Letter to the Editor

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The Fuzzy Connection between SARS-CoV-2 Infection and Loss of Renal Function

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Dear Editor,

We appreciate the highly time-appropriate information presented in the article "Coronavirus Disease 19 Infection Does Not Result in Acute Kidney Injury: An Analysis of 116 Hospitalized Patients from Wuhan, China" by Wang et al. [1]. COVID-19 (coronavirus disease) is a newly emerged pandemic caused by a novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). As a close relative of other epidemic coronaviruses (SARS and Middle East Respiratory Syndrome), SARS-CoV-2 is highly contagious and exhibits flu-like symptoms with pneumonic illness [1]. Although 85% of patients recover from infection without needing hospitalization, elderly people and people with another condition that compromises the immune system such as hypertension or diabetes appear to develop serious illness [1, 2]. Detection of viral RNA in urine and kidney tissue of SARS-CoV-2-infected patients indicates the kidney as a target of infection [2]. Several of the recent reviews have mentioned kidney manifestations of COVID-19 [3, 4]. While angiotensin-converting enzyme 2 (ACE2) receptors are the functional receptor for the SARS coronavirus, their presentation in the lower respiratory tract, kidney, duodenum, and small intestine might provide possible routes of entry for the SARS-CoV [5]. Studies in a mouse model of SARS-CoV infection demonstrated that ACE2 expression is vital for viral entry and ACE2 overexpression is directly proportional to disease severity [6]. As bradykinin B1 receptor mediates the cross talk between ACE2 and the kinin-kallikrein system in setting the inflammation, selective bradykinin B1 receptor blockers could be used as a promising agent to prevent tissue inflammation during COVID-19 infection [7].

Significant functional impairment of the kidney in COVID-19 patients [8, 9] is well known, and we see that in our patients frequently. Development of acute kidney injury (AKI) is also associated with significant increased mortality during hospital stay [9]. AKI can be a surrogate for multi-organ failure and serves as a negative prognostic factor for survival in COVID-19 patients [10].

We wonder whether the incidence of AKI by reporting time, and not the total incidence of AKI, was reported in this study by Wang et al. [1] They report that as of February 13, 2020, 7 (6.03%) acute respiratory distress syndrome patients transferred to ICU died of respiratory failure. This is much lower than the overall mortality of more than 52% reported for acute respiratory distress syndrome in COVID-19 patients from Wuhan [10]. We suspect the same methodologic problem underlies the underestimation of AKI in COVID-19 in his study. Therefore, larger studies in this regard are necessary.

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Author Contributions

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