Severe Covid-19 disease: rather AVDS* than ARDS?

*Acute Vascular Distress Syndrome

Yazine Mahjoub[1*], Daniel Oscar Rodenstein[2] and Vincent Jounieaux[3]

[1] Cardiac Vascular Thoracic and Respiratory intensive care unit
Department of anaesthesia and critical care
Amiens University medical centre, Amiens, France

[2] Pneumology department
Cliniques Universitaires Saint-Luc,
Université Catholique de Louvain. Brussels. Belgium

[3] Pneumology department
Amiens University medical centre, Amiens, France

*corresponding author
Yazine Mahjoub; MD, PhD.
Head of the Cardiac, Thoracic-vascular and Respiratory Intensive Care Unit.
Department of anesthesia and Critical Care.
University Hospital Centre. Amiens. France
Email: Mahjoub.Yazine@chu-amiens.fr
Phone: +33 3 22 08 78 73

Word count: 400

Dear Editor,

We read with great interest the editorial by Gattinoni et al. describing two time-dependent chronological phenotypes of patients suffering from Covid-19 pneumonia [1]. We do not only agree about the role of vasoplegia accounting for hypoxemia but we believe that this lung vascular disorder is the common denominator of Covid-19 pneumonia which is present at all stages of the disease, making useless to distinguish types 1 and type 2 Covid-19 ARDS.

In spontaneously breathing patients hospitalized for Covid-19 pneumonia, we have observed a severe hypoxemia despite Chest CT scans showing quite limited grass-ground like opacities. Because of the limited alveolar damage, such hypoxemia can be best explained by a low ventilation-to-perfusion (VA/Q) ratio where VA is preserved and Q is increased which we suggest to be related to an increase in pulmonary vascular flow and not to an inhibited hypoxic pulmonary vasoconstriction (supposing that Alveolar PO_2 is preserved). Moreover, assuming that alveolar ventilation is conserved, compensatory hyperventilation rapidly leads to hypocapnia which is known to be a very powerful inhibitor of the hypoxic ventilatory response [2]. This may explain the surprizing lack of dyspnoea noted in these patients.

Some of these patients may worsen (extension of grass-ground like opacities, appearance of lung consolidations) requiring ICU transfer. These patients effectively present a non-typical ARDS with refractory hypoxemia, a slightly decreased lung compliance, low lung recruitability (type 1 described by Gattinoni) and, in our experience, rarely show right ventricular dysfunction as in typical ARDS (personal data). In a recent post-mortem study on Covid-19 patients, Varga et al. found endothelial cell infection by SRAS-Cov2 and endotheliitis [3]. This might be explained by the presence of ACE-2 receptors on vascular endothelial cells

[4]. All these findings suggest a specific pulmonary vascular disorder induced by SARS-CoV-2, leading us to consider it as an acute vascular distress syndrome (AVDS) more than an atypical ARDS. Nevertheless, some patients do evolve to a more typical ARDS (type 2 described by Gattinoni et al.) due to extensive lung consolidations. Finally, when such patients recover from Covid-19 pneumonia, we observe a return to normoxia despite the persistence, and sometimes the worsening, of lung consolidations compared to the early stage of ARDS.

To conclude, according to our findings and to recent publications, we hypothesize that Covid-19 patients, at any stages of their disease, are characterized by an increased pulmonary blood flow with intrapulmonary right to left shunt with limited alveolar injury.

Ethical Approval and Consent to participate
Not applicable

Consent for publication
Not applicable

Availability of supporting data
Not applicable

Competing interests
The authors state that they have no conflict of interest.

Funding
Only institutional funds were used for this study**.**

Authors' contributions
YM and VJ and DOR wrote the first draft of the manuscript

Acknowledgements
none

Authors' information

Yazine Mahjoub; MD, PhD.
Head of the Cardiac, Thoracic-vascular and Respiratory Intensive Care Unit.
Department of anesthesia and Critical Care.
University Hospital Centre. Amiens. France
Mahjoub.Yazine@chu-amiens.fr
Phone : +33 3 22 08 78 73

Daniel Oscar Rodenstein, MD, PhD
Emeritus Professor, Pneumology Unit.
Cliniques Universitaires Saint-Luc,
Université Catholique de Louvain. Brussels. Belgium
rodenstein-boutsen@skynet.be

Vincent Jounieaux MD, Ph.D
Head of the Respiratory Department.
University Hospital Center. Amiens. France
Corresponding author
jounieaux.vincent@chu-amiens.fr

References

1. Gattinoni L, Chiumello D, Rossi S. Covid-19 pneumonia: ARDS or not? Crit Care.2020;24:154

2. Jounieaux V, Parreira VF, Aubert G, Dury M, Delguste P, Rodenstein DO. Effects of hypoxic hyperventilation on the response to hypoxia in normal subjects receiving intermittent positive-pressure ventilation. Chest.2002; 121:1141-1148

3.Varga Z, Flammer AJ, Steiger P et al. Endothelial cell infection and endotheliitis in COVID-19. The Lancet. doi: 10.1016/S0140-6736(20)30937-5

4. Zou X, Chen K, Zou J, Han P, Hao J Single-cell RNA-seq data analysis on the receptor ACE2 expression reveals the potential risk of different human organs vulnerable to 2019-nCoV infection. Front Med. doi : 10.1007/s11684-020-0754-0, 2020.