15. PATHOPHYSIOLOGICAL MECHANISMS OF KIDNEY INVOLVEMENT IN COVID-19

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Introduction. Coronavirus disease 2019 (COVID-19) is a pandemic infection caused by the novel severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). Although the respiratory system is the major target, multiple organs, including the kidneys, can be affected. Kidney involvement is frequent, with clinical presentation ranging from abnormal proteinuria at hospital admission to progressive acute kidney injury (AKI). AKI is one of the most important complications in critically ill patients with COVID-19. The estimated incidence of acute kidney injury in patients with COVID-19 varies from 0.5% up to 45% depending on the severity of the disease. Mortality among hospitalised patients with acute kidney injury associated-COVID-19 is significantly higher than for those without kidney involvement.

Aim of study. Acute kidney injury is considered a marker of disease severity and negative prognostic factor for survival. In this context, we explored the potential pathways and pathophysiology of COVID-19 associated with AKI.

Methods and materials. The articles published during the years 2020-2022, were selected, using PubMed and Google Scholar database according to keywords: „COVID-19", „SARS-CoV-2", „Acute kidney injury", „Pathophysiology". 308 publications were found. Research includes data from 28 publications, analysed according to selection criteria.

Results. The etiology of renal impairment in patients with COVID-19 is multifactorial. Various mechanisms have been proposed for kidney injury in SARS-CoV-2 infection, both COVID-19-specific mechanisms, including direct invasion of the renal parenchyma with SARS-CoV-2 virus, the new coronavirus can exert direct cytopathic effects on kidney tissue and also non-specific mechanisms, such as: hemodynamic instability (hypovolemia or fluid overload), local and systemic immune and inflammatory responses, with macrophage activation and release of circulating proinflammatory cytokines (cytokine storm), hypoxia, sepsis, rhabdomyolysis, release of tissue factors and activation of coagulation pathways with the formation of microthrombi and alteration of the microcirculation. Other potential mechanisms are altered Renin-Angiotensin-Aldosterone regulation, organ interactions between lung, heart, and kidney and also therapeutic consequences (use of antibiotics and antiviral drugs with nephrotoxic potential, invasive mechanical ventilation). The most common histopathological findings are: acute tubular injury, thrombotic microangiopathy, endothelial injury and collapsing glomerulopathy.

Conclusion. This review highlights the importance of understanding the potential mechanisms of renal involvement in SARS-CoV-2 infection, for the early detection of renal injury and the avoidance of factors that contribute to progression of kidney injury, including adequate hemodynamic support and avoidance of nephrotoxic drugs, which will improve vital prognosis of COVID-19.