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FEATURES OF THE COURSE OF COVID-19 IN PATIENTS WITH ACUTE KIDNEY INJURY

Sidikova Maryam Amangeldiyevna
Samarkand State Medical University, Republic of Uzbekistan, Samarkand.

Resume. ***Introduction***. In the modern era, COVID-19 is the biggest challenge facing doctors and scientists round the world. SARS-CoV-2 is a multisystem infection that is not limited to lung damage and has an mmuno-mediated effect of negative effects on organs and systems, including the kidneys. To date, there is no precise understanding of the pathogenesis of nephrological disorders in patients with COVID-19. atients with chronic disease kidney diseases (CKD) are a group of particularly high risk of COVID-19 infection and high mortality in the development of the disease. ***OBJECTIVE***: to evaluate the features of the course of the new coronavirus infection (COVID-19) in patients with acute kidney injury and terminal renal insufficiency. ***PATIENTS AND METHODS.*** Clinical, laboratory, and instrumental parameters were studied in 119 patients (67 men and 52 women) diagnosed with COVID-19. The average age of the patients was 63.1±1.7 years. All patients were divided into two groups: group 1 – patients with CKD and HD, Group 2 – patients with newly diagnosed kidney damage due to coronavirus infection (COVID-19). Statistical data analysis was performed using the IBM SPSS Statistics 21.0 software package (USA) (Russified version). results. As a result of the study, it was found that in the clinical picture of patients with COVID-19, suffering from CKD and undergoing hemodialysis, was 2 times more likely to have a symptom such as myalgia, was the percentage of arterial blood hemoglobin saturation with oxygen (SaO2,%) is significantly lower compared with patients with newly diagnosed kidney damage on the background of infection. The duration of the temperature reaction during the disease was 5 times longer than in patients without CKD. Although the incidence of lung damage in patients of both groups was identical, mortality was significantly higher in the group of patients with CKD. ***CONCLUSION***. In the patients we examined, proteinuria, an increase in the level of nitrogenous metabolites, as well as D-dimers in both groups were associated with increased mortality. Mortality in patients with CKD and HD was several times higher than in those without pathology of the urinary system. The severity of the patients' condition was primarily due to the symptoms of damage to the respiratory system, but the degree of renal dysfunction is undoubtedly an important prognostic value. Thus, monitoring the condition of individual nephron structures in patients with COVID-19 is of great importance, and emergency nephroprotective measures may be crucial in combating the cytokine storm.

Keywords: coronavirus infection, COVID-19, chronic kidney disease, glomerular filtration rate, creatinine, course features

Introduction: Patients with CKD are a group of particularly high risk of COVID-19 infection and high mortality in the development of the disease. This is due to the fact that the cause of CKD in older age groups is often the main population diseases (diabetes mellitus, hypertension, obesity, atherosclerosis), which contributes to high morbidity and mortality from COVID-19 [1]. Among patients with CKD, the greatest difficulties are caused by patients receiving dialysis treatment. Isolation is impossible for them, among them a significant proportion of weakened patients with a high Charleson index, as well as people over 60 years of age [2]. According to world data, 59% of patients with a confirmed diagnosis of COVID-19 had changes in urine tests in the form of erythrocyturia and proteinuria, which indicated kidney damage even in those who had not previously suffered from diseases of the urinary system [3]. How exactly does SARS-CoV-2 damage the kidneys? The exact answer to this question has not yet been found. New information is emerging daily about the effects of covid infection on various organs and structures of the body, however, unambiguous there are no conclusions about how the kidneys are affected. To date, there are several theories that explain the pathogenic effects of the virus.

It is already known that SARS-CoV-2 is tropic to cells having angiotensin converting enzyme (APF2) receptors. The kidneys have a large amount of APF2 - receptors and may be a direct target for coronavirus. This fact is confirmed by studies when, during electron microscopy of the kidney tissue of patients who died from coronavirus, viral particles were detected, indicating a direct damaging effect [4, 5]. In any infection, regardless of the nature of the pathogen, activation of the systemic inflammatory response. COVID-19 is often accompanied by an overreaction on the part of the immune system, in which a systemic hyperinflammatory response ("cytokine storm") develops, leading to kidney damage in addition to direct exposure to the virus [6, 7]. Due to the peculiarities of penetration into the body, SARS-CoV-2 primarily affects the lungs, which leads to the development of respiratory failure. Hence, the third component of damage to the renal parenchyma is hypoxia. Finally, a distinctive feature of this variant of coronavirus is the rapid activation of hemostasis with the development of thrombovaculitis in small-caliber vessels of vital organs [8]. COVID-19 can be considered as a virus-mediated NET-oz model, which characterizes the close relationship of biological processes such as inflammation and thrombosis. NET-oz is understood as a specific variant of the programmed cell death of neutrophils. In contrast to apoptosis, neutrophils secrete so-called extracellular neutrophil traps (Neutrophil extracellular traps, NET), which can play an important role in the development of immunothrombosis. Separate series of papers have been published in which patients with COVID-19 and massive thrombosis showed an increase in antibody titers to phospholipids. This provokes pronounced endothelial dysfunction

and induction of platelet aggregation (the endothelium also carries APF2 receptors and is a target for the SARS-CoV-2 virus) [9]. The blockade of microcirculation and its irreversible nature often determine the outcome of the disease. And, in addition, it should not be forgotten that the methods of treatment of severe COVID-19 themselves can increase the risk of kidney damage formation by both prerenal and renal mechanisms. In this regard, it is possible to mention the difficulties of adequately assessing the water status of patients with an extremely high workload on medical personnel. In addition, some drugs that are used in the treatment of coronavirus infection are nephrotoxic [10, 11]. Thus, the issue of the effect of COVID-19 on the structure and function of the kidneys is currently multicomponent and requires a more in-depth and detailed study.

PATIENTS AND METHODS. The study included 119 patients diagnosed with COVID-19. Among them, men made up 67 people, women – 52 people. The average age of the patients was 63.1±1.7 years. The study was conducted on the basis of the departments of polyclinic therapy and infectious diseases with a course of tuberculosis Stavropol State Medical University in GBUZ IC "City Clinical Hospital No. 2", "Regional Clinical Infectious Diseases Hospital" and "Dialysis Center" in Kislovodsk. The duration of the study is 12 months (from March 2020 to March 2021). All patients underwent general analysis studies blood, urine, biochemical analyses (creatinine, urea, glucose, CRP, D-dimer, procalcitonin, fibrinogen), the calculated glomerular filtration rate (eGFR) according to the formulas CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration), performed computed tomography of the lungs. Blood was taken from the cubital vein in the morning on an empty stomach. The concentration of creatinine, urea and glucose was determined by colorimetric method on an automatic biochemical analyzer from Mindray BS-380 (China). All patients were divided into 2 groups: 39 patients – 16 women and 23 men, average age 63±1.9 years with a previously established diagnosis of CKD, who were on programmed hemodialysis (HD). The average hospital stay was 11.5±0.7 days. The 2nd group included 80 patients with newly diagnosed kidney damage on the background of current coronavirus infection (AKI). There were 44 men and 36 women, with an average age of 62.3±1.7 years. The duration of hospital stay was 10.9±0.7 days. Statistical data analysis was carried out using the package of applied statistical.

Results: The results of the analysis showed that the most common concomitant diseases in patients with SARSCoV-2 coronavirus infection in the group of patients with CKD were pathology of the cardiovascular system, hypertension, and less often diabetes mellitus (Fig. 1). In the group of patients who had not previously suffered from kidney disease, obesity, hypertension. Of the clinical manifestations dry cough was common in both groups: in the group of patients without CKD – in 60.4± 0.9% and with CKD – in 79.5±0.9% of cases. Such a symptom as myalgia was noted 2 times more often by patients of the HD group: 66 and 32.5 % accordingly, $p<0.05$. The

percentage of arterial hemoglobin oxygen saturation (SaO2, %) in the blood of patients with GL was lower: 86.4±0.14 and 91.1±0.7, respectively, $p<0.05$. During the study, an interesting fact was discovered: the duration of the temperature reaction is 38.5 ° C and higher in patients with AKI It was on average almost 5 times more (4.7±0.4 days) than in HD patients (1.1± 0.2 days). programs "SPSS Statistics 21.0" (USA) (Russified version). The results are presented as an arithmetic mean ± the error of the average. The correlation analysis was performed using Spearman's rank correlation coefficient. The statistical significance of the differences between the two The averages were determined using the Student's t-test; the frequencies were determined using Pearson's $\chi 2$ –test. The null statistical hypothesis of the absence of differences and connections was rejected at $p<0.05$. It was also noted that in all patients included in the study, the presence of viral pneumonia was confirmed during computed tomography (Fig. 2). The X-ray picture of CT2 (25-50% of the lungs were affected) and CT3 (50-75% of the lungs were affected) prevailed in the group of patients with AKI for the first time, although it was not statistically significant. The percentage of patients with minimal lung damage (CT 1, less than 25% of the lungs were affected) and maximum degree (CT4, more than 75% of the lungs were affected) in both groups was the same. However, mortality was significantly higher ($p<0.05$) in the HD group and accounted for more than 50% of all patients. It should be noted that all deceased patients received respiratory support on ventilators. There were no survivors. By conducting a comparative analysis of laboratory parameters in blood serum, we were able to establish that the main studied parameters (leukocytes, neutrophil-leukocyte index, ESR, CRP, fibrinogen, D-dimers, procalcitonin) were elevated in both study groups in the same degree, there were no significant differences. The neutrophil-leukocyte index in all patients in both groups was below 10, which means indicates the viral nature of the lesion. Creatinine levels in both groups did not depend on either saturation or severity of lung damage. It was noted that all patients with AKI and COVID-19 proteinuria was noted. A direct dependence of proteinuria on concentration is also shown D-dimers ($Rs=0.456$; $p<0.05$) and procalcitonin ($Rs=0.411$; $p<0.05$) in the blood of patients of both groups. The correlation of D-dimers and clinical outcome was direct and quite strong ($p=0.559$, $p <0.001$). The average creatinine level in patients of both groups differed: in the HD group – 561.3±49.1 mmol/l, in the AKI group – 211.2±40.3 mmol/l.

Discussion: COVID-19 is one of the biggest challenges facing doctors and scientists around the world in the modern era. SARS-CoV-2 is a multisystem infection. In patients with COVID-19, the most common concomitant diseases were: in the group of patients with CKD is diabetes mellitus, arterial hypertension and cardiological pathology, which is confirmed by previous studies, in patients with newly diagnosed kidney pathology – obesity and arterial hypertension. During the analysis, it turned out that with proteinuria, azotemia, and the level of D-dimers are associated with increased

mortality. In addition, mortality in patients with AKI and HD was several times higher than in those without pre-existing kidney disease. An interesting feature of the flow was noted in the study COVID-19 in the group of patients with CKD and HD we observed: the disease proceeded without typical symptoms – prolonged fever, cough and weakness, and the diagnosis was made based on computed tomography data. Lung damage by SARS-CoV-2 infection in the studied There were no significant differences in the groups. At the same time, mortality was significantly higher in the group of patients with HD, in whom the fatal outcome developed on average on the 9th day. A comparative retrospective analysis of the clinical, laboratory and instrumental data obtained during our study substantiates the main cause of death in patients with coronavirus infection as multiple organ failure. In our study , we confirmed previously available information that independent predictors of the severity of renal pathology, the development of AKI and fatal The outcome in patients infected with SARS-CoV-2 may be: old age, diabetes mellitus, hypertension, cardiovascular diseases, artificial lung ventilation, higher baseline serum creatinine levels.

Conclusion: The clinical manifestations of COVID-19 are mainly represented by symptoms of damage to the respiratory system, but neurological manifestations and/or complications should be given special attention. Their presence is associated with a high risk of severe disease and mortality.

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