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PATHOMORPHOLOGICAL CHANGES OF THE RESPIRATORY TRACT IN COVID-19 BY AUTOPSY DATA IN THE ODESA REGION FOR 2020–2023

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Introduction. The spread of COVID-19 in Ukraine was documented on March 3, 2020. The first fatal case from coronavirus infection in Odesa region was recorded on April 23, 2020. A total of 1294 patients died from complications of the coronavirus disease in Odesa and Odesa region in 2020, 5560 patients died in 2021, and a total of 917 people died from coronavirus infection in 2022.

The aim of the study was to identify and study morphologic changes in the respiratory tract of patients who died from coronavirus infection in Odesa region.

Materials and methods. 50 cases of those who died between 2020 and 2023 with diagnosed COVID-19 (from the total number of those who died from complications of coronavirus disease in Odesa and the region) were randomly selected. Autopsy material was examined using routine morphologic methods.

Results and Discussion. Of the 7,785 people who died from complications of coronavirus disease: 3,922 cases were men (50.4 %), women – 3863(49.6 %). The largest group of the deceased that were people aged ≥ 71 years composed 3373 cases (43.3 % of the total number of those who died from complications of coronavirus disease). Of these, 1,691 were males (50.1 %) and 1,682 were females (49.8 %). Interstitial inflammatory infiltrate, cytopathic viral damage of alveolar epithelium, edema, hyaline membrane formation were found in the lung tissue of 47 deceased. The presence of multinucleated symplasts in the epithelium, desquamated atypical alveolocytes with large polymorphic nuclei, inclusions in the nuclei and cytoplasm, accumulations of erythrocytes, alveolar macrophages, marked edema, fibrin deposits were noted. Blood coagulation disorders occur due to damage to the endothelium of blood vessels, as well as liver cells, with the further development of thrombosis and hemorrhage. Hyperplasia of bronchiolar epithelium with areas of squamous cell metaplasia and dysplasia was also found.

Conclusions. The average age of the dead (men and women) was 64.5 ± 7.9 years. The revealed morphological changes in the respiratory tract are a consequence of both viral and leukocytic aggression. These changes underlie further progression of the disease and development of its complications.

Keywords: COVID-19, autopsy, SARS-CoV-2, pathomorphology, microscopic examination.

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ПАТОМОРФОЛОГІЧНІ ЗМІНИ РЕСПІРАТОРНОГО ТРАКТУ ЗА НАЯВНОСТІ COVID-19 ЗА ДАНИМИ АУТОПСІЙ В ОДЕСЬКІЙ ОБЛАСТІ ЗА 2020–2023 РОКИ

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Дослідження зосереджено на виявленні та інтерпретації морфологічних змін у респіраторному тракті пацієнтів, які померли від коронавірусної інфекції. Аутопсійний матеріал (із загальної кількості померлих від ускладнень коронавірусної хвороби в Одесі та області за 2020–2023 рр.) досліджували за допомогою рутинних морфологічних методів. Виявлені морфологічні зміни в респіраторному тракті є наслідками як вірусної, так і лейкоцитарної агресії та лежать в основі подальшого прогресування захворювання й розвитку його ускладнень.

Ключові слова: COVID-19, аутопсія, SARS-CoV-2, патоморфологія коронавірусної хвороби.

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Стаття поширюється на умовах ліцензії

Introduction

On March 11, 2020, the WHO announced the beginning of the COVID-19 pandemic caused by the SARS-CoV-2 pathogen [1]. The first case of SARS-CoV-2 in the Odesa region was registered on March 25, 2020. The causative agent of the new COVID pandemic was SARS-CoV-2, a single-stranded RNA virus of the Coronaviridae family. The pathogenesis of COVID-19 is complex and still not fully understood [2; 3]. Despite previous data on the impact of viral infection on the entire body [4], it should be noted that society was not ready for the variable manifestation of SARS-CoV-2. At the same time, despite the rapid spread of the disease, which led to the recognition of the pandemic status worldwide, the virus itself and its manifestations were constantly changing, which led to the presence of differences in manifestations during the pandemic in different regions [5; 6].

Autopsy remains a research method that allows you to detect changes in organs and tissues throughout the body, and the results of autopsies of the first deaths from SARS-CoV-2 infection in Wuhan have long remained a reference point for the medical community around the world, but even in 2020, data were obtained that differed from the initial results [7; 8]. In particular, despite the large list of generally recognized changes in the lungs, numerous publications have differences in the description of morphological changes. This may be due, on the one hand, to regional characteristics, and, on the other hand, to changes in the virus itself during the pandemic and the corresponding change in the consequences of its presence in the body.

Based on the above, the **purpose** of this study was to identify and study morphological changes in the respiratory tract of patients who died from coronavirus infection in the Odesa region.

Materials and Methods

Among 7785 people who died from complications of coronavirus disease, 50 cases of people who died between 2020 and 2023 with diagnosed COVID-19 (from the total number of deaths from complications of coronavirus disease in Odesa and the region) were randomly selected. Their autopsies were performed at the Odesa Regional Bureau of Forensic Medical Examination. SARS-CoV-2 was confirmed by PCR either in vivo or postmortem. Autopsy material was fixed in 10 % neutral buffered formalin solution for at least 72 hours, with further standard histologic dehydration plus paraffinization, after that serial sections were prepared and stained with hematoxylin and eosin and picrofuchsin according to van Gieson. Macro- and microscopic changes in the respiratory tract were also evaluated [9]. The study was performed in accordance with the principles of the Helsinki Declaration of the World Medical Association "Ethical Principles of Medical Research Concerning Human Subjects" (2013), the procedure was done after approval from the Regional Ethical Review Board at Odesa National Medical University, protocol 11, 6th March, 2023.

Research results and their discussion

Of the 7,785 deceased patients (due to complications of coronavirus disease): 3,922 were men (50.4 %), women – 3,863(49.6 %). The largest group of the decease that were people aged ≥ 71 years totaled 3,373 cases (43.3 % of the total number of those who died from complications of coronavirus disease). Of these, 1,691 were males (50.1 %) and 1,682 were females (49.8 %). The average age of the dead (men and women) was 64.5 ± 7.9 years. According to medical history data, all the deceased had comorbid pathology (ischemic heart disease, atherosclerosis, hypertension, diabetes mellitus, obesity), multiple severe concomitant diseases, and various immunodeficiency states. During macro- and microscopic examination of the trachea and lungs of those who died from complications of COVID-19, we identified morphological features distinguishing COVID-19 from other acute respiratory viral infections. In the trachea we determined variably expressed hemorrhagic changes of the mucous membrane, weakly expressed in the proximal part and moderately or sharply manifested in the distal part and main bronchi, the mucous membrane was covered with mucus. There were also signs of blood circulation disturbance of microcirculatory vessels in submucous layer of trachea and bronchi in the form of various microangiopathies: stasis, thrombi, perivascular diapedesis hemorrhages and edema. The respiratory epithelium showed edema, dystrophy, foci of damage (Fig. 1), foci of desquamation (Fig. 2), enlarged nuclei were detected in bronchial epithelium cells (Fig. 3), basal cell hyperplasia of respiratory epithelium with formation of foci of squamous cell metaplasia, which leads to a sharp decrease in the barrier function of the epithelium.

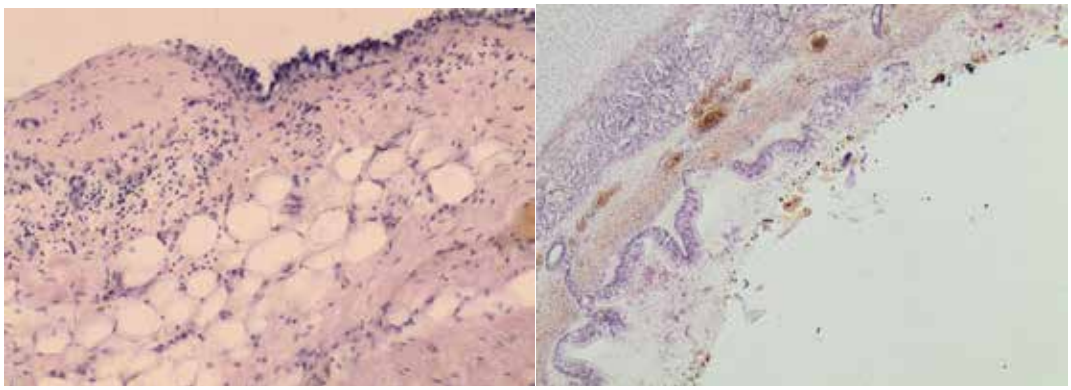

Fig. 1. Tracheal mucosa in COVID-19. Edema, epithelium is sloughed, focally necrotized, foci of epithelial proliferation with the presence of large epitheliocytes with hyperchromic nuclei, microcirculatory hemorrhage, erythrocyte sludge are determined. Inflammatory infiltration is weakly expressed. Hematoxylin and eosin staining, ×120

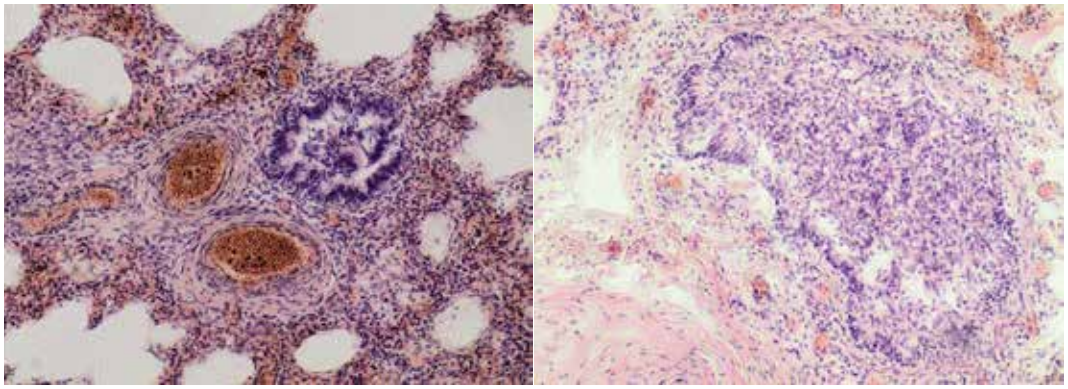

Fig. 2. Desquamation of bronchial epithelium, pronounced hyperemia. Hematoxylin and eosin staining, ×40

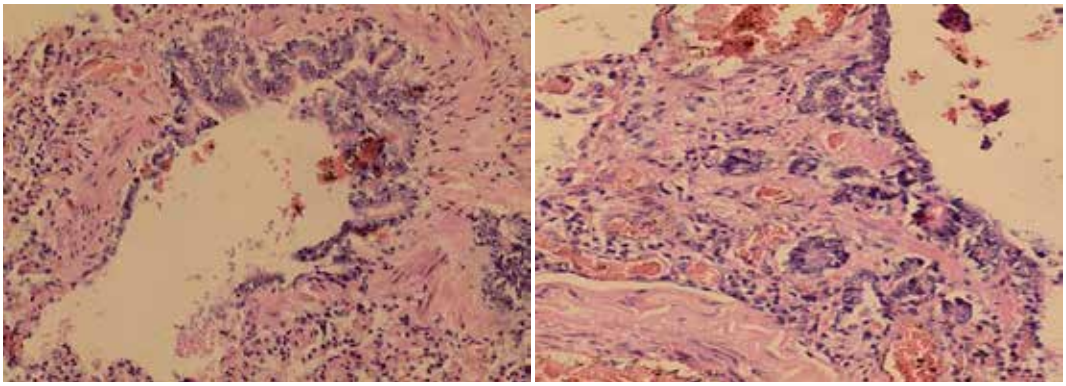

Fig. 3. Bronchial wall. Focal epithelium is absent, desquamated with foci of necrosis, enlarged epitheliocytes with hyperchromic nuclei (virus-associated changes) are determined. Hematoxylin and eosin staining, ×120

According to the results of our own observations we revealed the following morphological signs of coronavirus lung lesion: macroscopically the mass and size of the lungs were increased, the tissue was airless, unevenly compacted, on the section – dark pink, in some places red with a matte tint, in the posterior-lower sections with a whitish-gray tint, fleshy density to the touch. Dark bloody and frothy fluid flowed from the surface of lung sections when pressing. Pieces of lung tissue sank when immersed in formalin. In some cases subpleural foci of wedge-shaped, dark-red color, dense consistency were observed. The lumen of the pulmonary artery branches was obturated with crumbling masses of red color. Pleura was smooth, with injected vessels and hemorrhages – "lacquered lungs" (Fig. 4). Microscopically, at the beginning of the disease (exudative stage) diffuse alveolar lesions, "shock lungs" with accumulation of fibrinous exudate in the alveoli, acute alveolar distress syndrome, signs of viral hemorrhagic pneumonia, thrombosis of pulmonary artery branches, presence of hyaline membranes in the alveoli are detected.

Also microscopically, thickening of alveolar septa due to edema and hemorrhage, with signs of diffuse alveolar damage, acute bronchiolitis, pronounced edema and hemorrhagia in the interstitial tissue (Fig. 5), pulmonary artery thrombosis (Fig. 6), bronchospasm (Fig. 7), dystelectasis and atelectasis. Blood coagulation disorders occur due to damage to vascular endothelium as well as liver cells, with further development of thrombosis and hemorrhage. The development of vasculitis of small branches of the pulmonary artery is also characteristic of COVID-19 [17].

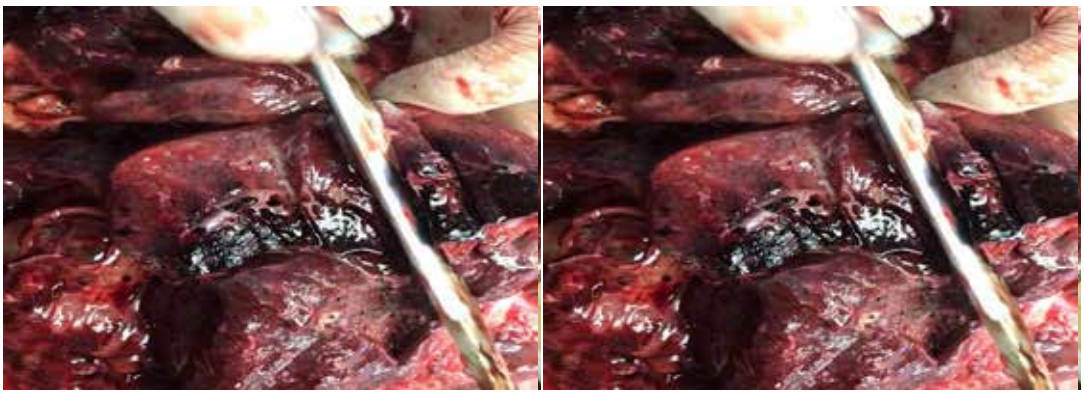

Fig. 4. "Lacquered lungs" of a deceased patient with verified COVID-19

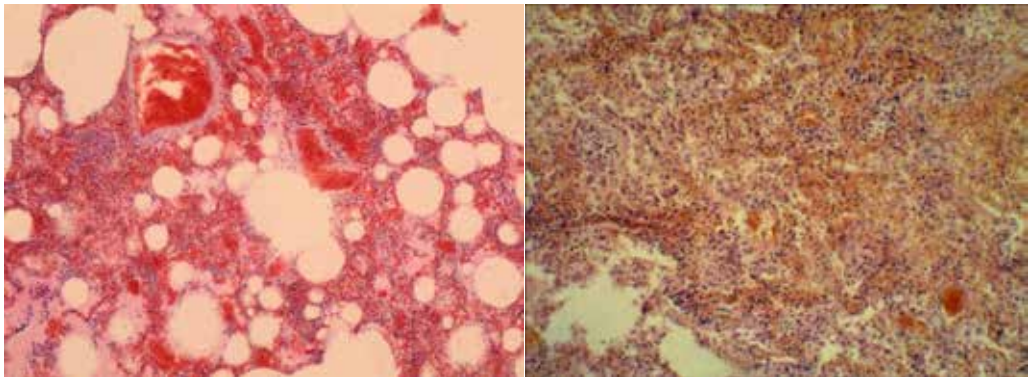

Fig. 5. Diffuse alveolar lesion. Hemorrhages in the walls of alveoli with desquamation of alveolar epithelium. Hematoxylin and eosin staining, ×100

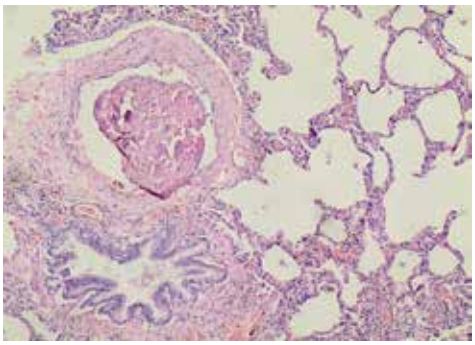

Fig. 6. Small bronchus spasm, epithelium is preserved, enlarged epitheliocytes with hyperchromic nuclei are focally determined. The lumen of the small branch of the pulmonary artery is obturated by a white thrombus with signs of organization. Hematoxylin and eosin staining, ×120

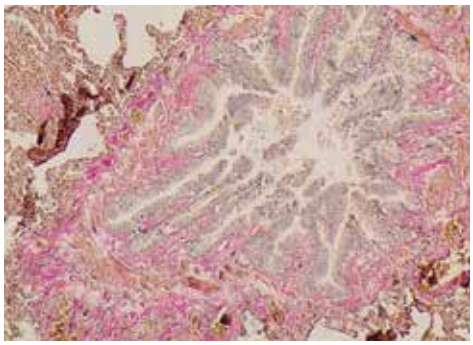

Fig. 7. Bronchial spasm. Epithelium is preserved, stasis of MCB vessels. Van Gieson staining, ×250

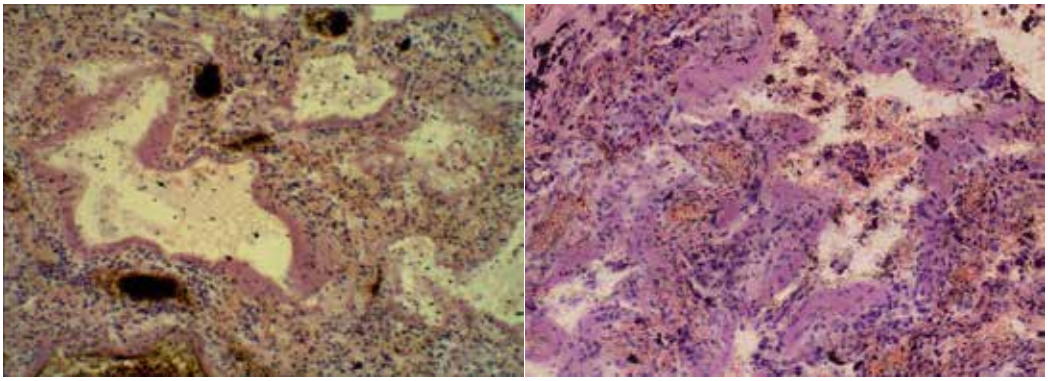

Fig. 8. Diffuse alveolar lesion, exudative phase. Multiple hemorrhages in the walls of alveoli with desquamation of alveolar epithelium, hyaline membranes. Thrombosis of microcirculatory vessels with perivascular inflammatory infiltrate. Hematoxylin and eosin staining ×100

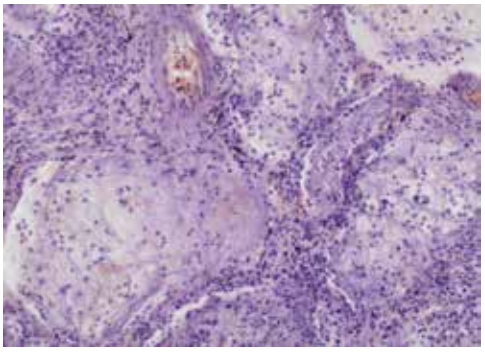

Fig. 9. Diffuse alveolar lesion. Fibrinous purulent exudate in the lumen of dilated alveoli. Hematoxylin and eosin staining, ×60

Interstitial inflammation differed in severity: from moderate in interstitial tissue to weak in vessel walls. The presence of multinucleated giant symplasts, desquamated alveolocytes with large polymorphic nuclei, granular cytoplasm, inclusions in nuclei and cytoplasm, as well as accumulations of erythrocytes, alveolar macrophages, edematous fluid, fibrin were observed in the lumen of dilated alveoli. The presence in most cases in alveoli and bronchioles of hyaline membranes (Fig. 8) lining their inner surface was also noted.

In 11 cases, virus-induced cytopathic changes in the respiratory tract and activation of bacterial and fungal flora caused the development of bilateral lobular pneumonia. Neutrophilic leukocytes and fibrin were present in large quantities in the lumen of the alveoli (Fig. 9). This morphologic picture is probably due to the immunosuppressive effect of the virus and the development of opportunistic bacterial and bacterial-fungal respiratory tract infections [17].

In the proliferative stage reparative processes of lung tissue develop with proliferation of fibroblasts and myofibroblasts, hyperplasia of type I pneumocytes, metaplastic changes in bronchial epithelium, focally with formation of adenomatous structures, dysplasia of squamous epithelium (Fig. 10), fibrosis (Fig. 11) and sclerosis of pulmonary interstitium, "carnification". Fragments of hyaline membranes may be preserved.

Among the factors leading to the changes we have described, it should be noted that in the pathogenesis of SARS-CoV-2, a very high affinity of its S-protein for the angiotensin-converting enzyme 2 (ACE-2) receptor has been identified. This protein is expressed at the receptor for ACE-2 in respiratory tract epithelia, alveolocytes, alveolar monocytes, vascular endothelium, gastrointestinal epithelia, kidney, myocardium, and some parts of the CNS. The direct action of the virus damages the endothelium of pulmonary vessels and other peripheral vessels, which induces hypercoagulability and an aggressive immune response. Due to this diffuse, massive aggression of SARS-CoV-2 towards the vascular endothelium, COVID-19 "masks" are very common in the form of exacerbation and aggravation of comorbid diseases: IHD, hypertension, diabetes mellitus, metabolic syndrome, immunodeficiency states [5; 10]. In case of the SARS-CoV-2 damage to lungs, type 1 and 2 alveolocytes and vascular endothelial cells are affected, which leads to impaired function of the aerohematic barrier and surfactant alveolar complex [11]. The main target cells for SARS-CoV-2 are alveolar epithelial cells, in the cytoplasm of which the virus replication occurs. After virions are assembled, they pass into cytoplasmic vacuoles, which migrate to the cell membrane and exit into the extracellular space via exocytosis. Expression of virus antigens on the cell surface before virions leave the cell does not occur, so antibody formation and interferon synthesis are stimulated relatively late. The formation of syncytium under the influence of the virus enables the latter to spread rapidly in tissues.

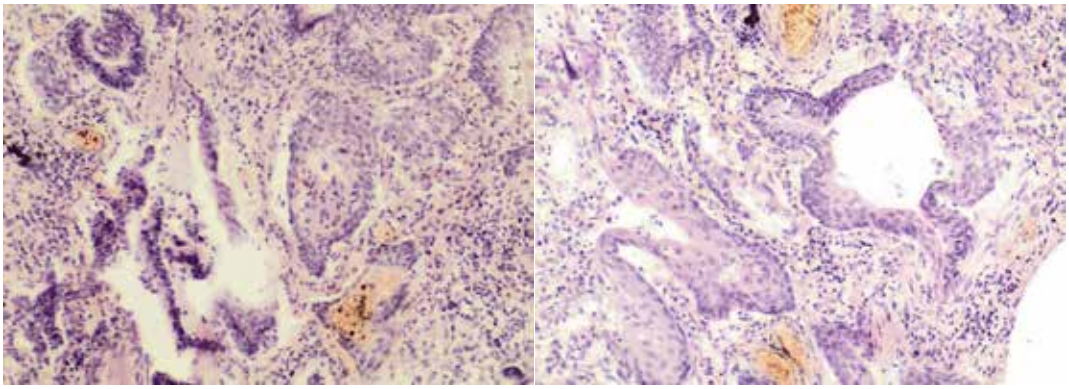

Fig. 10. Diffuse alveolar lesion, late stage. Squamous cell metaplasia of alveolar epithelium, fibrin and erythrocytes in the lumen of alveoli. Hematoxylin and eosin staining, ×120

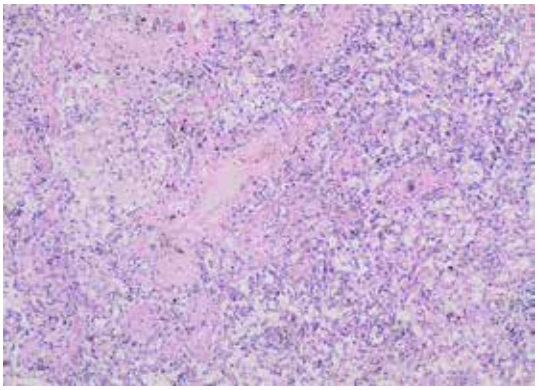

Fig. 11. Diffuse alveolar lesion, late stage. Diffuse fibrosis of the interalveolar septa. There is also focal edema and inflammatory infiltration of the interalveolar septa. Hematoxylin and eosin staining, ×40

Damaged epithelial cells produce cytokines [1] that attract leukocytes and activate neighboring endothelial cells, which stimulate infiltration which is inherent to the consequences of viral infection [12]. Also leukocytes induce production of reactive oxygen species, which in turn also damage the alveolar-capillary barrier, and activated macrophages cause apoptosis of epithelial cells [13]. Lung damage begins with diffuse alveolar damage, then against the background of cytokine storm there is tissue and vascular damage, development of inflammatory reaction and coagulopathy, destructive-productive thrombovasculitis of small arteries, development of acute alveolitis and viral-bacterial pneumonia with further fibrosis, focal adenomatosis and epithelial dysplasia [1; 7]. Viral and/or simultaneous bacterial infection, in addition to diffuse damage to alveocytes, leads to increased permeability of cell membranes, increased transport of fluid rich in albumin and fibrin into the interstitial lung tissue and alveolar lumen with the subsequent development of interstitial and alveolar edema. Along with this, changes in the elastic properties of surfactant are observed [14]. Destruction of surfactant leads to the development of alveolar collapse, a sharp disturbance of gas exchange leads to the development of acute respiratory distress syndrome. In addition to the activation of the inflammatory response, hypoxemia observed in patients with severe pneumonia and ARDS can potentiate the development of multiorgan failure, disseminated intravascular coagulation syndrome and in some cases lead to death [15]. Pathomorphologic changes in the lungs in COVID-19 are determined not only by the direct cytotoxic effect of SARS-CoV-2 on type II alveocytes, but also by the development of diffuse pulmonary intravascular coagulopathy. Changes in the lung tissue in COVID-19 are diffuse in nature, with the results of the CT examination showing the "ground-glass opacity" sign.

Our data could be used for explanation of severe epidemiologic consequences of SARS-CoV-2 pandemic events [16] with transformed inflammatory process in different organs and tissue [17; 18] and appearance of autoimmune processes.

The lungs bore the brunt of COVID-19 in autopsy studies, as has been described in previous studies [6; 19; 20]. A notable finding in our study was diffuse alveolar damage, which is commonly considered a histological hallmark of acute respiratory distress syndrome. It has three phases: an exudative phase that usually appears within 1–7 days, a proliferative phase that usually appears after 1–3 weeks, and a fibrotic phase that usually occurs after three weeks of illness [6]. In our data, we cannot state that the prevalence of proliferative diffuse alveolar damage was higher than that of exudative diffuse alveolar damage. The fibrotic phase was not observed frequently enough.

As in other studies, we found microscopic and macroscopic evidence of secondary respiratory infections in the form of bronchopneumonia and lung abscesses [6; 20]. Such infections among COVID-19 patients may be caused by prolonged hospitalization, prolonged mechanical ventilation, use of central venous catheters, immunosuppressive drugs such as steroids and tocilizumab, and potential gaps in routine infection prevention measures due to overburdened hospitals during this pandemic. An important feature of autopsy findings is evidence of thrombi and pulmonary embolism.

According to the currently accepted views on the pathogenetic mechanisms of SARS-CoV-2 lesions, in the lungs there is both a direct action of the virus on bronchial, alveolar epithelium and endothelium of small vessels, and damage associated with the action of "altered" macrophages, lymphocytes, neutrophils. The morphologic picture observed in the lungs generally corresponds to the action of the described pathogenetic mechanisms.

Conclusions

The results of the study showed that predominantly among those who died from complications of COVID-19 were people older than 71 years (43.3 %) with extensive comorbid pathology (IHD, diabetes mellitus, hypertension, malignant tumors, immunodeficiencies). Among those examined, bilateral bacterial pneumonia was diagnosed in 22 %. The predominant cause of death was severe respiratory failure, respiratory distress syndrome.

Autopsy macroscopically revealed: increase in the size of lungs, loss of airiness, presence of subpleural dark-red areas, smooth shiny pleura – "lacquered lungs".

Diffuse alveolar damage, edema and hemorrhages of interstitial tissue are determined microscopically. In the lumen of alveoli there is an accumulation of a large amount of fluid, fibrin and the presence of hyaline membranes along the walls of preserved alveoli. The presence of multinucleated symplasts, alveolocytes with polymorphic nuclei, neutrophils in the lumen of alveoli attracted attention. In vessels there was endothelium sloughing and the presence of endotheliocytes with polymorphic nuclei.

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