Letter to the Editor

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Pulmonary vasculature in COVID-19: mechanism to monitoring!

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Although the different mechanisms affecting the extent of hypoxemia in severe acute respiratory syndrome corona virus 2 (SARS-CoV-2) pneumonia, the relevance of the proposed phenotypic classification of COVID-19 related acute respiratory distress syndrome (ARDS), and the ideal ventilation strategies according to the existing ARDSnet protocol continue to be debated, the recognition of COVID-19 as an endothelial disease with vascular endothelium breakdown at the root of organ dysfunction is beyond debate [1]. The pulmonary vasculature in particular is prone to insult given that the protective endothelial glycocalyx (EG) is peculiarly thin in the pulmonary capillaries. The disruption of the EG due to systemic inflammation and the resultant cytokine storm, along with dynamic alteration in the endothelial angiotensin-converting enzyme 2 and angiotensin peptide levels, disturb the pulmonary vascular homeostasis, culminating in vascular hyperpermeability and pulmonary edema, leading to substantial oxygenation impairment [2].

While a rigid division of the distinct COVID-19 phenotypes into Type-L (low ventilation/perfusion ratio, lung weight, and recruitability with preserved compliance) and Type-H (high right-to-left shunt, lung weight, and recruitability with reduced compliance) has been referred to as anecdotal by a few, the proponents of the classification themselves suggest a possible transition from Type-L to Type-H as the disease evolves [1]. The latter research group cites negative intrathoracic pressure (patient self-induced lung injury) and inflammation-associated enhanced permeability as the major causative factors for interstitial lung edema in such circumstances [1]. It is noteworthy that autopsies performed on those who succumb early during the COVID-19 disease process showed remarkable pulmonary vascular congestion [2]. Vascular disease can also explain the massive elevations seen in the D-dimer levels, which heralds multisystem involvement with vasculopathy at the heart of the matter [2].

As an extension of the aforementioned realization of the mechanistic role of vascular dysfunction in SARS-CoV-2ARDS, monitoring of the pulmonary vasculature by lung ultrasound and/or transpulmonary thermodilution (TPTD) based assessment of extravascular lung water (EVLW) can be of considerable assistance in characterizing lung edema. Serial EVLW assessment can also help to monitor lung protective ventilation and recruitment maneuvers, guide fluid-diuretic therapy, and evaluate overall response to treatment [3]. Moreover, the pulmonary vascular permeability index (PVPI, preload-indexed EVLW) may also be computed (in the presence of other TPTD-derived preload variables), which when elevated pinpoints enhanced capillary permeability as the primary cause of pulmonary edema [3].

Groeneveld and Verheij [4] outlined that a link between pulmonary vascular injury and an increase in PVPI extends from the cohort of mechanically ventilated patients with pneumonia to those with extrapulmonary sepsis-induced forms of ARDS, which supports the role of monitoring for the same during the various progressive stages of

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COVID-related ARDS. A prospective multicenter large-scale study by Kushimoto et al. [5] discovered that PVPI values ranging from 2.6 to 2.85, rendered a definitive ARDS diagnosis with a specificity of 0.90 to 0.95, while a PVPI value < 1.7 effectively ruled out a diagnosis of ARDS with a specificity of 0.95. In addition, by following the quantitative diagnosis of pulmonary edema ($EVLW > 10$ ml/kg), monitoring of PVPI can also assist in the management of patients with COVID-19 with associated cardiac morbidities by an augmented delineation of the cardiogenic causes (elevated EVLW with normal PVPI) from the non-cardiogenic causes (elevated EVLW and PVPI, signifying 'leaky' pulmonary capillaries).

While the results of the prospective cohort study 'Extra vascular lung water and pulmonary permeability in critically ill patients with SARS-CoV-2 (COVID-19) (PiCCOVID)' (NCT04376905) are ardently awaited, the aforementioned discussion adequately highlights that a more objective form of disease progression and therapeutic response monitoring can develop as our comprehension of the COVID-19 related pathophysiology improves.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

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