Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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European Association of Urology

Letter to the Editor

Reply to Peng He, Xiaohui Wang, and Hao Li's Letter to the Editor re: Yu Xiao, Kaiyu Qian, Yongwen Luo, et al. Severe Acute Respiratory Syndrome Coronavirus 2 Infection in Renal Failure Patients: A Potential Covert Source of Infection. Eur Urol 2020;78:298–9

We appreciate the letter from He *et al.* on our recent paper [1]. The authors provided strong clinical data in support of our primary findings. As He et al mentioned, chronic kidney disease (CKD) patients have a high rate of infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in hemodialysis (HD) centers. Therefore, we once again call on medical providers to pay special attention to SARS-CoV-2 infection of dialysis patients.

So far, no molecular mechanism has been identified to explain why the SARS-CoV-2 infection rate is higher among patients with CKD than in the normal population. We hypothesized that an immunosuppressive state could be linked to greater susceptibility to SARS-CoV-2. Therefore, organ transplant recipients and long-term users of immunosuppressive agents may be more susceptible to SARS-CoV-2 compared with the normal population.

In 1971, a novel human polyomavirus, named BK polyomavirus (BKV), was identified from the urine of an immunosuppressed renal transplant recipient. Subsequent studies found that 80% of the normal population tested positive for anti-BKV antibodies without any symptoms, while nearly 10% of renal transplantation patients suffered from polyomavirus-associated nephropathy. Thereafter, it was generally accepted that intense immunosuppression might be a risk factor for BKV nephropathy [2].

Researchers in Italy recently reported that among 20 patients with COVID-19 who underwent transplantation, five died, four were admitted to the intensive care unit, and three were discharged; among 21 patients with COVID-19 undergoing HD, five died and four were discharged; and among five patients with CKD, two died and two were discharged [3]. The results indicate higher severity and mortality among these immunosuppressed patients. The authors point out that treatment can be divided into two phases. The first phase is associated with viral replication, for which antiviral drugs may be considered. The second phase involves lung and cytokine release syndromes, for which immunomodulatory drugs may be of benefit [3]. Furthermore, Zhu *et al.* [4] enrolled ten renal transplant recipients with COVID-19 and ten of their family members diagnosed with COVID-19 pneumonia as a control group. They found that the transplant recipients had more severe disease and a longer virus shedding time than those in the control group. After a regimen involving cessation or reduction of immunosuppressive agents and administration of antiviral and supportive treatment, nine of the ten patients survived. The authors concluded that immunosuppression may have two opposing effects on the disease: making the early course of infection more severe and reducing mortality by suppressing the overactive immune response, in agreement with the opinions of Alberici *et al.*.

HD patients and renal transplant recipients are more vulnerable to SARS-CoV-2, possibly because of long-term immunosuppression, and are more likely to develop severe infection that may even be fatal. The treatment of COVID-19 in immunosuppressed patients may be more complicated than that in the normal population. More attention and special efforts are required to deal with the virus in this patient cohort.

***Conflicts of interest*:** The authors have nothing to disclose.

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