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IMPAIRED RENAL FUNCTION IN COVID-19 INFECTION

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ABSTRACT

The SARS-CoV-2 virus (COVID-19) is highly contagious, virulent and shows tropism to many cells of human organs. To enter the cell, the SARS-CoV-2 virus uses the angiotensin converting enzyme type 2 (APV2) receptor, which is highly expressed in the kidneys. Through the APF2-dependent signaling pathway, SARS-CoV-2 can directly infect the epithelium of the renal tubules and podocytes.

Key words: SARS-CoV-2 virus, COVID-19, infection, kidney.

INTRODUCTION

Infection caused by coronavirus 2 (Severe acute respiratory syndrome coronavirus 2, SARS-CoV-2) is a systemic disease with predominant damage to the lungs, heart, kidneys, and nervous system. According to current observational data, acute kidney injury (AKI) is the 2nd most common cause of death in patients with SARS-CoV-2 after acute respiratory distress syndrome.

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Predisposing factors of kidney damage COVID-19 include chronic kidney disease (CKD), cardiovascular pathology, congestive heart failure and, as a consequence, the development of cardiorenal syndrome, the presence of immunodeficiency conditions, including those associated with the use of immunosuppressants and/or radiation therapy, hypovolemia, taking nephrotoxic drugs, etc. [1-5]. As is known, the most common causes of CKD are diabetes mellitus, hypertension, obesity, atherosclerosis, old age, which also belong to the

main risk factors for infection with SARS-CoV-2, in addition, their presence complicates the clinical course and worsens the outcomes of the disease.

Potential risk factors for acute kidney injury (AKI) in COVID-19 are presented in Table 1.

Patients with CKD are a group of high mortality with the development of COVID-19 infection. A particular risk of severe infection should be expected in patients with diabetic nephropathy, hypertensive nephropathy, in persons who have undergone kidney transplantation, who have been receiving glucocorticosteroids and immunosuppressants for a long time, including glomerular kidney diseases in patients on dialysis.

T. Oyelade et al. the development of fatal cases has been recorded in almost every second patient with COVID-19 and CKD [1]. According to Z.Shahid et al. in 48% of patients over 70 years of age with COVID-19 infection, concomitant pathology was presented with CKD, and the proportion of such patients it was almost twice as high compared to diabetes mellitus. The lethal outcome in patients with COVID-19 without concomitant diseases was 1.4%, while against the background of CKD - 13.2%, which was comparable with the group of patients with cardiovascular diseases [3]. Other studies have also revealed that CKD directly correlates with the severity of the clinical course of COVID-19 [4, 5].

In patients without previous kidney damage against the background of coronavirus infection, both mild renal dysfunction and CKD may develop. According to the International Society of Nephrology (ISN)) kidney damage is observed in severe COVID-19 in 25-50% of cases and is manifested by proteinuria and hematuria, acute kidney damage develops in about 15% of cases [6].

According to published data in the USA [7] and in Italy [8], AKI was observed in more than 20% of cases in patients in critical condition. In his observations, H. Rab noted the development of OP in 5% of hospitalized patients in the general cohort and in 50% of patients in the intensive care unit [9].

Table 1

Potential risk factors for acute kidney injury in COVID-19

Demographic risk factors	Risk factors for AKI upon admission	Risk factors for AKI during hospitalization
Old age	Severity of COVID-19	Nephrotoxins (drugs, rent gene contrast agents)
Diabetes mellitus	Degree of viremia	Vasopressors
Hypertension	Respiratory status	Ventilation, high positive pressure at the end of exhalation

Cardiovascular diseases or congestive heart failure	Damage to non-respiratory organs, for example, diarrhea	Fluid overload or hypo volemia
High body mass index	Leukocytosis, lymphopenia	
Chronic kidney disease	Elevated markers of inflammation (ferritin, C-reactive protein, D-dimers)	
Genetic risk factors (e.g. apolipoprotein 1 genotype; ACE 2 polymorphism)	Hypovolemia/ dehydration	
Immunosuppressive state	Rhabdomyolysis	
smoking	Exposure to drugs, for example, ACE inhibitors and/or angiotensin receptor blockers, statins, nonsteroidal anti-inflammatory drugs	

Goal. To evaluate the effect of coronavirus infection on renal dysfunction of those who brought and did not bring coronavirus infection in patients with chronic kidney disease (CKD).

Material and methods: 120 patients were examined in the Department of Nephrology at the TMA clinic. The patients were divided into two groups: group 1–a group of patients with CKD who had not had COVID 19 (n=64); group 2 - a group of patients with CKD who had had COVID 19 (n=56).

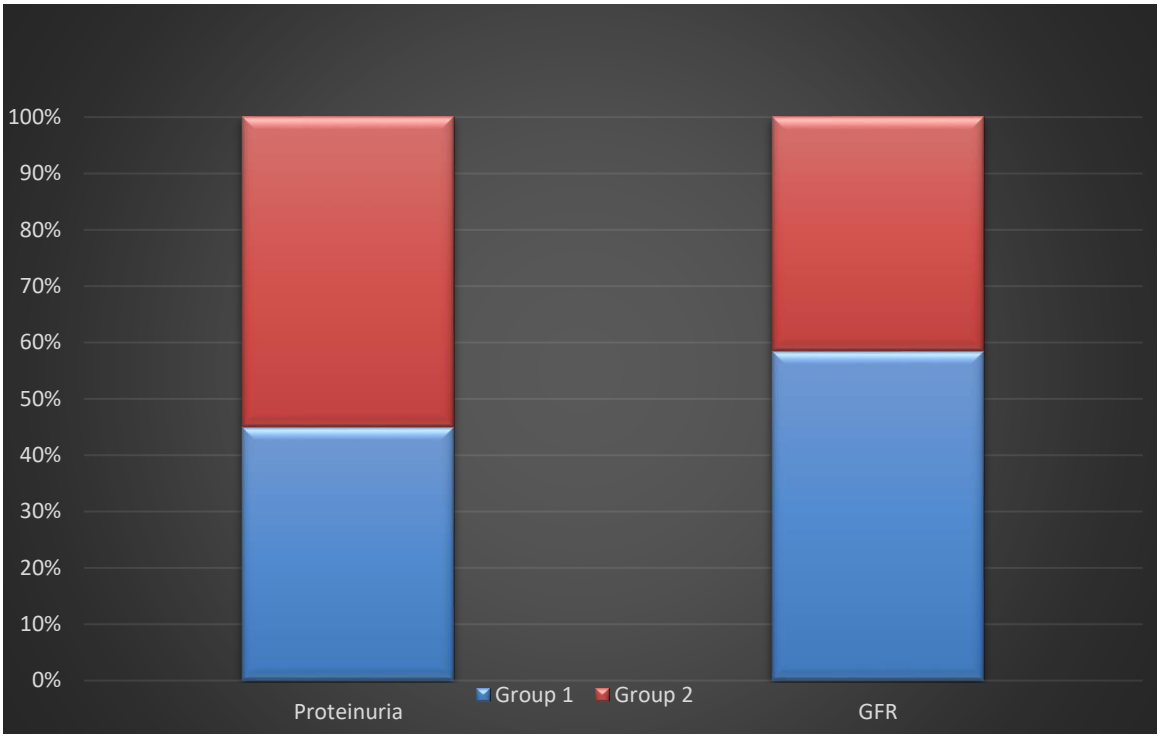
All patients underwent general clinical and laboratory -instrumental examination: included a general analysis of blood, urine, from biochemical analyses of the concentration of urea, creatinine and serum electrolytes, lipid spectrum. The glomerular filtration rate (GFR) was determined by the formula CKD-EPI (1.73 ml/min/m2), [NKF K/DOQI, 2002]. According to the results obtained, the stage of CKD was determined.

In the statistical processing of the results, the “STATISTICA 5.0" programs were used.

Results: In the study groups, the values of proteinuria, GFR were evaluated to assess renal dysfunction. When comparing the results of the first and second

groups, there was a significant decrease in kidney function in patients with COVID-19, respectively, proteinuria 0.9±0.12-1.1±0.34, and GFR 64.9±1.89-46.2±1.7 ($p<0.05$). An increase in protein in urine was observed with a decrease in GFR. It was found that there is a significant negative correlation between proteinuria and GFR.

A comparative study of the results from groups 1 and 2 showed that proteins in urine were significantly higher in group 2 than in group 1 and there was a positive correlation between them. GFR decreased significantly in group 2 compared to group 1, and the GFR index showed a significant negative correlation with blood hypertension (see Figure 1).



Note: Confidence between groups *-($p<0.05$

Figure 1. The ratio of proteinuria and GFR in patients in groups

The results obtained by us allow us to note that a higher level of proteinuria is accompanied by a decrease in the level of GFR in patients who have had a caronovirus infection than those who have not had a coronavirus infection. This will make it possible to predict and prevent the progression of CKD earlier.

Conclusion. In patients with SARS-CoV-2 infection, the prevalence of kidney damage is high and usually leads to a poor prognosis, which increases the importance of nephroprotection. According to the new data obtained, CKD or AKI, first diagnosed during hospitalization, should be recognized as risk factors for severe COVID-19. In case of kidney damage caused by SARS-CoV-2, general

principles of treatment are used - symptomatic and renal replacement therapy, and the administration of nephrotoxic drugs is monitored.

Further research is recommended to send to the study of risk factors for development of severe AKI with coronavirus infection, evaluation of new biomarkers for diagnosis and prognosis, to identify different phenotypes of AKI, the use of substitution renal replacement therapy in the early stage of the development of kidney injury, the impact of the virus SARS-CoV-2 on the formation of long-term kidney fibrosis and recovery, the definition of tactics and dispensary observation of patients.

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