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Brig SK Mazumdar Marg, Timarpur, New Delhi, Delhi 110054 India

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FEATURES OF THE ETIOLOGY AND MECHANISM OF KIDNEY DAMAGE IN PATIENTS WITH COVID-19

Safarova Gulnoz Avazkhonovna
Bukhara State Medical Institute

Abstract: Initially, COVID-19 was considered as a predominantly respiratory infection, the mortality from which is associated with the progression of respiratory failure [1]. Currently, COVID-19 is recognized as a multisystem disease with a wide range of manifestations [2].

Keywords: AKI, nephropathy, COVID-19, renal replacement therapy, etiology of nephropathy, pathogenesis of nephropathy, focal segmental glomerulosclerosis.

Relevance. One of the most common variants of the complicated course of COVID-19 is acute kidney injury (AKI). The frequency of AKI, according to various authors, varies widely and requires further study [3, 4, 5]. Data on the prevalence of severe AKI, including those with renal replacement therapy (RRT), among patients with COVID-19 in the Russian population are limited [6]. Because AKI increases patient care costs, epidemiological data are essential for healthcare planning [7].

The purpose of this study: To study the literature on the development of nephropathy in patients with COVID-19.

The etiology of AKI in patients with COVID-19 remains unclear. To clarify the reason for the high incidence of kidney damage, a large number of studies have been conducted on the potential direct cytopathic effect of the SARS-CoV-2 virus on the kidneys. According to researchers, the potential mechanism of direct viral exposure to the kidneys is realized through type 2 angiotensin-converting enzyme (ACE) receptors, which are located in the cells of the proximal tubules and podocytes, since the virus enters the cell in the lungs through type 2 ACE receptors [5] . The earliest evidence suggesting a likely direct viral infection of the kidneys in COVID-19 was provided by a study from Michigan, which demonstrated virus-like inclusions in tubular cells by electron microscopy in autopsy material from the kidneys of patients with COVID-19 [6]. Initially, these changes were interpreted by other researchers as extruded microvesicles not associated with the virus [7]. In the first study aimed at confirming the renal tropism of SARS-CoV-2, researchers from Germany detected viral ribonucleic acid (RNA) by in situ hybridization and PCR in all parts of the kidneys in an autopsy study, in the highest concentration in the glomeruli [13]. The study by Braun, F. for the first time correlated the tropism of SARS-CoV-2 to the kidneys and clinical outcomes [18]. In the above work, it was demonstrated that among those who developed AKI, renal messenger RNA of SARS-CoV-2 was detected in 72%, while in those without AKI only 43%. Furthermore, since the presence of messenger RNA of the virus in renal autopsy specimens suggests that the virus may invade the renal parenchyma, reports of a collapsing variant of focal segmental glomerulosclerosis (FSGS) in patients with COVID-19 also support the probable renal tropism of SARS-CoV-2. This was first shown in six patients with

confirmed COVID-19, AKI, and proteinuria who underwent kidney biopsy [19]. Histological examination of the kidneys showed collapsing glomerulopathy and tubular damage. Although viral particles were not detected in kidney tissue in this work, the appearance of FSGS in COVID-19 resembles podocytopathies associated with human immunodeficiency virus, parvovirus B-19, and cytomegalovirus, for which direct viral damage to the kidneys has been proven. In addition, SARS-CoV-2 particles have been detected in urine samples [10, 11].

A meta-analysis that included 288 histological examinations of the kidneys in patients with COVID-19 showed a wide variety of pathologies ranging from collapsing FSGS and acute tubular injury to vasculitis, thrombotic microangiopathy and pigmentary nephropathy [12]. At the same time, in another retrospective multicenter study, where clinical data were assessed and immunohistochemical examination of 273 kidney tissue samples was carried out, data on direct viral effects on the kidneys were not revealed [13]. In the same study, compared to a five-year database of biopsies, there was an increase in the incidence of myoglobin nephropathy and proliferative glomerulonephritis with monoclonal immunoglobulin (Ig G) deposits in patients with COVID-19 (3.3% and 1.7%, respectively).

Findings. In COVID-19 disease, the “collapsing” type is most common. The idiopathic "collapsing" variant of FSGS is characterized by a global or segmental "collapse" of the glomerular capillaries due to wrinkling and retraction of the 5MK as a result of primary obliteration of the lumen of the glomerular capillaries, signs of hypertrophy and hyperplasia of visceral cells. Tubulointerstitial damage is proportional to the degree of glomerulosclerosis. Isolation of this type is especially important because it is resistant to modern methods of therapy. That is why this problem requires further study.

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