole portion. It is very dense (the density of a board), on a section it has a porous appearance with layers of dense connective tissue. A decay cavity sometimes forms in the center of the node. Alveococcus is prone to hematogenous and lymphogenic metastasis. Hematogenous metastases of alveococcus at its primary localization in the liver appear in the lungs, then in the organs of the large blood circulation (kidneys, brain, heart, etc.). In this regard, alveococcus clinically behaves as a malignant tumor (cancer). Complications with echinococcosis are more often associated with the growth of a bubble in the liver or metastases of alveococcus. The development of amyloidosis is possible.

Cysticercosis. - chronic helminthiasis, which is caused by the larval stage (fin) of the pig hookworm (Solitary). Etiology, epidemiology, pathogenesis. The disease develops in humans, as well as in some animals (pigs, dogs, cats), which are intermediate hosts of the parasite and its phynozoic stage. Animals become infected by eating human feces containing helminth eggs. A person becomes infected by consuming pork meat, in which the fina (cysticerc) is parasitized. The development of fins in an adult parasite occurs in the human intestine. Humans can develop cysticercosis when parasitised by the pig solitaire in the intestines. This happens when the solitaire eggs fall into the stomach, where their shell dissolves, the embryos penetrate through the stomach wall into the lumen of the vessels, are transferred to various tissues and organs, where they turn into cysticerci. Pathological anatomy. A cysticercus has the appearance of a bubble the size of a pea. A head with a neck extends inward from its wall. An inflammatory reaction develops around the cysticercus. The infiltrate consists of lymphocytes, plasma cells, fibroblasts and eosinophils. Young connective tissue gradually appears around the infiltrate, which matures and forms a capsule around the cysticercus. In the brain tissue, microglial cells participate in the formation of the capsule around the cysticercus. Over time, the cysticerc dies and calcifies. In the brain tissue, microglial cells participate in the formation of the capsule around the cysticercus. Over time, the cysticerc dies and calcifies. In the brain tissue, microglial cells participate in the formation of the capsule around the cysticercus. Over time, the cysticerc dies and calcifies.

Opisthorchosis is a disease of humans and mammals caused by parasites of the trematode species. The first description of the morphology of opisthorchiasis belongs to the Russian pathologist K.N. Vynogradov (1891). Etiology. For humans, the invasion of cat fluke is of greatest importance). Epidemiology and pathogenesis. Humans and carnivores infected with opisthorchosis are a source of infestation for Bithynium molluscs, which ingest the eggs of the parasite that have entered the water with the feces of sick people and animals. In the body of molluscs, the larval stages of the helminth multiply, which ends with the release of cercariae into the water. They penetrate through the skin of fish into their subcutaneous tissue and muscles, transforming here into metacercariae. Infection with opisthorchosis in humans and mammals occurs when raw fish with helminth larvae (metacircaria) are consumed. The bile ducts are the optimal habitat for flukes. Pathological anatomy. The main changes develop in the bile ducts and liver parenchyma. Inflammation develops in the intrahepatic bile ducts, where a large number of parasites are located. The duct walls are infiltrated with lymphoid elements, plasma cells, and eosinophils. The epithelium forms reactive growths with the formation of iron structures in the subepithelial layer. As a result, sclerosis of duct walls and periductal sclerosis develops. Areas of necrosis appear in the liver parenchyma, which are replaced by growing connective tissue. Sclerotic changes in the liver have a focal nature and are associated with the predominant localization of parasites in the biliary tract. Inflammation also occurs in the wall of the

gallbladder. In the pancreas, the expansion of the ducts is emphasized, in which clusters of helminths are found, hyperplasia of the mucous membrane, inflammatory infiltrates in the wall of the ducts and in the stroma of the gland. Complications are associated with the addition of a secondary infection of the biliary tract, which leads to the development of purulent cholangitis and cholangiolitis. With a long course of opisthorchosis, cirrhosis of the liver is possible. As a result of prolonged and perverse proliferation of the epithelium of the bile ducts, cholangiocellular liver cancer sometimes develops.

Schistosomiasis is a chronic helminthiasis with predominant damage to the genitourinary system and intestines. Etiology. The causative agent of this helminthosis in humans is schistosoma from the group of trematodes: Schistosoma haematobium, Schistosoma mansoni and Schistosoma japonicum. Schistosoma haematobium causes schistosomiasis of the genitourinary system, which was first discovered by Bilharts and is therefore called bilharziosis. Epidemiology and pathogenesis. The eggs of the parasite go through their development cycle in the body of freshwater molluscs to the stage of cercariae, which enter the human body through the skin. Cercariae mature very quickly and turn into schistosomula, penetrating into the peripheral veins, where sexually mature individuals are formed. From here, fertilized females go to their favorite place of residence: pelvic veins, mesenteric and hemorrhoidal veins, as well as the wall of the large intestine. Here the females lay their eggs, which causes tissue damage. Part of the eggs is excreted with urine and feces into the external environment, being a source of spread of helminthiasis. Outbreaks of urinary schistosomiasis are mainly found in Africa. Schistosoma mansoni is found in South and Central America, Africa, Schistosoma haematobium - in Japan and Southeast Asian countries. Pathological anatomy. With schistosomiasis, changes are observed first of all in the places of egg deposition, that is, in the bladder, the wall of the large intestine. The most common is urinary schistosomiasis, which affects the bladder. In the early period of the disease, inflammation, hemorrhages, and desquamation of the epithelial cover develop in the surface layers of the mucous membrane of the bladder. Then the changes spread to the deeper layers of the wall. In the submucosal layer around the eggs, schistosomes with leukocyte infiltrates appear, they cover the entire thickness of the mucous membrane, in which ulcers are formed. Over time, the exudative tissue reaction changes to a productive one, granulation tissue with a large number of epithelioid cells forms around the eggs, and schistosome granuloma forms. The process takes a chronic course, the result of which is sclerosis and deformation of the bladder wall. Dead eggs are calcified. The spread of the parasite in the veins of the small pelvis leads to the appearance of lesions in the prostate gland, epididymis. With slow healing of bladder ulcers and a long course of the disease, the development of cancer is possible. With schistosomiasis of the large intestine, similar wall changes develop in it, ending with sclerosis. There are cases of schistosome appendicitis. Hematogenous spread of the process is possible. Parasites enter the liver, lungs, brain, and inflammatory infiltrates of lymphocytes, neutrophils, and epithelioid cells appear at the site of their

entry. Granulation tissue is quickly formed, sclerosis develops.

Trichinosis is a chronic helminthiasis with a predominant lesion of the striated muscles, where the young forms of the parasite are localized. Etiology and pathogenesis. The causative agent of the disease is Trichinella spiralis. Human infection occurs when consuming pig meat infected with trichinella. Pathological anatomy. Trichinosis is characterized by typical changes in the striated muscles. However, they are affected unevenly. Thus, the respiratory muscles, diaphragm, masticatory muscles, pharyngeal muscles, oculomotor muscles, biceps, etc. are most intensely affected. At a macroscopic examination, it can be seen that the muscles are dotted with very small nodules, sometimes yellowish, then whitish, soft or dense up to calcified. Trichinella in various stages of development are detected microscopically in the muscles. It was established that young trichinella bore the sarcolemma, penetrate the muscle fiber, grow, twist into a spiral, and the muscle fiber in particular swells, transverse striation disappears. After the death of muscle fibers, an inflammatory infiltrate consisting of histiocytes, lymphocytes, and single eosinophilic leukocytes appears around them. A picture of trichinellosis myositis develops. Gradually, a connective tissue capsule forms around the spirally bent parasite, the parasite dies and calcifies. From all the above, it turns out that trichinella, which got into the muscle fibers of a person, inevitably dies and its epidemiological role in the spread of the disease ends. Trichinella are carried by the blood flow not only into the muscles, but can get stuck in the capillaries of internal organs, causing inflammatory reactive changes around them. After the death of muscle fibers, an inflammatory infiltrate consisting of histiocytes, lymphocytes, and single eosinophilic leukocytes appears around them. A picture of trichinellosis myositis develops. Gradually, a connective tissue capsule forms around the spirally bent parasite, the parasite dies and calcifies. From all the above, it turns out that trichinella, which got into the muscle fibers of a person, inevitably dies and its epidemiological role in the spread of the disease ends. Trichinella are carried by the blood flow not only into the muscles, but can get stuck in the capillaries of internal organs, causing inflammatory reactive changes around them. After the death of muscle fibers, an inflammatory infiltrate consisting of histiocytes, lymphocytes, and single eosinophilic leukocytes appears around them. A picture of trichinellosis myositis develops. Gradually, a connective tissue capsule forms around the spirally bent parasite, the parasite dies and calcifies. From all the above, it turns out that trichinella, which got into the muscle fibers of a person, inevitably dies and its epidemiological role in the spread of the disease ends. Trichinella are carried by the blood flow not only into the muscles, but can get stuck in the capillaries of internal organs, causing inflammatory reactive changes around them. Gradually, a connective tissue capsule forms around the spirally bent parasite, the parasite dies and calcifies. From all the above, it turns out that trichinella, which got into the muscle fibers of a person, inevitably dies and its epidemiological role in the spread of the disease ends. Trichinella are carried by the blood flow not only into the muscles, but can get

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Ascariasis is a chronic helminthiasis with localization of the parasite in the small intestine.

Etiology and pathogenesis. The causative agent of the disease - roundworms belong to the class of nematodes, of different sexes. The female lays a huge number of eggs in the intestines of the patient, which are released into the external environment with feces. A person becomes infected by ingesting roundworm eggs with water or contaminated food. In the small intestine, the egg shell dissolves and the released larva, penetrating into the blood and lymphatic vessels, enters the lungs and is released into the lumen of the respiratory tract (migratory stage of ascariasis, migroascaridosis). Microascaridosis continues for 7-15 days after infection. Then the larvae are swallowed with saliva, again end up in the intestines, where mature roundworms develop (intestinal stage of ascariasis). The pathological anatomy of ascariasis is mainly related to the presence of ascaris in the small intestine. Their number can reach several hundred. Adult roundworms can penetrate into the excretory ducts of the pancreas and liver, into the lumen of the appendix and cause inflammatory processes in them. If too many roundworms accumulate in the lumen of the small intestine, intestinal obstruction or, in some cases, breakthrough of the wall of the small intestine by roundworms can occur. During the migration of the larvae, hemorrhages are observed in the lungs. Sometimes, during the migration of ascaris larvae, quickly appearing and disappearing foci of inflammation ("volatile infiltrates" in the terminology of clinicians) appear in the lungs, which are accompanied by a small discharge of sputum containing a huge number of eosinophils. There is eosinophilia in the blood, a rash on the skin. Morphologically, in these cases, acinous-lobular foci of inflammation, rich in eosinophils, are found in the lungs. Ascaris larvae are usually not detected in areas of inflammation.

Malaria is an acute or chronic recurrent infectious disease that has different clinical forms depending on the maturation period of the causative agent. Etiology and pathogenesis. The disease is caused by Plasmodium, discovered in erythrocytes by Laveran (1880). The vector of the pathogen is the mosquito (Anopheles). Having entered the blood during a mosquito bite, plasmodia go through a complex development cycle, parasitize in human erythrocytes, reproducing asexually, which is called schizogony. Parasite schizonts accumulate particles of a dark brown pigment - hemelanin - in the cytoplasm. During hemolysis, parasites and hemelanin are released from the erythrocyte. Moreover, the pigment is phagocytosed by cells of the macrophage system, and the schizonts are re-introduced into erythrocytes. In this connection, suprahepatic (hemolytic) anemia, hemelanosis and

hemosiderosis of elements of the reticuloendothelial system develop, culminating in sclerosis. During periods of hemolytic crisis, acute vascular disorders (stasis, diapedesis hemorrhage) appear. In connection with persistent antigenemia in malaria, toxic immune complexes appear in the blood. Their action is associated with damage to the microcirculatory channel (increased permeability, hemorrhages), as well as the development of glomerulonephritis. There are several types of malaria plasmodium, which differ in terms of their maturation. In this regard, three-day, four-day and tropical forms of malaria are distinguished. Pathological anatomy. With the three-day, most frequent form of malaria, anemia develops due to the destruction of erythrocytes, the severity of which is aggravated by the property of plasmodia of three-day malaria to settle in young erythrocytes - reticulocytes (M.V. Voyno-Yasenetsky). Products released during the breakdown of erythrocytes, especially hemelanin, are captured by the cells of the macrophage system, which leads to an increase in the spleen and liver, bone marrow hyperplasia. Organs loaded with pigment acquire a dark gray, and sometimes black, color. The spleen increases especially quickly, first as a result of full blood, and then - hyperplasia of cells that phagocytose pigment. In this connection, its pulp becomes dark, almost black. In the acute stage of malaria, the spleen is loose, full-blooded, in the chronic stage, it becomes dense as a result of the developing sclerosis; its weight reaches 3-5 kg (malarial splenomegaly). The liver is enlarged, full-blooded, with a gray-black surface on the section. Marked hyperplasia of stellate reticuloendotheliocytes with hemomelanin deposition in their cytoplasm. In chronic malaria, there is a thickening of the liver stroma and the growth of connective tissue in it. Bone marrow of flat and tubular bones has a dark gray color, hyperplasia of its cells and deposition of pigment in them is noted. There are areas of bone marrow aplasia. Hemomelanosis of the organs of the reticuloendothelial system is combined with their hemosiderosis. Suprahepatic (hemolytic) jaundice also develops. The pathological anatomy of four-day and three-day malaria is similar. In tropical malaria, the changes are little different from those described in the three-day form, although they have some peculiarities. They are explained by the fact that erythrocytes containing maturing schizonts of tropical malaria accumulate in the terminal areas of the bloodstream, which leads to the development of parasitic stasis. In places of accumulation of maturing schizonts during their division into merozoites, neutrophils and macrophages phagocytize both infected erythrocytes and immature schizonts, as well as decay products and pigment, plasmodia that appear after division (M. V. Voyno-Yasenetsky). Parasitic stasis is associated with life-threatening changes in the brain, which are observed in malarial coma. In such cases, the cortex and other areas of the gray matter of the brain have a dark brown (smoky) color. In the white matter there are numerous point hemorrhages that surround vessels filled with agglutinating erythrocytes with parasites in the cytoplasm or hyaline thrombi. Around such vessels, in addition to hemorrhages, 1.5-2 days after the onset of coma, reactive growth of glial cells occurs, which leads to the formation of peculiar nodules - i.e. with. Durk's

granuloma. A complication of acute malaria can be glomerulonephritis, chronic - exhaustion, amyloidosis. Death is usually observed in tropical malaria complicated by coma.

Amebiasis is a chronic parasitic disease, the basis of which is ulcerative colitis. Etiology and pathogenesis. Amoebiasis is caused by the simplest of the class of rhizopods - Entamoeba hystolytica. The causative agent was discovered by F.A. Leshom (1875) in the bowel movements of patients with amebiasis. The disease occurs mainly. image in countries with a hot climate. Infection occurs through the alimentary route by encysted amoebas, protected from the action of digestive juices by a special membrane that melts in the cecum, where the most pronounced morphological changes are usually observed. The histological properties of the amoeba explain its deep penetration into the intestinal wall and the formation of non-healing ulcers. In some people, the carrier of amoeba in the intestine is noted. What conditions contribute to the transition of the carrier to the disease remains unclear. Pathological anatomy. Getting into the wall of the large intestine, the amoeba and its waste products cause edema and histolysis, mucosal necrosis, and ulcer formation. Areas of necrosis of the mucous membrane slightly protrude above its surface, they are colored dirty gray or green. The section shows that the zone of necrosis penetrates deep into the submucosa and muscle layers. When an ulcer is formed, its edges become undercut and hang over the bottom. As the necrosis progresses, the size of the ulcer increases. Amoebae are found at the border between dead and preserved tissues. It is characteristic that the cellular reaction in the intestinal wall is weakly expressed. However, as the secondary infection joins, a leukocyte reaction occurs, pus appears. Sometimes a phlegmonous and gangrenous form of colitis develops. Deep ulcers heal with a scar. Necrotic-ulcerative changes are most often and sharply expressed in the cecum (chronic ulcerative colitis). However, it is not uncommon for ulcers to form throughout the colon and even in the ileum. Regional lymph nodes are somewhat enlarged, but amoebas are not found in them, amoebas are usually found in the blood vessels of the intestinal wall. Complications of amebiasis are divided into intestinal and extraintestinal. Of the intestinal ones, the most dangerous are: ulcer breakthrough, bleeding, the formation of dense infiltrates around the affected intestine, which often stimulate the tumor. The most dangerous of extraintestinal complications is the development of a liver abscess. amoeba is usually found in the blood vessels of the intestinal wall. Complications of amebiasis are divided into intestinal and extraintestinal. Of the intestinal ones, the most dangerous are: ulcer breakthrough, bleeding, the formation of dense infiltrates around the affected intestine, which often stimulate the tumor. The most dangerous of extraintestinal complications is the development of a liver abscess. amoeba is usually found in the blood vessels of the intestinal wall. Complications of amebiasis are divided into intestinal and extraintestinal. Of the intestinal ones, the most dangerous are: ulcer breakthrough, bleeding, the formation of dense infiltrates around the affected intestine, which often stimulate the tumor. The most dangerous of extraintestinal complications is the development of a liver abscess.

Balantidiasis is characterized by the development of chronic ulcerative colitis. An isolated lesion of the appendix is rarely noted. Etiology. The causative agent of balantidiasis is the infusoria Valantidium coli. Pathological anatomy. Changes in balantidiasis are similar to those in amebiasis, but in balantidiasis, which is much less common than amebiasis, the intestinal damage is not so pronounced. First, there is damage to the surface layers of the mucous membrane with the formation of erosions. In the future, as the balantidia penetrate into the submucosal layer, ulcers develop, which have different sizes and shapes, their edges are undermined, gray-dirty remnants of necrotic masses are visible at the bottom. Balantidia are usually found in the vicinity of foci of necrosis, as well as in the crypts and thickness of the mucous membrane far from ulcers. They can penetrate into the muscle layer, into the lumen of lymphatic and blood vessels. Local cellular reactions in balantidiasis are weakly expressed, eosinophils predominate among the cells of the infiltrate. The most important of the complications of balantidiasis is a breakthrough ulcer with the development of peritonitis. Joining the ulcer process of a secondary infection can lead to septicopyemia.

Toxoplasmosis is a disease caused by simple unicellular microorganisms and occurs both in humans and animals. By origin, toxoplasmosis can be congenital or acquired. Congenital toxoplasmosis affects newborns and young children, acquired - older children and adults. It has been established that toxoplasmosis is widespread among the population of many countries, and a large number of people suffering from abortive and latent forms are emphasized. The etiology of toxoplasmosis has been established. The causative agent of the disease is toxoplasma (from the Greek toxon - arc), which has an arc-shaped shape. For the first time, toxoplasma was discovered in 1908 by Nicole and Manso in Gonda rodents.

Pathogenesis. The ways of toxoplasma penetration into the body have not been definitively established. It is suggested that parasites are introduced through damaged skin or mucous membranes (for example, gastrointestinal tract, respiratory tract), through bites of blood-sucking ectoparasites. A person usually gets infected from dogs or cats if hygiene rules are not followed in dealing with them. Transmission of the disease from person to person is possible only through the placenta of the fetus from the infected mother, which is observed in congenital toxoplasmosis. Getting into the bloodstream, toxoplasmas spread throughout the body, penetrate the cytoplasm of cells of various organs and multiply by simple division. Cells containing toxoplasmas increase in size and are called pseudocysts. When the pseudocysts are destroyed, the parasites enter the bloodstream again and infect new cells. Specific antibodies are formed in the body of a patient with toxoplasmosis, the serological determination of which has diagnostic value. Pathological anatomy. Congenital toxoplasmosis is accompanied by changes in the brain and membranes of the eye. Internal organs are affected by a generalizing infection. Foci of necrosis appear in the brain and eye membranes, which quickly calcify. In the brain, these foci of necrosis have a yellowish color, the size of a millet grain to a pea and are

located in the cortex of the large hemispheres and in the subependymal zone of the lateral ventricles of the brain. Reactive, mostly productive, inflammation occurs around foci in the brain tissue, as well as in adjacent areas of soft meninges and in the ependyma. Against this background, malignant processes develop in the meninges, ventricles of the brain, blood circulation is disturbed and hydrocephalus occurs. It gradually increases, and by the time the fetus is born, it can reach a significant degree and lead to atrophy of the brain substance. Upon microscopic examination, toxoplasmosis pseudocysts and free-lying toxoplasmas are found in fresh foci of necrosis. Gradually, pseudocysts and masses of necrosis become calcified. In the eye, foci of necrosis are formed in the retina and vascular tract, accompanied by productive inflammation with the formation of connective tissue and deformation of eye tissues. Infection of the fetus in the early stages of pregnancy leads to delayed brain development and improper formation of other organs. Thus, toxoplasmosis is one of the etiological factors in the formation of ugliness. Acquired toxoplasmosis is characterized by weak necrotic phenomena and the predominance of productive inflammatory changes in the organs. With the septic form of acquired toxoplasmosis, meningoencephalitis, hyperplasia of the spleen and lymph nodes, and interstitial inflammatory processes in internal organs develop. Acquired toxoplasmosis often proceeds in a hidden form and is recognized only by immunological reactions. In some cases, women learn about toxoplasmosis only after childbirth, when the newborn is diagnosed with this disease. The lethality of toxoplasmosis is high in the congenital form of the disease. Newborns, those born with hydrocephalus and eye damage die early. interstitial inflammatory processes in internal organs. Acquired toxoplasmosis often proceeds in a hidden form and is recognized only by immunological reactions. In some cases, women learn about toxoplasmosis only after childbirth, when the newborn is diagnosed with this disease. The lethality of toxoplasmosis is high in the congenital form of the disease. Newborns, those born with hydrocephalus and eye damage die early. interstitial inflammatory processes in internal organs. Acquired toxoplasmosis often proceeds in a hidden form and is recognized only by immunological reactions. In some cases, women learn about toxoplasmosis only after childbirth, when the newborn is diagnosed with this disease. The lethality of toxoplasmosis is high in the congenital form of the disease. Newborns, those born with hydrocephalus and eye damage die early.

1. Theoretical questions

Questions for self-control

1. Define mycosis?
2. What groups are mycoses divided into?
3. How are visceral mycoses divided?
4. What is dermatomycosis?
5. How are dermatomycoses divided?
6. How O.K. divided visceral mycoses. Khmelnytskyi?
7. What refers to primary visceral mycoses?

8. What mycoses make up the group of opportunistic mycoses?
9. Name the main types of helminths?
10. Give the main characteristics of the simplest ?

2 Practical tasks

1. Prepare an essay on the topic: "Helmintoses"
2. Make a graph of the logical structure of "Helmintoza".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

32 Pathomorphology: nats. nearby / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina and others; under the editorship V.D. Markovsky, V.O. Tumansky. - K.: VSV "Medicine", 2015. - 936 p.

33 The basics of pathology according to Robbins: trans. 10th Eng. ed.: in 2 vols. I / Vinay Kumar, Abdul K. Abbas, John C. Aster.; of science ed. trans. prof.: I. Sorokina, S. Hychka, I. Davydenko. - K.: VSV "Medicine", 2019. - 952 p.

34 Shlopov V.G. Fundamentals of human pathological anatomy - Kyiv, 2002. - 497p

Electronic information resources

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

82 www.ama-assn.org– American Medical Association /American Medical Association

83 www.who.int- World Health Organization

84 www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine

85 http://bma.org.uk– British Medical Association

86 www.gmc-uk.org- General Medical Council (GMC)

87 www.bundesaerztekammer.de– German Medical Association

88 http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory

http://www.webpathology.com/- Web Pathology