This article has been accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the Version of Record.

Please cite this article as https://doi.org/10.4097/kja.20536
Title: Pulmonary vasculature in COVID-19: mechanism to monitoring!

Authors: (First name, middle name, & last name)                          E – mail address

1. Rohan Magoon, DM, MD, Assistant Professor                          rohanmagoon21@gmail.com

Department of Cardiac Anaesthesia, Atal Bihari Vajpayee Institute of Medical Sciences (ABVIMS) and Dr. Ram Manohar Lohia Hospital, Baba Kharak Singh Marg, New Delhi-110001, INDIA.

We do not have any conflict of interest, any commercial or financial interest in this material & agree to abide by the rules of your journal regarding publication of this article.

Running title: Pulmonary vasculature in COVID-19

Keywords: Acute respiratory distress syndrome; COVID-19; Extravascular lung water; Pulmonary vascular permeability indices; SARS-CoV-2 pneumonia.

Name and location of the institute where the work was carried out: -
Department of Cardiac Anaesthesia, Atal Bihari Vajpayee Institute of Medical Sciences (ABVIMS) and Dr. Ram Manohar Lohia Hospital, Baba Kharak Singh Marg, New Delhi-110001, INDIA.

Corresponding Author:

Dr.Rohan Magoon, DM, MD
Assistant Professor, Department of Cardiac Anaesthesia, Atal Bihari Vajpayee Institute of Medical Sciences (ABVIMS) and Dr. Ram Manohar Lohia Hospital, Baba Kharak Singh Marg, New Delhi -110001, INDIA.

Email: rohanmagoon21@gmail.com
Tel: +91-9711128628
Pulmonary vasculature in COVID-19: mechanism to monitoring!

- Letter to the Editor -

To The Editor,

Although the mechanisms relating to the severity of hypoxemia in SARS-CoV-2 pneumonia, relevance of the proposed phenotypic classification of COVID-19 related acute respiratory distress syndrome (ARDS), and the ideal ventilation strategies in light of the existing ARDSnet protocol, continue to be debated, the emergence of COVID-19 as an endothelial disease with vascular endothelium at the cornerstone of the organ dysfunction, is beyond debate [1]. In this context, the pulmonary vasculature is peculiarly prone to insult given the endothelial glycocalyx (EG) is particularly thin in the pulmonary capillaries wherein the disruption of the protective EG (owing to the microcirculatory dysfunction associated with the systemic inflammation and cytokine storm) and the dynamic alterations in the endothelial angiotensin-converting enzyme 2 (ACE-2) and angiotensin peptides, can disturb the pulmonary vascular homeostasis culminating as vascular hyperpermeability and pulmonary edema resulting in a substantial oxygenation impairment [2].

While a tight categorization of distinct phenotypes: 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 as anecdotal by few, the proponents of the classification themselves suggest a possible transition from Type-L to Type-H as the disease evolves, citing negative intrathoracic pressure (patient self-induced lung injury (P-SILI)) and inflammation associated enhanced permeability as the causative factors of interstitial lung edema [1]. It is noteworthy that the autopsies performed on those who succumbed early to COVID-19 have been discovered to be remarkable for pulmonary vascular congestion [2].
disease can also explain the massive elevations in the D-dimer levels heralding the constellation of a multisystem involvement with vasculopathy at the heart of the matter [2].

As an extension of the aforementioned realization of the mechanistic role of vascular mechanisms in SARS-CoV-2 ARDS, monitoring pulmonary vasculature by ultrasonographic or transpulmonary thermodilution (TPTD) assessment of extravascular lung water (EVLW) can be of considerable assistance in characterizing lung edema, monitoring lung protective ventilation and recruitment manoeuvres, guiding fluid-diuretic therapy and the overall response to treatment [3]. Moreover, 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 an enhanced capillary permeability as the primary cause of pulmonary edema [3].

Appropriate to the context, Groeneveld and Verheij [4] outlined that the link between pulmonary vascular injury and rise in PVPI extends from the cohort of mechanically ventilated pneumonia patients to those ailing from extrapulmonary sepsis-induced forms of ARDS, supporting the role of monitoring the same in the various stages of progression of 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 specificity of 0.90–0.95, and a PVPI value < 1.7 effectively ruled out a diagnosis of ARDS with specificity of 0.95. In addition, following the quantitative diagnosis of pulmonary edema (EVLW>10 mL/Kg), monitoring PVPI can assist the management of COVID-19 patients 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)’ are ardently awaited, the aforementioned discussion adequately highlights that a much more objective form of
disease-progression and therapeutic-response monitoring can potentially evolve as an improved comprehension of the COVID-19 related pathophysiology transpires.
References


