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Pathomorphological Damage to the Lung in Covid-19

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Abstract: During a pandemic, SARS-CoV-2 has been found to primarily affect the upper respiratory tract and lungs. Due to increased replication of the virus with the epithelium of the respiratory tract, acute respiratory distress syndrome develops; due to viremia, impaired immune system, hypoxia of tissue structures, clinical worsening of the patient's condition is noted within 2 weeks. In this work, 8 cases of autopsy material were taken to study pathomorphological changes in the lungs. It was found that under the influence of the virus, damage was observed to the integumentary epithelium of the bronchi and alveoli, tissue structures of the vascular wall and interstitial tissue, due to which the development of destruction in the form of atelectasis, distelectasis and the development of distress syndrome was noted. The peculiarity of damage to the lung tissue, vascular walls and interstitium manifested itself in the form of proliferation of connective tissue cells with the development of fibroplastic alveolitis.
Key words: virus, coronavirus, respiratory system, lung, alveolocyte, pneumonia, pneumonitis, distress syndrome.

The urgency of the problem. On March 11, 2020, the World Health Organization (WHO) declared a pandemic due to coronavirus infection, and it was considered the 11th pandemic of the XX-XXI centuries by SARS-CoV-2. SARS-CoV-2 is a single-stranded RNA virus belonging to the Coronaviridae family. The S-protein of SARS-CoV-2 is similar to angiotensin-converting enzyme 2 (APF2) and its affinity is 10 times stronger than that of the previous virus SARS-CoV, which ensures a high level of infectivity [3, 5].

APF2 receptor expression is detected in respiratory epithelium, alveolocytes, alveolar monocytes, vascular endothelium, gastrointestinal epithelium, urinary tract epithelium, macrophages and even other cells. SARS-CoV-2 is characterized by active replication in the epithelium of the upper respiratory tract. Therefore, the course and outbreak of COVID-19 causes severe acute respiratory syndrome (SARS) and SARS, whose strong replication causes viremia, immune disorders, hypoxia, and damages a number of organs, namely the heart, kidney,

gastrointestinal tract and other organs, the receptor for APF2-enzyme is expressed in the cells of these organs and causes clinical severity in the 2nd week after infection [1, 2].

At the same time, the main and fundamental essence of this disease is the development of microangiopathy in the form of destructive-productive thrombovasculitis and hypercoagulable syndrome and damage to the immune system. In severe and critical development of COVID-19, vascular inflammation affects the body's coagulation, including IL-6 as an important trigger, activates the blood coagulation system and slows down the fibrinolytic system. The direct effect of the virus on the vascular endothelium provokes hypercoagulation and causes an aggressive immune response, as a result of which the appearance of antiphospholipid antibodies increases the coagulopathy. The severe and rapid course of COVID-19 is due to a sharp decrease in the number of lymphocytes and an increase in neutrophils in the patient's body [3, 4, 5]. But the reasons for the development of lymphopenia in COVID-19 remain unknown. Based on some data, lymphopenia can be attributed to the death of lymphocytes by apoptosis or pyroptosis, as well as pathological mitosis of macrophages.

In response to SARS-CoV-2, a hyperergic immune reaction in the patient's body causes a strong systemic inflammatory syndrome, a severe alteration of the lung alveolar tissue and other organs leads to the development of septic shock. In addition to the above, many aspects of the pathogenesis and morphogenesis of COVID-19 are still unclear and undefined, including the temporary loss of smell in the respiratory tract (anosmia).

The purpose of the study is to study the pathogenesis, morphogenesis and pathomorphology of lung damage in COVID-19.

Material and methods. As a research material, 28 people who died of pneumonia caused by the coronavirus during the pandemic period in August-September 2020 were dissected and examined by the autopsy method at the Uz SSV Republican Pathological Anatomy Center. During the autopsy, samples were taken from all internal organs, including the lungs, for histological examination. The sections were processed in the usual way and paraffin blocks were prepared. Histological sections were taken from it and stained with hematoxylin-eosin. It was studied under a light microscope and the necessary areas were photographed. For the preparation of this article, histological sections from the lungs were studied in 10, 20, 40 objects of a light microscope, and microphotographs were taken that show the most significant pathomorphological changes that develop in lung tissue under the influence of coronavirus. From respiratory tracts, terminal bronchioles and alveolar tissue were studied.

Research results and discussion. The results of the investigation showed that the SARS-CoV-2 virus primarily damages type II and III alveolocytes. At the same time, the lower part of the respiratory tract, i.e. the bronchioles and respiratory bronchioles, develops a number of pathological changes, damaging the mucosa covering epithelium. Microscopically, the covering epithelium swells due to dystrophic changes in both its cytoplasm and nucleus, its shape changes, its nucleus is irregularly located, escapes from its basement membrane, some desquamates and migrates from its place. Others adhere to each other and form hyperchromic pads, others take on a multiline form, and still others become flattened, turning into a thin eosinophilic membrane, in which the nuclei also become smaller and flattened. In this case, the bronchiole cavity is filled with a large number of desquamated epithelium, erythrocytes, lymphoid cells, macrophages and other tissue fragments. It is observed that the basal membrane of the wall of the bronchiole is severely swollen, myxmatous, and it is determined that there are disorganized fibrous structures in its composition. It is determined that activated macrophages, lymphoid cells, erythrocytes and necrobiotic detritus are present in it (Fig. 1).

Therefore, due to the damage and destruction of the covering epithelium and basal membrane of the bronchiole wall, the virus and its toxins spread to the lung tissue around the bronchioles and cause inflammation.

Microscopic examinations showed that SARS-CoV-2 mainly damages type II alveolocytes. Micrographs show that type II alveolocytes in all alveoli are severely enlarged, both cytoplasm and nucleus. In particular, it is observed that the cytoplasm has increased in size and entered into an unclear shape, it is stained with eosin in a chaotic manner, it is desquamated, and it falls into the alveolar cavity (Fig. 2). In some places, it is determined that they are connected to each other, forming large multinucleated cells. It can be specially noted that the alveolar tissue damaged by the virus has lost its normal histotopography, the tissue structures are chaotically located. Due to migration of covering epithelium, alveoli walls are broken and destroyed by blood vessels and connective tissue. Strong swelling, myxomatosis and infiltration of lymphoid cells are detected in them. In other areas of lung alveolar tissue, it is observed that discirculatory changes, i.e., diffuse hemorrhages predominate. In these areas, the space of the alveoli, the interstitial tissue is diffusely filled with erythrocytes (Fig. 3). In these areas as well, it is determined that alveolocytes have undergone dystrophy and destruction, large multinucleated giant cells have appeared.

Therefore, it is observed that the alveoli are affected by pathological atelectasis, dystelectasis and distress syndrome due to the severe damage and destruction of the epithelium covering the alveolar wall, blood vessel and interstitial connective tissue under the influence of the virus.

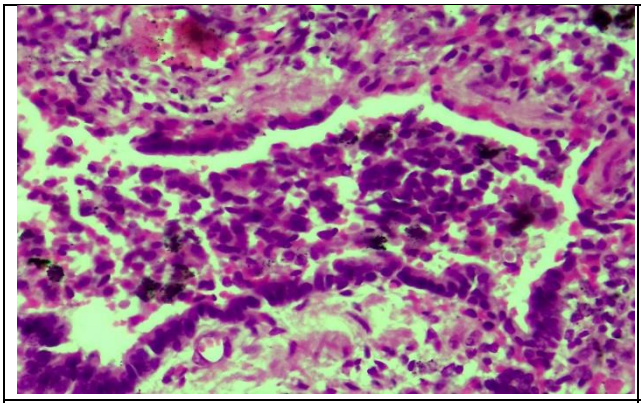

Figure 1. Due to the effect of coronavirus, the bronchiole cavity is filled with cellular mass, the covering epithelium undergoes various changes, the basement membrane is swollen, infiltrated with myxomatosis and inflammatory cells, and blood is shed. Paint: G and E. X: 10x40.

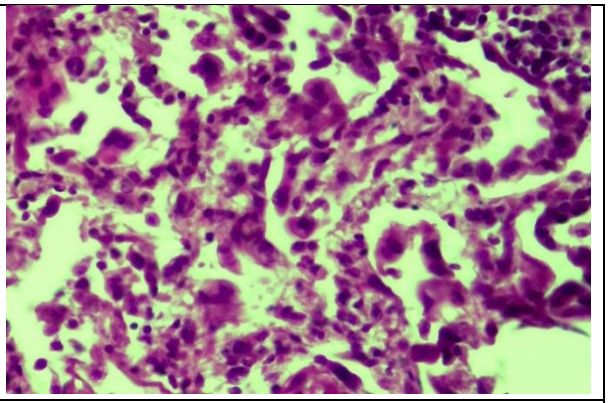

Figure 2. Under the influence of the coronavirus, alveolocytes of type II increased in size and shape due to dystrophy and destruction, interstitial tissue was destroyed and infiltrated with lymphoid cells. Paint: G and E. X: 10x40.

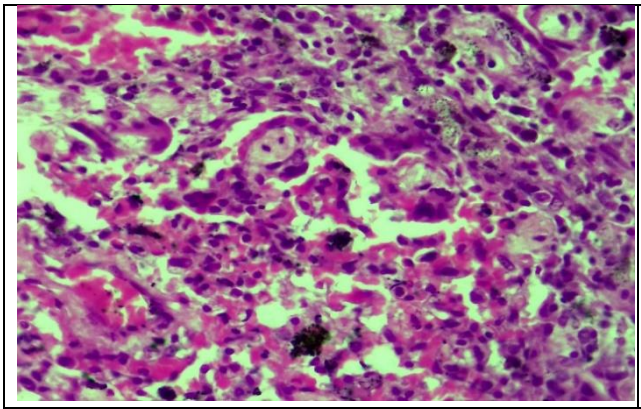

Figure 3. Due to the effect of coronavirus, the alveolar cavity and interstitial tissue are diffusely filled with erythrocytes, the alveolocytes are dystrophied and destroyed, large multinucleated giant cells appear. Paint: G and E. X: 10x40.

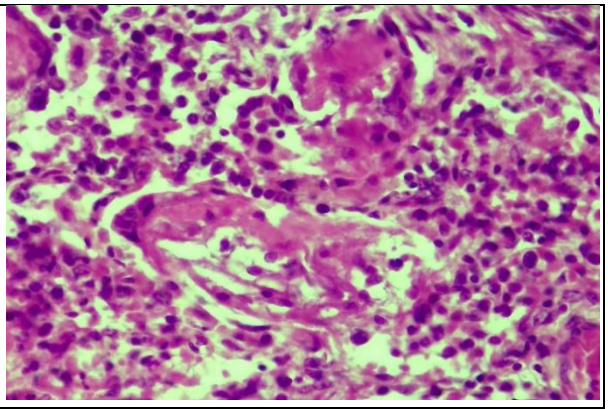

Figure 4. Type II alveolocytes bound with eosinophilic fiber protein and filled the alveolar cavity. Paint: G and E. X: 10x40.

As a result of severe dystrophy and destruction of type II alveolocytes, pathological protein substances are synthesized from them instead of the standard surfactant. As a result, fibrous structures, which were initially randomly located in the alveolar cavity, form a unique network and fill the alveolar cavity. In this case, alveolar wall structures were completely destroyed and diffusely infiltrated with lymphoid cells (Fig. 4). As a result of the movement of the lung tissue and air entering the alveoli, coarse protein substances formed in the alveolar cavity accumulate at the edge of the alveolar cavity, that is, on the inner surface of the alveolar wall, forming hyaline membranes (Fig. 5). As a result, oxygen exchange on the surface of the alveolar wall becomes difficult and hypoxia develops. Therefore, in most cases, damage to type II alveolocytes, production of coarse fibrillar protein instead of surfactant, and formation of hyaline membranes are confirmed under the influence of coronavirus.

It is known that the enzyme ASE2 of cells is an integral part of the renin-angiotensin system (RAS) and its function controls the homeostasis of the cardiovascular system, controls systolic blood pressure, osmotic and electrolyte balance. Under the influence of coronavirus, the activity of this enzyme increases, and this mechanism is strengthened, the bronchial wall shrinks smooth muscle tissue, lung fibroblasts proliferate, alveolar epithelium undergoes apoptosis, vascular wall permeability increases, and leads to acute respiratory distress syndrome. If ASE2 acts through the Mas receptor, it causes vasodilation and lowers blood pressure. Based on these mechanisms, if we shed light on the microscopic changes of blood vessels in lung tissue damaged by SARS-CoV-2, the following can be said. All the vessels of the lung tissue are vasodilated, widened and full. In particular, the venous vessels are sharply dilated, filled with blood, the permeability of their walls increases, and blood is poured around them by the diapedesis method (Fig. 6). Arteries are also relatively widened, plump, but all layers of their walls are thickened due to edema, myxomatosis and inflammatory infiltrate. Innumerable capillaries in the interalveolar tissue are also diffusely dilated and full, blood is poured diapedesically into the surrounding tissue and alveolar space (Fig. 7).

It is known from the above-mentioned mechanisms that under the influence of SARS-CoV-2, the endothelium of blood vessels is also damaged. As a result, strong dystrophy and destruction processes develop in endothelial cells, their cytoplasm swells, desquamates, and moves out of place. Damage to the endothelium of blood vessels is the main local cause of the thrombosis

process, which leads to the coagulation of blood cells and fibrinogen in the vessel cavity. As shown in the microphotograph below, it is confirmed that fibrin protein and white blood cells have accumulated in the space of postcapillary venules, in other words, microthrombi have appeared (Fig. 8).

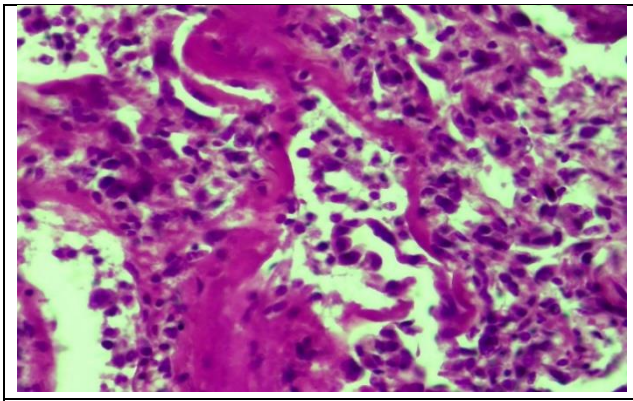

Figure 5. The resulting eosinophilic hyaline membranes covered the inner surface of the alveoli. Paint: G and E. X: 10x40.

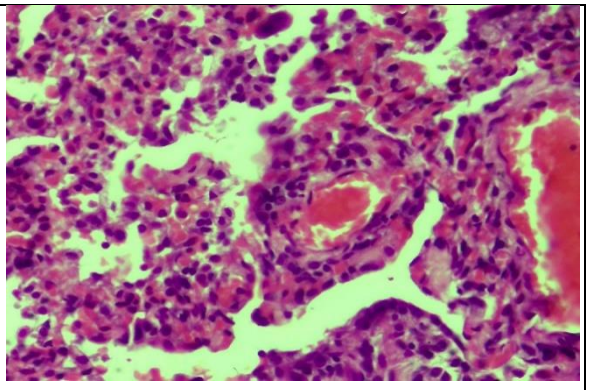

Figure 6. Under the influence of the coronavirus, the large veins and arteries have thickened due to disorganization and inflammation. Paint: G and E. X: 10x40.

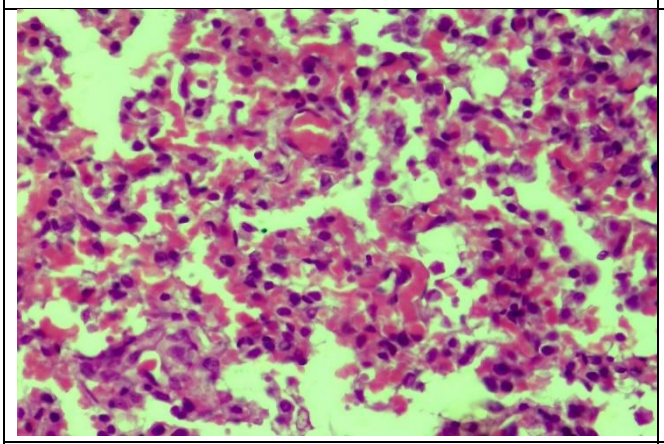

Figure 7. In the tissue between the alveoli, the capillaries are filled diffusely, and blood is poured around and into the alveolar cavity by diapedesis. Paint: G and E. X: 10x40.

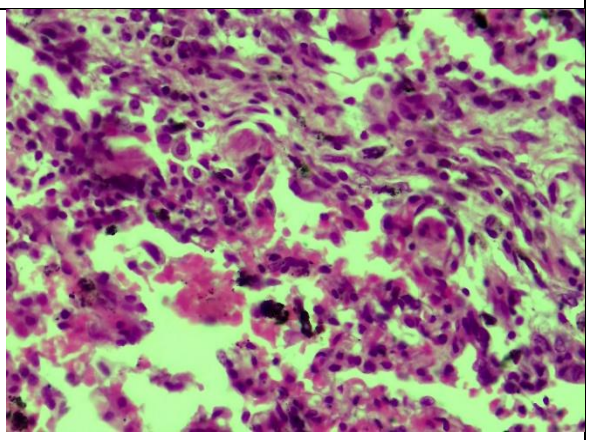

Figure 8. Fibrin thrombi appeared in the space of postcapillary venules of lung tissue. Paint: G and E. X: 10x40.

Summary

- It was observed that type II and III alveolocytes were initially damaged under the influence of coronavirus, their nucleus and cytoplasm were deformed and took different forms, polymorphous and giant cells appeared, desquamated and filled the alveolar cavity.
- Pathomorphological changes specific to COVID-19 occurred in the pulmonary blood vessels, the endothelium suffered dystrophy, destruction and desquamation, the basement membrane was severely swollen and disorganized, as a result of which plasma fluid and proteins poured into the wall and cavity of the alveoli, hemorrhagic exudate and foci of hemorrhage appeared. is determined.
- Due to the effect of coronavirus, it is determined that fibroblasts proliferated in the tissue of the bronchi, around blood vessels, and between the alveoli, a specific productive infiltrate appeared, and finally, interstitial fibromatous alveolitis developed in the lungs.

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