COVID-19 and the Kidney

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Coronavirus disease 2019 (COVID-19) is a new respiratory infectious disease that originated in Wuhan, China, in December 2019 and caused by a new strain of zoonotic coronavirus, named severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2). SARS-CoV-2 belongs to β-coronavirus which also includes the severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS) viruses.[1] COVID-19 rapidly spread and now it is global pandemic disease. Currently (available data up to 23 July 2020) the coronavirus is affecting 212 countries and territories around the world, with over 15 million cases and 630,000 confirmed deaths (global mortality:5.4%).[2] A flu-like syndrome of mild severity has been observed in most cases (80%) of COVID-19, but in 20% of cases there have been other complications such as interstitial pneumonia with variable degree of respiratory failure as well as thromboembolic complications, including venous thromboembolism (VTE), ischaemic stroke and acute coronary syndrome (ACS)/myocardial infarction.[3] Although the principal features associated with COVID-19 are diffuse alveolar damage and acute respiratory failure, kidney impairment has also often developed, with frequent onset of acute kidney injury(AKI) in patients infected by SARS-CoV-2. In addition more than 20% of deceased patients were affected by chronic kidney disease (CKD).[4] Many studies reported that SARS-CoV-2, like SARS-CoV, uses Angiotensin converting enzyme 2 (ACE-2) to enter target cells.[4] The expression of ACE-2 has been shown not only in lungs but also in the liver, stomach, ileum, colon, esophagus and kidney. AKI (7%), myocardial dysfunction with acute cardiovascular events (12%) and gastrointestinal disorders are among the most frequent clinical manifestations of COVID-19, suggesting that SARS-CoV-2 can infect these organs. Studies performed on SARS-CoV suggested that AKI in SARS patients was the result of specific pathogenic conditions, such as the cytokine release syndrome,[5] rather than active viral replication in the kidney. Rhabdomyolysis due to muscle damage can cause kidney dysfunction by direct toxicity or by activating RASS resulting in acute tubular necrosis. The SARS-CoV-2 contribution to the development of CKD could involve pathways similar to those described for the acute kidney injury. The direct tubule-glomerular cellular injury, due to virus, often manifest with proteinuria and hematuria that, in turn, could start a chronic, non-reversible, process. It has been shown that proteinuria exerts a direct toxic effect on renal tubular cells and promotes renal fibrosis over time. In conclusion, the renal damage observed in COVID-19 patients is the complex mechanisms induced directly and indirectly by SARS-CoV-2 that predispose to the development of renal dysfunction.

References

1. Sun P, Lu X et al. Understanding of COVID-19 based on current evidence. J. Med. Virol. 2020; 10-13
2. Numbers, S.I.N. Coronavirus disease 2019 (COVID-19) Sci.Total Environ 2020
3. Boraschi P. COVID-19 Pulmonary involvement: Is really the interstitial pneumonia? Acad Radiol.27, 900
4. Hoffman M. et al SARS-CoV-2 Cell entry depends on ACE2 and TMPRSS2 and is blocked by a clinically proven protease inhibitor. Cell 2020
5. Tisoncik J.R. et al, Into the eye of the cytokine storm. Microbiol. Mol. Biol. Rev.2012, 76, 16-32
6. Abbate M, Zoja C, Remuzzi G. How does proteinuria cause progressive renal damage? J. Am. Soc. Nephrol. 2006; 17: 2974-2984

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