

The Mechanism of Development of Kidney Complications in Children with a History of COVID-19

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Annotation: The article provides information about endothelial dysfunction and mechanisms of its development in patients during the coronavirus pandemic. The frequency of renal complications in children against the background of COVID-19 is described, which is associated with endothelial dysfunction. Studies on the frequency of urinary tract infections in children and adolescents during the pandemic are shown.

Keywords: COVID-19, SARS-CoV-2, glomerulonephritis, pyelonephritis, ACE2, children.

The coronavirus disease 2019 (COVID-19) has dramatically changed all aspects of human life worldwide, causing unprecedented morbidity and mortality [3]. Despite intensive efforts in vaccination programs, severe COVID-19 continues to pose a public health threat, and complications of the disease are only beginning to be reported [4].

In the early days of the COVID-19 pandemic, it was reported that clinical symptoms of the disease in children were milder than in adults [1]. However, according to a number of authors, there is evidence that serious systemic inflammatory reactions occur in young patients 2-4 weeks after acute infection caused by the SARS-CoV-2 virus [5, 6, 7]. Multisystem inflammatory syndrome in children resembles the symptoms of Kawasaki disease, in which autoreactive antibodies are produced on the mucosal surface during an acute immune response to a viral infection and accumulate around IgA-producing plasma cells [2]. The characteristics of immune response formation to previous infections may alter the response to SARS-CoV-2 and induce systemic inflammation [8]. COVID-19dagi og'ir yoki uzoq muddatli asosiy patofizyologik jarayon bu endotelial disfunktsiyadir [5].

Endothelium is a continuous monolayer of endothelial cells that forms the inner cell lining of arteries, veins, and capillaries and serves as a barrier between tissues and blood that has the functional capacity of an endocrine organ. The endothelium participates in a number of pathophysiological processes and dynamically interacts with blood components and other cells [7]. Under physiological conditions, it restores the integrity of blood vessels when they are damaged and participates in the coagulation process, preventing excessive thrombus formation [9]. It also participates in the accumulation, adhesion and interaction of platelets and leukocytes with thrombogenic surfaces. Under conditions of increased oxidative stress, the biosynthesis and availability of NO decreases, and the endothelium loses its protective properties, undergoes impaired vasodilation and a severe pro-inflammatory, proatherosclerotic and prothrombotic profile [8].

As another aspect of the pathophysiology of endothelial dysfunction in COVID-19, high expression of angiotensin-converting enzyme 2 (ACE2) is targeted to pericytes, leading to endothelial cell and microvascular dysfunction. Considering that ACE2 is highly expressed in podocytes and epithelial cells of renal tubules, the tropism of the coronavirus for kidneys and other organs of the urinary system is predicted [11].

Nephrotic syndrome has been described as the most common manifestation of urinary system pathology in children and adolescents infected with COVID-19 [11]. The mechanisms by which the infectious disease causes nephrotic syndrome discussed have been suggested in several studies in adults, but there are few comparable data in pediatric patients. SARS-CoV-2 virus particles have been found in post-mortem kidney biopsy specimens, indicating direct podocytopathic damage [12]. The report found evidence of tubuloreticular inclusions, often a sign of viral replication and significant interferon production in glomerular endothelial cells [13].

It is claimed that COVID-19 is mediated through multiple immunological pathways [12]. The results of basic scientific research in the early days of the pandemic show that tissue damage by the SARS-CoV-2 virus is determined by the appearance of a cytokine storm [15]. Podocytopathy is believed to be caused by a cytokine storm that triggers an immunological response with overproduction of Th2-derived cytokines.

The development of glomerulonephritis in children and adolescents after infection with COVID-19 has been reported [14]. Currently, the pathogenesis of this pathology of the urinary system is multifactorial and is believed to be related to direct viral attack on glomerular structures and the release of cytokines [17, 20].

Acute necrotizing glomerulonephritis and acute renal failure (ARF) secondary to vasculitis in acute COVID-19 infection are caused by glomerular hypoperfusion and tubular necrosis, which leads to fibrinoid necrosis in the arterial walls of glomerular and intrarenal vessels [21, 22].]Currently, there are no clear data on the frequency of acute renal failure in children with coronavirus disease [22]. In China, the incidence of AKI among children and adolescents hospitalized with COVID-19 was 1.3–1.8% [22 , 21].

E.C. Björnstad et al [22] conducted a multicenter study of confirmed COVID-19 children aged 1 month to 18 years hospitalized in the intensive care unit at 41 centers in 6 countries in North America and Europe (32 in the USA), and almost half (44%) of the 106 children had AKI. developed: 47% (n = 22) had stage 1, stage 2 - 23% (n = 11) and 30% (n = 14) of patients had stage 3.

In a patient cohort of 2,546 children with COVID-19 from North America, 10.8% of patients developed AKI, according to R. Raina et al., which K.R. Derespina et al reported that 12.9% of patients developed AKI in a study of intensive care unit patients in New York [27]. In contrast, studies from Italy and Spain reported a low incidence of AKI (1.2 and 0.8%, respectively) [33, 35].

J.A. Kari et al [29] conducted a multicenter retrospective cohort study including children with

confirmed COVID-19 at three tertiary centers.

In addition, the incidence of MIS-C was higher in children with acute renal failure than in children with normal renal function (15% vs. 1.5%). Patients with AKI were more likely to require intensive care unit admission (32 vs 2.8%) and had higher mortality (42 vs 0 %). Symptoms of renal failure persisted in 9% of patients, significantly influenced by associated processes such as hypotension, heart failure, acute respiratory failure, hypernatremia, abnormal liver enzymes, elevated C-reactive protein levels, and positive bacterial blood cultures. showed. The authors focus on the asymptomatic nature of acute renal failure in this group of patients. Oliguria and hypervolemia and no need for renal replacement therapy were found in 95% of those examined [29].

Given the documented association of AKI with high rates of morbidity and mortality, J.A. Kari et al [29] emphasize the importance of early diagnosis and prevention. Also, according to the authors, glomerular filtration rate (GFR) and creatinine level should be closely monitored in children infected with SARS-CoV-2, especially if they have co-morbidities (respiratory, cardiac, hematological diseases, renal diseases). they emphasize that it is necessary to go [29].

Currently, there are limited data on COVID-19 in children with chronic kidney disease [34, 35]. In a retrospective study, S. Krishnasamy et al [34] examined the presentation and outcome of SARS-CoV-2 infection in 88 children with chronic kidney disease at a single center in India. In 50% of patients, it was documented that the underlying disease included nephrotic syndrome. According to the stages of chronic kidney disease, this cohort was divided as follows: stage 1-4 - 18.2%, stage 5 - 17% and terminal - 14.8% of patients. 19.3% of those tested had a moderate to severe course of COVID-19.

The novel coronavirus disease has also posed a serious threat to the mental health of children and adolescents due to its direct impact and the unique combination of social isolation, economic recession and the transition of schools to distance learning [38]]. L. Harper et al. [39] believe that such psychosocial factors may influence the development of bladder and bowel dysfunction, which requires further research.

Conclusion. Summarizes information on endothelial dysfunction and mechanisms of its development during coronavirus infection, which is important not only for understanding the multisystem attack of COVID-19, but also for improving approaches to patient management. Considering the short-term and long-term adverse effects of COVID-19, further research is needed to translate the presented data into clinical practice.

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