Novel Coronavirus(Covid19), Its Renal Manifestations, and Complications

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At the end of 2019, one of the most destructive viruses of all time was recongnized in Wuhan (Hubei, China). The virus has been named severe acute respiratory syndrome coronavirus 2(SARS-CoV-2) and belong to the family of Coronaviridae, a group of related RNA viruses that cause a variety of diseases in mammals and birds. Of the more than 100 known coronaviruses, 7 routinely infect only humans. Four of these cause mild to moderate disease and three [SARS-CoV, Middle East respiratory syndrome coronavirus(MERS-CoV) and SARS-Cov-2] can cause severe and even fatal complications. SARS-CoV-2 mainly affects the respiratory systems, although other organ systems such as urinary tract are also influenced. This feature that substantially increase the virus pathogenicity is partly associated with the mechanism of viral entry into cells. Infection is initiated by interaction of the viral structural protein(spike protein) and host cell receptors (angiotensin converting enzyme 2) that are expressed on target organs such as the lungs, heart and renal system.[1] Thus, this coronavirus can have a devastating effect on different organs.

Based on current published evidence, some patients with coronavirus disease 2019 (COVID-19) may experience serious kidney problems via acute kidney injury,[2] proteinuria, hematuria, the increased blood urea nitrogen and/or creatinine. Kidney damage may be caused directly by an infection from SARS-Cov-2, or indirectly by the body's war against infection.

Although the exact mechanism(s) underlying kidney involvement in COVID-19 has not been defined, evidence suggests that SARS-CoV-2 may result in direct injury to the cells of the renal tubules. This notion is further supported by the findings of other studies that show the accumulation of SARS-CoV-2 antigens in kidney tubules.[3]

In addition to the direct effect, SARS-CoV-2 may indirectly cause harm to the kidneys through uncontrolled production of cytokines, especially proinflammatory cytokines such as tumor necrosis factor alpha and interleukin 6.[4] This phenomenon, known as "cytokine storm", is evident during sepsis and can be caused by any type of infectious pathogen including SARS-CoV-2. There is evidence to show that sepsis is one of the main mechanisms of kidney damage in SARS-CoV-2 infected patients.[5] and the unregulated release of cytokines , seems to be a possible mechanism for such devastating conditions.

Two other scenarios by which an indirect effect can occur are 1) too little oxygen as a result of the pneumonia which is commonly seen in severe cases of the disease and 2) intrarenal vascular obstruction secondary to blood clot formation after virus infection.

Another aspect which has to be considered in relation to the COVID-19 is the higher risk of infection in patients with various types of kidney disease (including, patients undergoing maintenance hemodialysis or peritoneal dialysis). These patients are also more vulnerable to developing a severe form of COVID-19 illness.

Higher prevalence of comorbidities (including elevated blood pressure and diabetes) in patients with renal impairment may explain why this population are at greater risk of more serious illness with COVID-19.[6]

Studies also indicate that kidney disease severity increases with aging in COVID-19 patients. The combination of increased comorbid conditions and decreased immune system's functionality among older people can make them more prone to infections. On the other hand, patients with kidney failure usually receive immunosuppressive drugs for treatment. These agents work by downregulating the immune system and have the ability to interfere with the processes in the body that fight infection. Therefore, this therapeutic regimen can increase the risk of infection.

On the basis of the reports mentioned above, kidneys are one of the most common organs that may be affected by the SARS-CoV-2. Until now, the exact mechanism of kidney involvement in COVID-19 is not known. Moreover, there are no proven therapeutic agents against this newly identified coronavirus.

Therefore, better understanding of the pathogenic mechanisms of kidney damage in the setting of COVID-19 is a strategic necessity for accurate diagnosis, predicting the course and prognosis of disease. Advances in rapid identification of patients with COVID-19 who are at risk of kidney failure is also crucial for the quick initiation of suitable therapy. Therefore, further studies are needed to explain the pathophysiologic changes that eventually leads to the initiation or development of kidney complications in patients with COVID-19.

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