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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Letter to the editor

Renal failure in COVID-19 ARDS: Could it be partially avoided?

We read with great interest the study by Roedl et al.[1] presenting mortality among intensive care unit patients. In our opinion, a few, very interesting findings need further clarification, which may strengthen the study's value. Renal failure needing renal replacement therapy was much more frequent in mechanically ventilated (MV) patients than in those subjected to noninvasive ventilation/High Flow Nasal Oxygen Cannula (NIV/HFNC) (42% vs 5%, respectively).[1]

In addition, disease severity Sequential Organ Failure Assessment (SOFA) was higher in MV patients than in those subjected to NIV/HFNC on admission, further increasing only in MV patients at 24 h. Accordingly, 43% of MV patients needed vasopressors, reaching 73% within 24 h (compared with only 5% in those subjected to NIV/HFNC).[1] We wonder if shock was the main contributor to increased SOFA scores and whether shock resulted in renal malfunction.

Renal damage mechanisms are not completely clear in patients with Corona Virus Disease-2019 (COVID-19), although prerenal aetiology seems to be prevalent in hospitalised patients, constituting a negative prognostic factor.[2] Renal hypoperfusion leads to acute kidney injury, finally warranting renal replacement therapy.[3] Therefore, it must be clarified if shock, needing vasopressors, was established after MV initiation. Furthermore, data on baseline renal function (i.e., creatinine) and time of deterioration, as well as data on the first 48-h fluid balance, in both groups would be informative.

Patients with COVID-19 and respiratory failure are hypovolemic because of fever in the preceding days and respiratory distress compromising adequate fluid intake. However, restricted fluid resuscitation—usually adding diuretics—is indicated in patients with Acute Respiratory Distress Syndrome (ARDS), trying to keep the lung “dry”, a strategy helpful to avoid intubation. Sedation, used for intubation, releases the sympathetic tone inducing vasodilation, further aggravating shock. Finally, heart–lung interactions contribute to the magnitude of shock when MV is initiated. A substantial amount of the positive pressure (positive end-expiratory pressure [PEEP], plateau pressure) is transmitted to the pleural pressure when lung compliance is relatively normal, as in COVID-19,[4] deteriorating venous return.[5] Moreover, relatively high positive pressure levels, in a patient with hypovolemia on vasopressors, may compromise right heart and cardiac output by compressing the pulmonary vessels, already affected by capillary thrombosis.[6]

Although PEEP was wisely set lower than that proposed by the ARDSnet protocol in the study by Roedl et al,[1] as compliance was relatively preserved, we wonder if even lower PEEP and tidal volumes (to further decrease plateau pressure) may be initially indicated until the patient is adequately resuscitated.

In conclusion, we wonder whether fluid administration along with a ventilatory approach concerning lower PEEP, at least immediately after intubation, may partially prevent hypoperfusion, renal failure, end-organ damage, and an unfavourable outcome in MV patients with COVID-19.

Conflict of Interest

The authors report no relationships that could be construed as a conflict of interest.

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