PATHOMORPHOLOGICAL ASSESSMENT OF RENAL VESSEL CHANGES IN COVID-19

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Relevance. At the end of 2019, humanity faced the spread of a new coronavirus infection (NCoV) with a pandemic character. The characteristic features of SARS-CoV-2 are that the virus is highly contagious, it penetrates into various tissues of the human body, it is capable of causing diseases - severe damage to many organs is caused by the effects of viral infections.

Although the main target of the virus is lung tissue, the kidney is one of the most affected organs. Mechanisms of kidney damage in infection, forms of nephropathies are also diverse.

There is increasing evidence that COVID-19 can cause kidney damage, with proteinuria, hematuria, and elevated serum creatinine on admission. The high frequency of acute kidney injury and the spectrum of pathologic abnormalities, including acute tubular necrosis, endothelial damage and capillary occlusion, deposition of the complement complex in the tubules, and glomerular damage identified in autopsy reports also allow establishing a link between coronavirus infection and kidney pathology.

Aims and objectives of the work: pathomorphological assessment of changes in the blood vessels of the kidneys of those who died from COVID-19.

Materials and methods: Medical history, autopsy report data of 86 patients who died of COVID-19 and were examined at RPAC were analyzed. At the autopsy, the cut pieces taken from the organs were frozen for 72 hours in a formalin solution prepared in 10% phosphate buffer, stained with hematoxylin-eosin method.

Results: In the morphological study of blood vessels of the kidneys, the entrance artery of the kidneys, the arcuate artery between the cortical and medullary layers, the artery that brings blood to the capillaries, and the efferent artery, including the secondary peritubular artery branches of the efferent artery, were studied. When the wall of the artery entering the kidneys was

studied, it was found that the outer adventitia, the middle muscle layer, which formed the wall of this artery, did not develop significant pathomorphological changes. Only in the intima of the inner surface of the artery wall, a slight twisting, flattening of the endothelial cells, hyperchromization of the basal membrane, and deformation of the connective fibers were found. These changes were manifested by the general pathomorphological changes developed in response to the process of viral infection - that is, general surface disorganization of tissue structures.

When the wall of the arcuate arteries located between the layers of the renal cortex and medulla was studied, it was observed that all layers of the arterial wall were equally developed swelling and deformation. As a result, it was found that the cavity of the arteries narrowed, the erythrocytes in the cavity were located in a disorderly state, the artery wall adhered to the inner surface, and specific collections appeared. It was observed that the intima layer of the arterial wall was more swollen, thickened, bulging in some areas, and sinking into the muscle layer in other areas.

It was found that plasma proteins were concentrated on the inner surface of the intima layer, and a homogenous protein with pale eosinophilic appearance was formed. Endothelial cells were observed to be stretched and deformed due to swelling and disorganization. It was found that the basal membrane was slightly thickened and deformed in some places due to edema and plasmarrhagia. It was observed that macrophages and lymphocytes appeared in some areas of the intima of the arterial wall.

When the muscle layer was studied, it was found that muscle cells and myofibrils were located in one direction, fragmented and vacuolated due to the presence of interstitial swelling in some places. In the adventitial layer, similar to the intima, it was found that strong pathomorphological changes have developed due to swelling and disorganization. It was observed that the blood vessels in the adventitia were full, blood was poured around them by the diapedesis method, the fibrous structures of connective tissue were homogenized due to mucoid swelling and were destroyed in some places.

It was observed that the relatively smaller network of arcuate arteries between the kidney layers was also strongly deformed, swelling, mucoid swelling and fibrinoid necrosis developed in its wall layers. It was found that the cavity of this artery was also sharply narrowed, and erythrocytes were randomly located in it. It was found that some of the endothelial cells of the inner layer were swollen, others were displaced and desquamated, and

lymphocytes were attached to some of them. It was found that the wall of these arteries has strong destruction foci in the muscle layer, which has turned into a structureless tissue due to myolysis, myorexis and destruction of the muscle cells there. Areas of fibrinoid necrosis, hemorrhages, and strong edematous foci were found in the interstitial connective tissue around the artery.

Summary. Thus, after studying the scientific articles, it can be concluded that the kidneys are often the target of the SARS-CoV-2 virus.

Several interrelated pathogenetic mechanisms are involved in the development of the pathological process, which leads to the development of a vicious circle: cytopathic effect of the virus on kidney tissues and vessels due to the expression of APF2 receptors; the formation of a cytokine storm and a systemic inflammatory reaction leading to coagulopathy leads to the development of systemic vasculitis of organs and tissues with the formation of many microstromes.