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Editorial

# Pathophysiology and Hemodynamics of Pulmonary Hypertension in COVID-19

**Vijaya L Kankanala[1]* and Sushanth Dosala[2]**

[1]*Lead Primary Care Physician, Presbyterian Medical Group, High Resort Internal Medicine, New Mexico, USA*

[2]*Clinical Research Coordinator, Department of Internal Medicine, University of Illinois, USA*

***Corresponding Author:** Vijaya L Kankanala, Lead Primary Care Physician, Presbyterian Medical Group, High Resort Internal Medicine, New Mexico, USA.

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It has been a well-established fact that COVID-19 has caused a significant spectrum of disease activity in different organ systems, with the cardiovascular system being the most frequent system involved after the lungs, with an incidence of 30%.

Pulmonary hypertension is a pulmonary vascular disease characterized by pulmonary vascular remodeling and vasoconstriction, leading to elevated pulmonary artery pressure and right heart failure ultimately. Bilateral lung involvement with extensive alveolar and interstitial infiltrates, thickening of alveolar septa, vascular congestion and lung edema are some of the pathologies associated with lung damage from COVID-19 [1]. Due to the lung parenchymal damage and altered circulation in pulmonary micro and macro circulation, pulmonary hypertension develops.

Different pathways have been investigated, including the arginine-NO and Renin-angiotensin system in the development of pulmonary hypertension associated with coronavirus. Also, other considerations are possible downregulation of angiotensin II and decreased angiotensin levels leading to pulmonary vasoconstriction and dysregulation of hypoxic vasoconstrictive mechanisms. Upregulation of endothelin -1 is also well-known physiology behind the development of Pulmonary hypertension [2].

The pathophysiology of this type of pulmonary hypertension is complex and multifactorial, and mechanisms such as oxidative stress, mitochondrial dysfunction, and DNA damage, inflammation, hypoxia associated with endothelial dysfunction, and pulmonary micro embolism have been considered potential factors for the alterations of pulmonary circulation. It has been proposed that this type of PH should be considered a combination of PH group 3 (due to fibrosis and or obstructive lung disease) and group 4 (due to pulmonary artery obstruction) [1].

In a recent study by Deng., *et al*. it was reported that 21% of patients with severe covid-19 had evidence of pulmonary hypertension, and 2% of patients with mild to moderate disease had pulmonary hypertension [3]. In a study by Li., *et al*. the mean pulmonary artery systolic pressure of 48 mm hg in patients who were succumbed to death compared to 28 mm hg in survivors [4].

Invasive hemodynamic monitoring is the gold standard way of measuring the pulmonary arterial pressure; however, given technical difficulties due to the pandemic, transthoracic echocardiogram has offered us significant insight into understanding the hemodynamics [6]. It has always been known that pulmonary hypertension is a negative outcome associated with severe ARDS and mechanically ventilated patients. A retrospective study noted that the incidence of pulmonary hypertension in mechanically ventilated patients is 24% [5]. This data can probably more or less be extrapolated to COVID-19 severe infection requiring mechanical ventilation, although real-time data is still biased due to technical difficulties.

A more interesting fact is that mild to moderate disease in younger population less than 55 years old with no significant comorbid conditions are associated with a pulmonary hypertension prevalence of 7.69% and right ventricular dysfunction in 10.28% [1]. Another noteworthy point in this study is that initial levels of inflammatory markers correlate with the severity and development of pulmonary hypertension [1]. Despite all the patients being treated with recommended anticoagulation during the hospital stay and 40 days after discharge, pulmonary hypertension developed, explaining endothelial dysfunction and microangiopathy.

Pulmonary function testing along with a Transthoracic echocardiogram would be a good breakdown in understanding the role of each of these physiologies at different stages of recovery. The role of endothelin receptor antagonists, ACE-2, inhaled nitric oxide is yet to show us the pathway in the long-term challenges we face with pulmonary hypertension in COVID-19 patients.

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