

Evaluation of Kidney Pathologies – Pathogenetic and Functional Changes Caused By SARS-Cov-2

Gapparova Guli Nurmuminovna

Samarkand State Medical University, Republic of Uzbekistan, Samarkand

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Annotation: The coronavirus is highly infectious and highly tropism for kidney tissue. The new coronavirus infection can cause a wide range of pathological abnormalities in the kidneys. Various clinical forms of kidney damage in COVID-19 are caused by many pathogenetic mechanisms, such as direct cytopathic effect of the virus on kidney structures, endothelial dysfunction, cytokine storm, hemodynamic and water exchange disorders, renin-angiotensin-aldosterone damage. SARS-CoV-2 interacts with ACE2 receptors located on the endothelium of blood vessels and has a negative effect on the microvascular shell. Kidney damage in patients with COVID-19 includes nosological forms such as collapsing nephropathy, glomerulopathy, nephritis, acute tubular necrosis, exacerbation of autoimmune glomerulonephritis. During clinical observations, scientists from different countries found a connection between confirmed COVID-19 and the following laboratory data: hematuria, proteinuria, high levels of nitrogen in the blood, increased creatinine, uric acid, etc. were observed. In addition, the presence of risk factors in the patient, such as chronic kidney disease, cardiovascular pathology, immunodeficiency, taking nephrotoxic drugs, diabetes, hypertension, obesity, atherosclerosis, old age complicates the course of the disease and worsens the outcome. Thus, the pathological effect of the coronavirus on the body as a whole and on the kidneys in particular, the high mortality rate of patients with kidney pathology determines the urgency of studying this problem and finding ways to solve it.

Keywords: COVID-19, kidneys, coronavirus, kidney damage, nephropathies, SARS-CoV-2.

Relevance of the research work: At the end of 2019, humanity faced a pandemic of a new coronavirus infection. A distinctive feature of SARS-CoV-2 is its high infectivity, tropism for various tissues of the human body, especially the kidney.

Despite the fact that the main organ of the virus is lung tissue, the kidneys are also one of the most affected organs. Mechanisms of kidney damage caused by viruses, forms of nephropathy are also different.

A number of indicators, such as proteinuria, hematuria, and an increase in the concentration of creatinine in the blood serum, are considered evidence that COVID-19 can damage the kidneys. The spectrum of acute kidney injury and pathologic abnormalities found at autopsy, including acute tubular necrosis, endothelial damage, tubular complement deposition, and glomerular damage, also suggest a link between coronavirus infection and kidney pathology.

All this determines the relevance of studying the mechanisms and clinical manifestations of kidney diseases in the era of COVID-19.

Pathogenesis of kidney damage in COVID-19.

Many pathogenetic mechanisms of kidney damage in patients with COVID-19 can be divided into several groups:

1. Direct cytopathic effect of the virus on kidney structures

It is known that the main receptor for the SARS-CoV-2 virus to enter the cell is the angiotensin-converting enzyme 2 (ACE2) receptor, which is expressed in various organs, including the lungs, heart, intestines and kidneys. [4-8]. In the kidneys, this receptor is located on podocytes, mesangial cells, Bowman's capsule parietal epithelium, proximal tubule cells, and the collecting duct. Recent research shows that RNA sequencing of human tissues has confirmed that ACE2 is almost 100 times higher in the kidneys than in the lungs [10]. Therefore, kidney diseases can occur as a result of the penetration of the coronavirus into kidney cells through ACE2 receptors.

Studies have shown that kidney cells contain ACE2, transmembrane serine protease 2, and cathepsin L RNA, which have single-cell RNA sequences (targets of SARS-CoV-2). Also, when quantifying the viral load of SARS-CoV-2 in some kidney structures obtained using tissue microdissection, damage to glomerular cells was found in all parts of the kidneys of the patient who underwent an autopsy [11].

2. Endothelial dysfunction

COVID-19 is a major risk factor for the development of coagulopathy. SARS-CoV-2 interacts with ACE2 receptors located on the endothelium of blood vessels, which causes endothelial dysfunction, which leads to vasoconstriction, hyperpermeability of blood vessels, impaired microcirculation, the development of vascular thrombophilia, numerous microthrombosis and blood can lead to death, necrosis, hemorrhagic infarction of various organs. [9, 13].

The reports of many scientific researchers (Z. Varga, A. J. Flammer) showed that against the background of COVID-19, damage to the kidneys and other organs is caused by direct viral damage, endotheliitis due to the inflammatory reaction. Electron microscopy of a section of a transplanted kidney from a patient who died of multiple organ failure developed against the background of COVID-19 revealed the incorporation of virions in the endothelial cells of blood vessels. Histological examination showed infiltration of the endothelium with inflammatory cells. The study of histological preparations taken from other patients also confirms the presence of endotheliitis in various organs - lungs, heart, kidneys, liver, intestines. Findings suggest that

endothelial dysfunction is a major factor in microvascular dysfunction, which subsequently causes multi-organ dysfunction [18]. COVID-19 is a common viral vasculitis that pathogenetically causes significant damage to arterioles [18].

3. Acute renal failure associated with cytokine storm and acute respiratory distress syndrome. factor tumors, etc develops an immune reaction characterized by synthesis. Inflammation causes tissue destruction, spreads to adjacent tissues, and progresses systemically. Cytokine storm is believed to be a key factor in the development and progression of nonpulmonary multiorgan failure [11, 22]. Acute renal failure, which develops against the background of acute respiratory distress syndrome, occurs in approximately 35-50% of patients and significantly increases the probability of death. There are several reasons for the development of O'BZ: hemodynamic effects (leading to increased pressure in the pulmonary artery, right ventricular failure, venous congestion and increased intra-abdominal / intrathoracic pressure), gas exchange disorders - hypoxemia/hypercapnia, decreased renal blood flow and acid-base imbalance), hyperinflammation and neurohormonal effects (for example, activation of the renin-angiotensin-aldosterone system) [15, 17].4. Gemodinamik o'zgarishlar

AKI can also cause other complications of COVID-19, such as right and left ventricular failure. The first leads to stagnation of blood in the kidneys, and the second leads to a decrease in cardiac function and the development of renal hypoperfusion [19, 20].

5. Violation of water exchange

Hypovolemia resulting from fever and tachypnea may affect the kidneys through a prerenal mechanism. This condition causes renal hypoperfusion and subsequent renal failure. Metabolic acidosis and hyperkalemia are also associated with this condition. This has a significant impact on the deterioration of kidney function and subsequent occurrence of O'BZ [16].

6. Renin-angiotensin-aldosterone system (RAAT) damage

It is also caused by the interaction of the virus with the ACE2 receptor, which leads to the disruption of RAAT, the accumulation of angiotensin II and bradykinin, causes pulmonary edema, myocarditis, and causes vasodilatation, which causes natriuresis [8, 13].

Kidney disease in COVID-19 is caused by the presence of the following risk factors in the patient: cardiovascular pathology, acute heart failure and, as a result, the development of cardiorenal syndrome, the presence of immunodeficiency conditions, and taking nephrotoxic drugs. Diabetes, hypertension, obesity, atherosclerosis, and old age are also risk factors for SARS-CoV-2 infection, the presence of which complicates the clinical course and worsens the outcome of the disease. According to the International Society of Nephrology, kidney damage is observed in 25-50% of severe cases of COVID-19 [12, 17, 21].

Clinical manifestations of kidney pathology caused by COVID-19.

Kidney damage caused by COVID-19 includes collapsing nephropathy, membranous glomerulopathy, nephritis, acute tubular necrosis, exacerbation of autoimmune glomerulonephritis.

During 2020-2022 in Samarkand, a total of 412 children were diagnosed with COVID-19 and the following laboratory data were obtained: average proteinuria (from 0.3 to 3 g/l) in 128 of 412 patients with COVID-19), hematuria was observed in 98, leukocyturia -289.

Chinese scientists were also able to identify a complication in the form of acute kidney injury in critically ill patients with COVID-19. The main pathologic finding was acute tubular injury. The level of IL-6 in the blood serum of elderly patients is a risk factor for O'BZ. Acute kidney injury in stage 3 inevitably led to death.

Another Chinese study looked at laboratory tests, clinical symptoms, and organ function status in 193 adult patients with COVID-19. Results were compared between three groups: non-severe

(128) and severe COVID-19 patients (65) and a control group. Researchers have found an increase in kidney dysfunction and O'BZ in patients with COVID-19. Proteinuria, hematuria, and elevated blood urea, serum creatinine, and uric acid levels were significantly associated with mortality in patients infected with COVID-19. It was shown that the risk of death in patients with secondary AKI infected with COVID-19 was 5.3 times higher than in patients without AKI.

CONCLUSION.

Thus, after studying Uzbek, Russian and foreign scientific articles, we can conclude 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: the cytopathic effect of the virus on kidney tissues and blood vessels caused by the expression of the ACE2 receptor; formation of a cytokine storm and a systemic inflammatory reaction leads to coagulopathy with the formation of many microthrombi, causes the development of systemic vasculitis of organs and tissues.

In the course of research, many experts around the world were able to determine the connection between exposure to the coronavirus and the occurrence of various forms of nephropathy: from acute kidney injury to kidney failure. As a result, it remains urgent to study the effects of COVID-19 on the state of the kidneys and to search for ways to reduce mortality from severe forms of nephropathy.

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