LETTER TO THE EDITOR

COVID-19–Associated Acute Kidney Injury

To the Editor:

In their case series of acute kidney injury (AKI) associated with coronavirus disease 2019 (COVID-19), Patel et al[1] proposed that a hypercatabolic state with muscle breakdown was the cause of AKI as they presented 3 cases that typify hypercatabolic state, with rapid increase in serum urea nitrogen levels, hyperuricemia, and hyperphosphatemia. However, in all 3 cases, the urea-creatinine ratio (UCR) was unchanged and was not elevated, as is observed in a hypercatabolic state.[2] Serum urea nitrogen and creatinine levels (UCR initial/peak: case 1, 17.5/16.2; case 2, 12/13; case 3, 9.7/13) in all 3 cases were essentially unchanged and were not in keeping with a true hypercatabolic state.[2] In addition, the near-normal creatine kinase levels do not support significant muscle breakdown unless these patients had pre-existing cachexia. The hyperuricemia and hyperphosphatemia could very well be the effects of AKI rather than initiating factors; however, their role in perpetuation of AKI can not be excluded. The rapid decrease in serum albumin level is more consistent with a capillary leak syndrome, as seen in severe sepsis-systemic inflammatory response syndrome that I agree results from the various inflammatory cytokines (interleukin 6, tumor necrosis factor, etc). I agree with the authors that better understanding of the factors associated with COVID-19–associated AKI and the role of cytokines is important.

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ARTICLE INFORMATION

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