LETTER TO THE EDITOR

Author Response

Pratibha Todur[1], Souvik Chaudhuri[2]

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Dear Editor,

We thank Magoon R et al. for raising such significant aspects regarding acute kidney injury (AKI) in acute respiratory distress syndrome (ARDS) patients.[1] We wanted to emphasize and deliberate on certain aspects.

First, we completely agree that in critically ill, the hemodynamics and vasopressor requirements, vasopressor regimens, and types (catecholamine sparing or not) are crucial aspects that herald the onset of AKI.[2] The article by Cartin-Ceba et al. identified comorbidities like diabetes, advanced age, higher severity of disease scores, need for vasopressors, and a higher baseline creatinine as risk factors for AKI onset in critically ill patients.[2]

In our study, the driving pressure, oxygenation, and modified nutritional evaluation (DRONE) score necessitates mean arterial pressure and vasopressor requirement assessments. The DRONE score has the modified nutritional risk in critically ill (mNUTRIC score) as one of its components.[3] The mNUTRIC score in itself includes factors like age, acute physiology, and chronic health evaluation (APACHE II score), and sequential organ failure assessment (SOFA) score as its elements. Thus, since the APACHE II score and the SOFA score were evaluated for the calculation of the DRONE score, the hemodynamics and vasopressor requirements had to be taken into consideration. In fact, 8 out of 13 variables that were outlined by Cartin-Ceba et al. as risk factors for AKI are actually encompassed in the mNUTRIC score-like age, comorbidities, severity of disease scores, and vasopressor use.[2]

Second, we focused on the recovery trajectories after AKI had already set in (classifying as AKI subphenotype-1 or 2), rather than the development or onset of AKI per se in ARDS patients.[3] True to the intriguing nature of this topic, a recent multicenter study showed that vasopressor use was not an independent predictor of renal recovery after AKI in the critically ill, whereas factors like initial AKI stage, hemoglobin level, diabetes mellitus, lactate level, and APACHE II score were independent predictors of renal recovery post-AKI.[4] Thus, factors that may herald the onset of AKI may not exactly mirror the factors that determine renal recovery after AKI. The ASSESS study revealed that patients in whom AKI lasted >72 hours had a greater risk of non-resolving AKI, along with those with proteinuria at 3 months.[5] To this end, we followed up the patients in our study for up to 120 hours for serum creatinine trajectories and found similar results.[3]

Third, our study was focused on AKI in ARDS patients (which is predominantly due to ventilator-induced kidney injury or VIKI) rather than sepsis-associated acute kidney injury (SA-AKI).[6] While poor oxygenation and lung compliance with higher driving pressures leads to VIKI, a far greater complicity of factors contribute to SA-AKI, like microcirculatory dysfunction and macrocirculatory abnormalities, mitochondrial dysfunction, metabolic reprogramming, and complement activation.[6] Even the biomarkers involved in VIKI and SA-AKI are different. Hypoxia, being a hallmark of ARDS, initiates the cascade of events causing renal tissue ischemia and eventually AKI in patients with invasive ventilation. However, in SA-AKI, a reduced global renal blood flow leading to tubular damage is the predominant reason leading to AKI. Thus, while a focus on vasopressor regimen and hemodynamics is quintessential for the prediction of SA-AKI, the spectrum shifts to the paradigm of ventilator mechanics and oxygenation for predicting VIKI. The recovery pattern from AKI (and therefore its subphenotypes) is another subject with an interplay of different variables altogether.

Thus, despite the advancement in knowledge about the lung-kidney crosstalk, we may just be at the tip of the iceberg, with its entirety remaining to be unraveled.

[1]Department of Respiratory Therapy, Manipal College of Health Professions, Manipal Academy of Higher Education, Manipal, Karnataka, India

[2]Department of Critical Care Medicine, Kasturba Medical College; Manipal Academy of Higher Education, Manipal, Karnataka, India

Corresponding Author: Souvik Chaudhuri, Department of Critical Care Medicine, Kasturba Medical College; Manipal Academy of Higher Education, Manipal, Karnataka, India, Phone: +91 9937178620, e-mail: souvik.chaudhuri@manipal.edu

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Orcid

Pratibha Todur https://orcid.org/0000-0003-0967-2252
Souvik Chaudhuri https://orcid.org/0000-0001-8392-2366

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