# Severe Hypoxemia in Early COVID-19 Pneumonia

Pradeep Bhatia, MD; Sadik Mohammed, MD

Department of Anaesthesiology and Critical Care, AIIMS, Jodhpur. (Rajasthan)

INDIA

**Address of Correspondence to:**

Sadik Mohammed,

Associate Professor, Department of Anaesthesiology and Critical Care, AIIMS,

Phase II, Basani, Jodhpur. Rajasthan. (INDIA) 342005.

E-mail: drmsadik@gmail.com

**Word Count: 359**

**Acknowledgments:** NIL

**Conflicts of Interest:** The authors declare no conflicts of interest

**Funding:** The authors have no sources of funding to declare for this manuscript

AJRCCM Articles in Press. Published June 24, 2020 as 10.1164/rccm.202004-1313LE

To the Editor:

Luciano Gattinoni is widely acknowledged and respected for his work on ARDS, and this time he has suggested a very interesting concept describing the pathophysiology of the atypical presentation of SARS-CoV-2 induced respiratory failure.[1] Based on detailed observation of several cases, the hypothesis of dividing the time-related disease spectrum within two primary "phenotypes" Type L and Type H looks logical and might be helpful in the management of COVID-19 patients. The suggested cause of hypoxemia in Type L is the loss of regulation of perfusion and loss of hypoxic vasoconstriction. Hypoxemia, leading to increase minute ventilation, primarily by increasing the tidal volume (up to 15-20 ml/kg), is associated with a more negative intrathoracic inspiratory pressure and the magnitude of this pressure swing is projected as a factor which may determine the transition from the Type L to the Type H phenotype. However, the authors did not give explanation for loss of regulation of perfusion and loss of hypoxic pulmonary vasoconstriction.

We believe that diffuse pulmonary micro vascular thrombosis is the cause of hypoxemia in early pneumonia by SARS CoV-2. The histologic and immunohistochemistry studies suggest that in severe COVID-19 infection, a catastrophic, complement-mediated thrombotic microvascular injury occurs, with sustained activation of the actin pathway and lectin pathway cascades,[2]

AJRCCM Articles in Press. Published June 24, 2020 as 10.1164/rccm.202004-1313LE

leading to the recommendation of the use of early anticoagulation with low molecular weight heparin.[3]

We agree with the authors that to reverse hypoxemia, oxygenation by high flow nasal cannula may be tried in type L patients. However, we have reservation on the “early intubation and the use of PEEP to prevent the transition to type H”, as the authors themselves have suggested that “the lung conditions are too good”. Effective oxygenation using HFNC/ECMO in type L should prevent pleural pressure swings and self-inflicted lung injury, leading to transition to type H. Additionally, some degree of “permissive hypoxemia”[4] may also be accepted in type L patients to avoid ergotrauma, caused during ventilating the compliant lungs.

However, other patients, who worsen to Type H due to cytokine storm, as the authors have suggested, should be treated as severe ARDS, including higher PEEP, if compatible with hemodynamics, prone positioning and extracorporeal support.

AJRCCM Articles in Press. Published June 24, 2020 as 10.1164/rccm.202004-1313LE

## References

1. Gattinoni L, Coppola S, Cressoni M, Busana M, Rossi S, Chiumello D. COVID-19 Does Not Lead to a "Typical" Acute Respiratory Distress Syndrome. *Am J Respir Crit Care Med* 2020;201(10):1299-1300.

2. Magro C, Mulvey JJ, Berlin D, Nuovo G, Salvatore S, Harp J, et al. Complement associated microvascular injury and thrombosis in the pathogenesis of severe COVID-19 infection: A report of five cases. Transl Res. 2020 Apr 15:S1931-5244

3. Working Group of 2019 Novel Coronavirus, Peking Union Medical College Hospital. Diagnosis and clinical management of Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) infection: an operational recommendation of Peking Union Medical College Hospital (V2.0). Emerg Microb Infect. 2020;9:582–585.

4. Abdelsalam M, Cheifetz IM. Goal-Directed Therapy for Severely Hypoxic Patients With Acute Respiratory Distress Syndrome: Permissive Hypoxemia. Resp Care. 2010;55(11):1483-90.

AJRCCM Articles in Press. Published June 24, 2020 as 10.1164/rccm.202004-1313LE