TASHKENT MEDICAL ACADEMY

100 TMA ANNIVERSARY

Journal of Educational and Scientific Medicine

Issue 3 | 2024

OAK.uz

Google Scholar

ISSN: 2181-3175

Journal of Education & Scientific Medicine

ISSN 2181-3175

Tashkent Medical Academy

Review Article

Open © Access

Pathogenesis of Kidney injury in patients with COVID-19

I.A. Khursandov[1]

ABSTRACT

The SARS-CoV-2 pandemic has shaken the world since its initial outbreak in December 2019. As of November 2021, the World Health Organization has reported more than 26.1 million cases and 5200000 deaths, and these numbers continue to rise as more and more episodes related to the effects of the disease emerge. Country-by-country mortality rates provided by Johns Hopkins University range from 0.1% to 9.2% with significant geographical differences. Along with damage to the respiratory system and the cardiovascular system, an important role is played by pathologies of the kidneys, which are characterized by the development of both acute and chronic renal failure. Particular difficulties arose in the treatment of patients in need not only of artificial ventilation, but also of hemodialysis, in the need for hospitalization of patients in intensive care units. This was the reason for the development of economic problems in the health care system. This review article is devoted to the pathogenesis of kidney damage in patients with COVID-19.

Ключевые слова: *COVID-19, острая почечная недостаточность, патогенез*

The pathogenesis of kidney damage in patients with COVID-19 is still not fully understood. The study of kidney damage in patients with COVID-19 is due to significant medical, economic and social problems of the consequences leading to the chronicity of diseases of this body system. Although this issue has not been sufficiently studied, there is nevertheless information regarding studies of this kind.

Researchers from Spain conducted an observational retrospective study, which reported that only 54.3% of patients achieved recovery of kidney function after the patient was discharged from the clinic for COVID-19 [4]. Along with this, scientists from Italy reported achievements at the level of 67% of cases of recovery of kidney function after COVID-19 [14].

Studies by scientists from Mexico have shown that the recovery of kidney function after COVID-19 also depends on the development of acute renal failure at the in-hospital or out-of-hospital stages. Thus, in the first case, recovery of kidney function after COVID-19 was achieved in 94% of cases, and in the second case, in 62% of cases [8].

As these literature data have shown, the consequences of kidney damage in patients with COVID-19 and the subsequent restoration of the function of this organ indicate that many issues of the pathogenesis of acute renal

[1] Corresponding author: Chief Physician of the Sultan Hospital Private Clinic, Termez, Uzbekistan, e-mail: ilyosxursandov@gmail.com

failure in this viral infection are still not fully understood.

One of the first conclusions about the role of COVID-19, acute respiratory distress syndrome, including the use of mechanical ventilation, and the development of acute renal failure was made by German clinicians in 2022 [16].

Acute renal failure associated with COVID-19 is common in critically ill patients. Renal tropism of severe acute respiratory syndrome coronavirus type 2 (SARS-CoV-2) appears to play only a minor role, while the pathological inflammatory response associated with severe COVID-19 is highly relevant. Both the effects of mechanical ventilation and acute respiratory distress syndrome caused by COVID-19 have a significant impact on the pathogenesis of acute renal failure. High ventilation pressure impairs renal perfusion and thus may contribute to the development of acute renal failure. The inflammatory response caused by acute respiratory distress syndrome, as well as endothelial dysfunction typical of COVID-19, combined with hypercoagulability, are additional factors affecting the kidneys.

It has been suggested that the SARS-CoV-2 virus has a direct cytopathic effect on the kidneys through angiotensin-converting enzyme type 2 receptors, which are found in proximal tubule cells and podocytes. This assumption was made by H.W. Pan, et al. [13] based on the results obtained on the study of the mechanisms of entry of the SARS-CoV-2 virus into lung cells, namely, through the receptors of angiotensin-converting enzyme type 2, the virus enters the cell in the lungs.

E.A. Farkash, et al. [27] were among the first to find evidence of a direct effect of the SARS-CoV-2 virus on the kidneys. Scientists from Michigan (USA), when conducting electron microscopy of the kidneys of patients who died from COVID-19, revealed the presence of virus-like inclusions in the cells of the tubules of the kidneys.

Meanwhile, C. Roufosse, et al. [11] in a similar study also revealed virus-like inclusions in the cells of the tubules of the kidneys. However, they reduced the interpretation of their results to the presence of extruded microvesicles, which are in no way related to the SARS-CoV-2 virus.

A targeted search for the direct effect of the SARS-CoV-2 virus on the morphofunctional structure of the kidneys was made by German scientists led by V.G. Puelles, et al. [19]. They managed to identify the ribonucleic acid of the SARS-CoV-2 virus on autopsy material. This was made possible by the use of in situ hybridization methods in combination with polymerase chain reaction. Characteristic changes in kidney damage were revealed mainly in the glomeruli of the kidneys and in the tubules. Such a total defeat was characteristic of the SARS-CoV-2 virus.

The results obtained and the development of research methods gave impetus to the subsequent search for pathogenetic mechanisms of kidney damage in patients with COVID-19. In particular, other studies have proven that there is a close correlation between SARS-CoV-2 tropism to the kidneys and the clinical outcomes of COVID-19 [25]. Matrix ribonucleic acid of the SARS-CoV-2 virus of the kidneys was identified in 2/3 of patients with kidney damage. Along with this, similar changes were also found among 1/3 of patients. It has also been proven that the SARS-CoV-2 virus can be in a viable state in the patient's kidneys and has the potential to multiply. This confirms the theory that the virus can penetrate directly into the renal parenchyma. However, to date, direct evidence of this phenomenon has not yet been obtained.

Kidney damage in patients with SARS-CoV-2 has a focal and segmental nature in its morphological structure, which can be equated to glomerulosclerosis in its course. Based on this, the presence of a specific tropism of SARS-CoV-2 on the kidneys was proven. This conclusion was obtained by H. Wu, et.al. in 2020, a morphological study of biopsy material from patients with a confirmed diagnosis of COVID-19, acute renal failure, and proteinuria [6].

Particles of SARS-CoV-2 were also found in the urine samples of patients. Histological studies showed the presence of collaborating glomerulopathy with destructive changes in the tubules of the kidney. Studies by H. Su, et al. [24] and J. Sun, et al. [15] did not detect the presence of the SARS-CoV-2 virus or its particles. However, focal-segmental glomerulosclerosis detected in kidney tissues, like podocytopathy, was associated with human immunodeficiency virus, parvovirus B-19 and cytomegalovirus, for which direct viral damage to the kidneys was proven.

Studies by V. Jeyalan, et al. [20], which were based on a meta-analysis of histological studies of the kidneys in patients with COVID-19, showed a wide variety of pathologies, ranging from collaborating focal segmental glomerulosclerosis and acute tubular injury, to vasculitis, thrombotic microangiopathy, and pigmentary nephropathy.

In a multicenter retrospective cohort study, a group of scientists led by R.M. May [1] determined the spectrum

of kidney pathologies in COVID-19. An international collaboration analyzed 284 kidney biopsies to improve understanding of kidney disease in COVID-19. The diagnoses were compared with figures for five years, when 63,575 native biopsies were performed before the pandemic, and 13,955 allografts were carried out to identify diseases that have become more frequent in patients with COVID-19. Genotyping for the APOL1, G1, and G2 alleles was performed in 107 African-American and Hispanic patients. The SARS-CoV-2 immunohistochemical assay was used to assess direct viral infection in 273 cases along with clinical information at the time of biopsy. The main indication for native biopsy was acute renal injury (45.4%), followed by proteinuria with or without concomitant acute renal injury (42.6%). There were more African Americans (44.6%) than patients of other ethnicities. The most common diagnosis on native biopsy was collapsed glomerulopathy (25.8%), which was associated with high-risk APOL1 genotypes in 91.7% of cases. Compared to the five-year biopsy database, the incidence of myoglobin casting nephropathy and proliferative glomerulonephritis with IgG monoclonal deposits was also increased in COVID-19 patients (3.3% and 1.7%, respectively), with a decrease in the incidence of chronic conditions (including diabetes mellitus, IgA nephropathy, and arterionephrosclerosis) as the primary diagnosis. In transplantation, the leading indication was acute kidney injury (86.4%), for which rejection was the predominant diagnosis (61.4%). No direct SARS-CoV-2 virus infection has been detected. Thus, a multicenter series of major cases identified kidney disease that disproportionately affects COVID-19 patients and demonstrated a high incidence of high-risk APOL1 genotypes in this group with no evidence of direct viral infection in the kidneys.

During the COVID-19 outbreak in Wuhan, China, significant abnormalities in blood clotting parameters in patients with severe pneumonia of SARS-CoV-2 coronavirus infection were of serious concern. In this regard, studies by N. Tang, et al. [2] on abnormal blood coagulation indicators associated with a poor prognosis in patients with COVID-19 presented retrospectively analyzed the results of conventional coagulation and the outcomes of 183 consecutive patients with confirmed SARS-CoV-2 infection at Tongji Hospital. All-cause mortality was 11.5%, those who did not survive had significantly higher levels of D-dimer and fibrin degradation product (FDP), longer prothrombin time, and activated partial thromboplastin time compared to survivors on admission ($p<0.05$); 71.4% of non-survivors and 0.6% of survivors met the criteria for disseminated intravascular coagulation during hospital stay. The study shows that abnormal clotting results, especially markedly elevated levels of D-dimer and fibrin degradation product, are common in deaths from SARS-CoV-2. Acute renal failure in patients with COVID-19 can also be the result of vascular microthrombosis [10].

The basis for such a conclusion can be the results of a number of studies. It has been proven that thromboembolic complications are common with COVID-19. In patients critically ill with COVID-19, the incidence of thrombotic events is 20-30% in studies from the Netherlands, Italy, and France, despite prophylactic anticoagulant therapy [9, 23].

For example, in an updated analysis by F.A. Klok, et al. from 2020 [9] dedicated to confirming the high cumulative incidence of thrombotic complications in patients with COVID-19 in critical condition in the intensive care unit of three Dutch hospitals, along with updating the database, all analyses were re-examined. The incidence of the combined outcome of symptomatic acute pulmonary embolism, deep vein thrombosis, ischemic stroke, myocardial infarction and/or systemic arterial embolism was re-evaluated in all COVID-19 patients admitted to the intensive care units of 2 Dutch university hospitals and 1 Dutch teaching hospital from the time of admission to the intensive care unit until death, discharge from the intensive care unit or on 22 April 2020, whichever comes first. A repeat study of the same 184 patients in the intensive care unit, of whom a total of 41 patients died (22%) and 78 were discharged alive (43%). The median duration of follow-up increased from 7 to 14 days. All patients underwent pharmacological thromboprophylaxis. The cumulative incidence of the combined outcome, adjusted for the competing risk of death, was 49%. The majority of thrombotic events were pulmonary embolism (87%). In a competing risk model, chronic anticoagulant therapy at admission was associated with a lower risk of a combined outcome. Patients who were diagnosed with thrombotic complications had a higher risk of death from all causes. The use of therapeutic anticoagulant therapy was not associated with all-cause mortality. In this updated analysis, the authors confirmed a very high cumulative incidence of thrombotic complications in critically ill COVID-19 patients.

The same results were obtained in the assessment of venous and arterial thromboembolic complications in patients with COVID-19 hospitalized at the academic hospital in Milan, Italy [28]. C. Lodigiani, et al. studied consecutive symptomatic patients with laboratory-confirmed COVID-19 admitted to the University Hospital of Milan, Italy (13.02.2020-10.04.2020). The primary out-

come was any thromboembolic complication, including venous thromboembolism, ischemic stroke, and acute coronary syndrome/myocardial infarction. The secondary outcome was open-label disseminated intravascular coagulation. The study included 388 patients (mean age 66 years, 68% men, 16% requiring intensive care). Thromboprophylaxis was used in 100% of patients in the intensive care unit and in 75% of patients in the general ward. Thromboembolic events occurred in 28 (21%). Half of the thromboembolic complications were diagnosed within 24 hours of hospitalization. Forty-four patients underwent imaging studies for venous thromboembolism, and it was confirmed in 16 (36%). Computed tomography of the pulmonary artery was performed in 30 patients, which corresponds to 7.7% of the total, pulmonary embolism was confirmed in 10 (33%). The incidence of ischemic stroke and acute coronary syndrome/myocardial infarction was 2.5% and 1.1%, respectively. Overt disseminated intravascular coagulation was observed in 8 (2.2%) patients. The high number of arterial and, in particular, venous thromboembolic events diagnosed within 24 hours of hospitalization and the high rate of positive results of venous thromboembolism imaging tests among the few COVID-19 patients tested suggest that there is an urgent need to improve specific diagnostic strategies for venous thromboembolism and to investigate the efficacy and safety of thromboprophylaxis in outpatients with COVID-19.

The high incidence of thromboembolism in patients with COVID-19 indicates a high probability of the participation of this pathological process in the mechanisms of kidney damage. Such conclusions were obtained by independent studies of autopsies of patients who died in China [24] and in the United States [17].

In studies of autopsy material of patients who died from COVID-19, a group of scientists led by Amy V. Rapkiewicz, et al. [17] proved that megakaryocytes and platelet-fibrin thrombosis characterize multi-organ thrombosis. This study was conducted due to the fact that a prothrombotic condition in COVID-19 is increasingly recognized. At the same time, the results of pathological autopsies can be the most reliable regarding the mechanisms of pathogenesis. They presented a series of COVID-19 autopsies, including the results of studies of lungs, heart, kidneys, liver and bones, from the New York Academic Medical Center. In seven patients (four women), regardless of anticoagulant status, autopsies showed platelet-rich thrombi in the microvasculature of the lungs, liver, kidneys and heart. Megakaryocytes were found in larger numbers than normal in the lungs and heart. In two cases, thrombi were found in the large pulmonary arteries. Blood clots were not found in the inferior vena cava, but deep veins of the legs were not opened. In two cases, cardiac venous thrombosis was developed, and in one case, septal myocardial infarction associated with thrombosis of the intramyocardial veins, without atherosclerosis, was detected. In one case, focal acute inflammation with a predominance of lymphocytes was observed in the myocardium, while no virions were found in cardiomyocytes. Otherwise, histopathological changes in the heart were limited to minimal epicardial inflammation (n=1), early ischemic injury (n=3), and mural fibrin thrombus (n=2). Platelet-rich pericanular fibrin microthrombi were a characteristic feature of the kidneys. Acute tubular necrosis, red blood cells, and granular casts have been observed in many cases. There was no significant glomerular pathology. Numerous platelet-fibrin microthroma were found in the hepatic sinusoids. Diffuse alveolar damage with a spectrum of exudative and proliferative phases, including hyaline membranes, and pneumocyte hyperplasia with viral inclusions in epithelial cells and macrophages were found in all lungs. In three cases, superimposed acute bronchopneumonia with focal necrosis was observed. In this series of seven COVID-19 autopsies, thrombosis was a prominent sign of multiple organs, in some cases despite complete anticoagulant therapy and regardless of the timing of the disease, suggesting that thrombosis plays a role very early in the disease process. The detection of megakaryocytes and platelet-rich blood clots in the lungs, heart, and kidneys indicates their role in thrombosis.

P. Kathrine, et al. [7] systematized a review of the efficacy and safety of anticoagulants in common chronic kidney disease. He suggested that patients with chronic renal failure have an increased risk of developing venous thromboembolism and atrial fibrillation. Anticoagulants have not been studied in randomized controlled trials with a CrCl of < 30 ml/min. A systematic review of randomized controlled trials and cohort studies was conducted, searching electronic databases from 1946 to 2022. The study included cases in which both thrombotic and bleeding were assessed with anticoagulants at CrCl < 50 ml/min. Initial searches were conducted on 14,503 articles, of which 53 were eligible for inclusion in the system. Direct oral anticoagulants were compared with warfarin in patients with venous thromboembolism and a CrCl of 30-50 ml/min. Observational data from haemodialysis showed a lower risk of recurrence of venous thromboembolism and major bleeding with apixa-

ban compared to warfarin. There are very few studies that have investigated the outcomes of low-molecular-weight heparin at the therapeutic and prophylactic dose for CrCl<30 mL/min. Outcomes for patients with atrial fibrillation on dialysis were that warfarin or direct oral anticoagulants had the same or higher risk of stroke compared with no anticoagulant therapy. For patients with atrial fibrillation and CrCl<30 mL/min who are not on dialysis, anticoagulant therapy should be considered on a case-by-case basis, with limited studies suggesting that direct oral anticoagulants may have a preferred safety profile. In conclusion, the authors noted that further studies are needed in patients with advanced chronic renal failure (CrCl<30 mL/min) to determine the safest and most effective treatment options for venous thromboembolism and atrial fibrillation.

Along with this, most of the causes of acute renal failure are associated with a wide range of pathogenetic factors, which sometimes have an absolutely opposite vector of direction. Meanwhile, the mechanism of combination of these factors contributes to the aggravation of the course of the disease. This can include the consequences of severe inflammatory reactions of the body, disorders in the hemodynamic system, hypoxia, and the toxic effect of infection. What unites all the above factors is their non-specificity. Such an impact is also possible with the development of COVID-19. This may include the use of mechanical ventilation, systemic hypoxia, hypotension, low cardiac output, and the use of drugs with nephrotoxicity. All of them can act as factors that contribute to damage to kidney tissue and lead to functional disorders of this organ [5, 21].

In deceased patients with COVID-19, pathomorphological changes in the kidneys are characterized by the development of necrobiotic processes in the tubules of the kidneys. Such a pathological process is called tubular necrosis.

A group of clinicians from the Academic Hospital of New Orleans (USA) led by M.B. Mohamed Muner, et al. [3] suggested a close relationship between the pathogenesis of acute renal failure and the severity of COVID-19. They conducted an observational study in patients hospitalized at Ochsner Medical Center for 1 month with COVID-19 and diagnosed with acute renal failure. They examined in-hospital mortality as outcome measures. Among 575 hospitalizations with COVID-19, the authors found 161 (28%) cases of acute renal failure (61% in the intensive care unit and 14% in the general ward). The patients were predominantly men (62%). In 83% of cases, patients suffered from chronic diseases of the cardiovascular system. The median body mass index was higher in patients with acute renal failure. Acute renal failure compared to pre-existing chronic renal failure occurred in 35%. In-hospital mortality for the cohort of patients with acute renal failure was 50%. Vasopressors and/or mechanical ventilation were required in 105 (65%) patients with acute renal failure. Hemodialysis was required in 89 (55%) patients. Patients with acute renal failure requiring haemodialysis had a higher median body mass index and younger age. Baseline values of ferritin, C-reactive protein, procalcitonin, and lactate dehydrogenase were higher in patients with acute renal failure; And among them, the values were higher in patients who needed to use hemodialysis. Ischemic acute renal tubular injury and rhabdomyolysis account for 66% and 7% of the causes, respectively. 13% had no obvious causes of acute kidney failure other than a diagnosis of COVID-19. Thus, the authors concluded that acute renal failure in patients with COVID-19 is associated with high hemodialysis and mortality requirements. Higher levels of body mass index and inflammatory markers are associated with acute renal failure, as well as the need for haemodialysis. Hemodynamic instability leading to ischemic renal injury is the predominant cause of acute renal failure in these conditions.

Thus, in the terminal phases of acute renal failure, histological studies confirm the presence of acute tubular injury as a result of the development of focal tubular necrosis [12, 22, 24].

The mechanism of the relationship between viral presence and immune system disorders in patients with death from COVID-19 was investigated by B. Schurink et al. [29]. In particular, it has been suggested that SARS-CoV-2 affects multiple organs and causes severe coagulopathy. Histopathological organ changes may be due not only to direct exposure caused by the virus, but also to the immune response. In this regard, the goal was to assess the duration of the presence of the virus, determine the degree of inflammatory response and study the main causes of coagulopathy. A prospective autopsy cohort study was conducted at the Medical Centers of the University of Amsterdam (the Netherlands). In the period from March 9 to May 18, 2020, an autopsy of 21 corpses of patients who died from COVID-19 was performed. In addition to histopathological assessment of organ damage, the presence of SARS-CoV-2 nucleocapsid protein and the composition of immune infiltrate and blood clots were also assessed, all of which were associated with the course of the disease. Our cohort included 16 (76%) men with an average age of 68 years. In 11

patients tested for SARS-CoV-2 tropism, infected viral cells were present in many organs, mainly in the lungs, but the presence in the lungs became sporadic with increasing disease progression. Other SARS-CoV-2-positive organs included the heart, kidneys, and gastrointestinal tract. On histological analysis of the organs, an extensive inflammatory reaction was present in the kidneys. In the brain, extensive inflammation was observed in the olfactory bulbs and medulla oblongata. Blood clots and neutrophil plugs were present in the lungs, heart, kidneys, liver, spleen and brain and were most often seen in the late stages of the disease. Neutrophil plugs were observed in two forms: exclusively consisting of neutrophils with neutrophil extracellular traps or in the form of their aggregates and platelets.

Thus, the researchers concluded that patients who died from COVID-19 had an extensive systemic inflammatory response with the persistent presence of neutrophils and neutrophil extracellular traps. However, in the late stages of COVID-19, cells infected with SARS-CoV-2 were only sporadically present. This is indicative of a maladaptive immune response and supports evidence of immunomodulation as a target in the treatment of severe COVID-19.

A multicenter clinical and pathological comparison of kidney biopsies of patients with COVID-19 complicated by acute renal failure or proteinuria was carried out by a group of American scientists led by S. Akilesh, et al. [18]. As a justification for the need for this study, the authors suggested that kidney biopsy data can inform doctors about the pathological processes associated with SARS-CoV-2 infection. They conducted a multicenter evaluation of kidney biopsy results in living patients to identify various signs of kidney disease pathology in patients with COVID-19 and their association with SARS-CoV-2 infection. A total of 14 native and 3 causal kidney transplant biopsies were identified in patients with documented recent or concomitant SARS-CoV-2 infection treated at 7 major hospital systems in the United States. Men and women were equally represented in this case series, with a higher proportion of black and Hispanic patients. In all 17 patients, SARS-CoV-2 infection was confirmed by reverse transcriptase polymerase chain reaction, but only 3 patients had severe symptoms of COVID-19. Acute kidney injury and proteinuria were the most common indications for biopsy, and these symptoms developed simultaneously or within 1 week of the onset of COVID-19 symptoms in all patients. The most common histological signs were acute tubular injury, collapse-glomerulopathy, and endothelial damage/thrombotic microangiopathy. 2 of the 3 transplant recipients developed active antibody-mediated rejection a few weeks after COVID-19. 8 patients required haemodialysis, but others improved with conservative treatment. Cases of even symptomatic mild COVID-19 were accompanied by acute kidney injury and/or severe proteinuria, which was the reason for a diagnostic kidney biopsy. Although most of them had acute tubular damage, rare pathologies such as collapsed glomerulopathy and acute endothelial injury were found, and most of these patients progressed to irreversible kidney damage and dialysis.

The spectrum of renal biopsies in hospitalized patients with COVID-19, acute kidney injury, and/or proteinuria was determined by French clinicians S. Ferlicot, M. Jamme, F. Gaillard, et al. [26]. They presented a multicenter retrospective case series of COVID-19 patients who developed acute kidney injury and/or proteinuria and underwent kidney biopsies in Paris and its metropolitan area. The study included 47 patients (80.9% males) with COVID-19 who underwent kidney biopsy between March 8 and May 19, 2020. The median age was 63 years. Concomitant diseases: arterial hypertension (66.0%), diabetes mellitus (27.7%), obesity (27.7%), chronic kidney (25.5%), heart (38.6%) and respiratory tract diseases in anamnesis. Initial symptoms were fever (85.1%), cough (63.8%), shortness of breath (55.3%), and diarrhea (23.4%). Almost all patients developed acute kidney injury (97.9%) and 63.8% required renal replacement therapy. Renal biopsy showed two major histopathological patterns, including acute tubular injury in 20 (42.6%) patients and glomerular injury consisting of collapsing glomerulopathy and focal segmental glomerulosclerosis in 17 (36.2%) patients. Two (4.3%) patients had acute vascular nephropathy, and eight (17%) had an alternative diagnosis, most likely not related to COVID-19. Acute tubular injury almost invariably occurred in the setting of severe forms of COVID-19, while patients with glomerular injury had different COVID-19 severity profiles, and collapsing glomerulopathy was observed only in patients with a combination of APOL1 risk variants. At the last follow-up, 16 of the 30 patients who initially required dialysis were still on dialysis and 9 had died. While acute tubular injury correlates with the severity of COVID-19, the pattern of glomerular damage is closely related to the expression of APOL1 risk variants.

Thus, there is now a significant body of evidence suggesting that SARS-CoV-2 can affect kidney tissue. However, the direct role of the virus in the development

of acute renal failure and its consequences has not been conclusively confirmed.

Conflict of interest – no

Funding – not provided

Ethical aspects – complied with

REFERENCES:

1. A multi-centre retrospective cohort study defines the spectrum of kidney pathology in Coronavirus 2019 Disease (COVID-19). / R.M. May, C. Cassol, A. Hannoudi, et al. // Kidney Int. 2021 Dec;100(6):1303-1315.
2. Abnormal coagulation parameters are associated with poor prognosis in patients with novel coronavirus pneumonia / N. Tang, D. Li, X. Wang, et al. // Journal of thrombosis and haemostasis: JTH. – 2020. - Vol. 18 - No4 – P. 844-847.
3. Acute kidney injury associated with coronavirus disease 2019 in urban New Orleans / M.M.B. Mohamed, I. Lukitsch, A.E. Torres-Ortiz, et al. // Kidney 360. – 2020. – Vol. 1 - No7 – P. 614–622.
4. Acute kidney injury in 3182 patients admitted with COVID-19: a single-center, retrospective, case-control study / F.L. Procaccini, R. Alcázar Arroyo, M. Albalate Ramón, et al. // Clinical Kidney journal. – 2021. – Vol. 14 - No6 – P. 1557-1569.
5. Acute Kidney Injury Secondary to Rhabdomyolysis and COVID- 19: A Case Report and Literature Review / V.K. Chetram, A.I. Ahmad, S. Farid, et al. // Case reports in nephrology. – 2021. – P. 5528461.
6. AKI and Collapsing Glomerulopathy Associated with COVID-19 and APOL1 High-Risk Genotype / H. Wu, C.P. Larsen, C.F. Hernandez-Arroyo, et.al. // Journal of the American Society of Nephrology: JASN. – 2020. – Vol. 31 - No8 – P. 1688-1695.
7. Chronic anticoagulation is not associated with a reduced risk of acute kidney injury in hospitalised COVID-19 patients / K. Parker, P. Hamilton, P. Hanumapura, et al. // BMC Nephrology. – 2021. – Vol. 22 - No1 – P. 224.
8. Community- and Hospital-Acquired Acute Kidney Injury in COVID-19: Different Phenotypes and Dismal Prognosis / A.J. Martínez-Rueda, R.D. Álvarez, R.A. Méndez-Pérez, et al. // Blood purification. – 2021. – Vol. 50 - No6 – P. 931-941.
9. Confirmation of the high cumulative incidence of thrombotic complications in critically ill ICU patients with COVID-19 / F.A. Klok, M.J.H.A. Kruip, N.J.M. van der Meer, et al. // Thrombosis Research. – 2020. – Vol. 191 – P. 148–150.
10. D-dimer is associated with severity of coronavirus disease 2019 (COVID-19): a pooled analysis / G. Lippi, E.J. Favaloro // Thrombosis and haemostasis. – 2020. - Vol. 120 - No5 – P. 876-878.
11. Electron microscopic investigations in COVID-19: not all crowns are coronas / C. Roufosse, E. Curtis, L. Moran, et al. // Kidney International. – 2020. – Vol. 98 - No2 – P. 505-506.
12. Histopathologic and Ultrastructural Findings in Postmortem Kidney Biopsy Material in 12 Patients with AKI and COVID-19 / P. Golmai, C.P. Larsen, M.V. DeVita et al. // Journal of the American Society of Nephrology: JASN. – 2020. – Vol. 31 - No9 – P. 1944-1947.
13. Identifying a potential mechanism of acute kidney injury during the Covid-19 outbreak: a study based on single-cell transcriptome analysis / X.W. Pan, D. Xu, H. Zhang, et al. // Intensive Care Medicine. – 2020. – Vol. 46 - No6 – P. 1114–1116.
14. Incidence, risk factors and outcome of acute kidney injury (AKI) in patients with COVID-19 / G. Alfano, A. Ferrari, F. Fontana, et al. // Clinical and experimental nephrology. – 2021. - Vol. 25 - No11 – P. 1203-1214.
15. Isolation of infectious SARS-CoV-2 from urine of a COVID-19 patient / J. Sun, A. Zhu, H. Li, et al. // Emerging microbes & infections. – 2020. - Vol. 9 - No1 – P. 991- 993.
16. Mayerhöfer T., Perschinka F., Joannidis M. Akute Nierenschädigung und COVID-19: pulmorenaler Crosstalk unter massiver Inflammation [Acute kidney injury and COVID-19: lung-kidney crosstalk during severe inflammation]. // Med. Klin. Intensivmed Notfmed. 2022 Jun;117(5):342-348. (in German).
17. Megakaryocytes and platelet-fibrin thrombi characterize multi-organ thrombosis at autopsy in COVID-19: a case series / A.V. Rapkiewicz, X. Mai, S.E. Carsons, et al. // EClinicalMedicine. - 2020. – Vol. 24 – P. 100434.
18. Multicenter Clinicopathologic Correlation of Kidney Biopsies Performed in COVID-19 Patients Presenting with Acute Kidney Injury or Proteinuria. / S. Akilesh, C.C. Nast, M. Yamashita, et al. // Am. J. Kidney Dis. 2021 Jan;77(1):82-93.e1.
19. Multiorgan and Renal Tropism of SARS-CoV-2 / V.G. Puelles, M. Lütgehetmann, M.T. Lindenmeyer, et al. // New England Journal of Medicine. – 2020. - Vol. 383 - No6 – P. 590-592.
20. Native and transplant kidney histopathological manifestations associated with COVID-19 infection: A

systematic review / V. Jeyalan, J. Storrar, H.H.L. Wu, et al. // World Journal of transplantation. – 2021. -Vol. 11 - No11 – P. 480-502.

21.Pathophysiology of COVID-19-associated acute kidney injury / M. Legrand, S. Bell, L. Forni, et al. // Nature Review Nephrology. – 2021. – Vol. 17 - No11 – P. 751-764.

22.Postmortem Kidney Pathology Findings in Patients with COVID-19 / D. Santoriello, P. Khairallah, A.S. Bomback, et al. // Journal of the American Society of Nephrology: JASN. – 2020. – Vol. 31 - No9 – P. 2158-2167.

23.Pulmonary embolism in COVID-19 patients: awareness of an increased prevalence / J. Poissy, J. Goutay, M. Caplan, et al. // Circulation. – 2020. – Vol. 142 - No2 – P. 184–186.

24.Renal histopathological analysis of 26 postmortem findings of patients with COVID-19 in China / H. Su, M. Yang, C. Wan, et al. // Kidney International. – 2020. - Vol. 98 - No1 – P. 219-227.

25.SARS-CoV-2 renal tropism associates with acute kidney injury / F. Braun, M. Lütgehetmann, S. Pfefferle, et al. // Lancet. – 2020. – Vol. 396 - No10251 – P. 597- 598.

26.The spectrum of kidney biopsies in hospitalized patients with COVID-19, acute kidney injury, and/or proteinuria / S. Ferlicot, M. Jamme, F. Gaillard, et al. // Nephrology, dialysis, transplantation: official publication of the European Dialysis and Transplant Association - European Renal Association. – 2021. – Vol. 36. - No7 – P. 1253-1262.

27.Ultrastructural Evidence for Direct Renal Infection with SARS- CoV-2 / E.A. Farkash, A.M. Wilson, J.M. Jentzen // Journal of the American Society of Nephrology: JASN. – 2020. – Vol. 31 - No8 – P. 1683-1687.

28.Venous and arterial thromboembolic complications in COVID-19 patients admitted to an academic hospital in Milan, Italy / C. Lodigiani, G. Iapichino, L. Carenzo, et al. // Thrombosis Research. – 2020. – Vol. 191 – P. 9–14.

29.Viral presence and immunopathology in patients with lethal COVID-19: a prospective autopsy cohort study. / B. Schurink, E. Roos, T. Radonic, et al. // Lancet Microbe. 2020 Nov;1(7):e290-e299.

COVID-19 bilan og'rigan bemorlarda buyrak zararlanishi patogenezi

Xursandov I.A.
"Sulton shifoxonasi" Xususiy klinikasi

ABSTRAKT

SARS-CoV-2 pandemiyasi 2019 yil dekabr oyida boshlanganidan beri dunyoni larzaga soldi. Jahon sog'liqni saqlash tashkiloti 2021 yil noyabr holatiga ko'ra, 26,1 milliondan ortiq holat va 5200000 o'lim holati haqida xabar bergan va kasallikning ta'siri bilan bog'liq epizodlar tobora ko'payib borayotgani sababli bu raqamlar ko'payishda davom etmoqda. Jons Xopkins universiteti tomonidan taqdim etilgan mamlakat bo'yicha o'lim darajasi 0.1% dan 9.2% gacha, geografik farqlar bilan farq qiladi. Nafas olish tizimi va yurak-qon tomir tizimining shikastlanishi bilan bir qatorda, o'tkir va surunkali buyrak etishmovchiligi rivojlanishi bilan ajralib turadigan buyraklarning patologiyalari muhim rol o'ynaydi. Nafaqat sun'iy shamollatish, balki gemodializga muhtoj bemorlarni reanimatsiya bo'limlarida kasalxonaga yotqizish zarurati bilan davolashda ham qiyinchiliklar paydo bo'ldi. Sog‘liqni saqlash tizimida iqtisodiy muammolarning yuzaga kelib ilishining sababi shu edi. Ushbu maqola COVID-19 bilan og'rishgan bemorlarda buyrak shikastlanishi patogeneziga bag'ishlangan.

Kalit so'zlar: COVID-19, o'tkir buyrak etishmovchiligi, patogenezi