LETTER TO THE EDITOR

Hemodynamics: Strangers to Lung-kidney Crosstalk in ARDS?

Rohan Magoon[1], Anupama Gill Sharma[2], Nidhi Yadav[3], Nang Sujali Choupoo[4]

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Dear Editor,

Todur et al. are applauded for their prospective observational study outlining lung-kidney crosstalk as an important determinant of acute kidney injury (AKI) prognosis in a cohort of acute respiratory distress syndrome (ARDS) patients on invasive mechanical ventilation (IMV).[1] The authors' demonstration of the role of driving pressure, oxygenation, and nutritional evaluation (DRONE) score in predicting non-resolving (subphenotype 2) AKI is noteworthy, but there do exist other facets of the research subject necessitating deliberation.[1]

Talking of the extrapulmonary consequences of ARDS, poor lung compliance is expected to lead to a reduction in the cardiac output, primarily owing to elevated intrathoracic pressures. Hence, hemodynamics per se is far from being strangers to the lung-kidney crosstalk featured in the Todur et al. research.[1] Appropriate to the context, one misses an account of the systemic blood pressure targets and/or vasopressor-inotropic management in the index study. After the assessment of the cumulative data from 31 diverse studies involving a total of 5,04,535 critically ill patients, Cartin-Ceba et al. associate vasopressor-inotrope use with an accentuated risk of AKI, on separate random-effects meta-analysis (odds ratio: 4.52; 95% confidence interval: 2.03, 10.05; $I^2 = 92\%$).[2] Meanwhile the value of I^2 for the parameter over 75% bespeaks the prevailing inter-study heterogeneity, specific literature on AKI in ARDS deserves concurrent mention.[2,3] In a study including 57 patients with severe ARDS due to influenza A (H1N1) pdm09 virus (pH1N1), Tignanelli et al. link vasopresssor use and duration with the need for continuous renal replacement therapy (CRRT). Out of 77% (44/57) patients on vasopressor support in their setting, those on CRRT have received vasopressors more often than those not requiring CRRT (89% vs 53%, $p = 0.008$) and received vasopressors for a longer duration as well (mean ± standard deviation, 10 ± 10.2 vs 1.9 ± 2.4 days, $p = 0.003$).[3]

Moreover, the nature of the vasopressor regimen (catecholamine or catecholamine-sparing) is equally relevant. With recent primary and *post-hoc* analytical literature (VANISH, VANCS, and ATHOS-3 randomized controlled trials) focusing on differences between the potential renal impact of norepinephrine with vasopressin in septic shock, the importance of reporting the vasopressor practices in observational studies akin to Todur et al. can indeed not be undermined.[1,4] Of further note, the authors utilized the respective highest and the lowest 48 hours values of driving pressure and PaO_2/FiO_2 on IMV, to compute the corresponding DRONE score while following up the serum creatinine trajectories of their patients till day 5 for AKI-subphenotype characterization, where the vasopressor-inotrope support would have also classified as a peculiarly dynamic parameter of prognostic importance.[1] To that end, it is only for the acknowledgement of the upcoming role of vasopressor quantification employing scoring systems norepinephrine equivalents (NEE), vasoactive-inotropic score (VIS), and cumulative vasopressor index), that the novel models like the MAVIC (mechanical ventilation, acute physiology and chronic health evaluation-III, vasopressors, inotropes, Charlson comorbidity index) are captivating enhanced attention for risk stratification in critical care.[5]

[1–4]Department of Anaesthesia, Atal Bihari Vajpayee Institute of Medical Sciences and Dr. Ram Manohar Lohia Hospital, New Delhi, India

Corresponding Author: Nang Sujali Choupoo, Department of Anaesthesia, Atal Bihari Vajpayee Institute of Medical Sciences and Dr. Ram Manohar Lohia Hospital, New Delhi, India, Phone: +91 9711128628, e-mail: sujalichoupoo@yahoo.com

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Orcid

Rohan Magoon https://orcid.org/0000-0003-4633-8851
Anupama Gill Sharma https://orcid.org/0000-0002-5364-7348
Nidhi Yadav https://orcid.org/0000-0003-3186-5712
Nang Sujali Choupoo https://orcid.org/0000-0001-6270-3981

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