LETTERS TO THE EDITOR

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The hyperdynamic circulatory profile of patients with COVID-19-related acute vascular distress syndrome. Letter regarding the article 'Haemodynamic characteristics of COVID-19 patients with acute respiratory distress syndrome requiring mechanical ventilation. An invasive assessment using right heart catheterization'

We read with great interest the article by Caravita *et al.*[1] who analysed the haemodynamic profile of 21 mechanically ventilated COVID-19 patients. The authors found that COVID-19 patients had a lower indexed pulmonary vascular resistance (PVR) (3.4 ± 1.6 mmHg/L/min/m^2 vs. 4.6 mmHg/L/min/m^2, $P = 0.002$) and higher cardiac index (3.7 ± 1.1 L/min/m^2 vs. 4.3 L/min/m^2, $P = 0.024$) than pooled 1937 patients with non-COVID-19 acute respiratory distress syndrome (ARDS), despite similar severity (non-different positive end-expiratory pressure support, lung compliance and PaO_2/FiO_2 ratio).

From these observations, the authors concluded that COVID-19 patients needing mechanical ventilation are characterized by an increased Qs/Qt ratio, low PVR, and high cardiac output associated with post-capillary pulmonary hypertension that could be explained by an inflammation-driven hyperdynamic circulation and a blunted pulmonary hypoxic vasoconstriction. This hyperkinetic circulatory state in which the pulmonary vascular bed shows a minimal increase in pulmonary arterial pressure with increased cardiac output looks strangely similar to the most frequent haemodynamic pattern observed in liver disease, including the hepatopulmonary syndrome.[2] Indeed, in liver disease, pulmonary arterial pressure can increase, usually <45 mmHg, in response to high cardiac output, due to passive distension of compliant arterial vessels and recruitment of upper lung blood vessels.

We previously suggested that an intrapulmonary right-to-left shunt, as seen in such hepatopulmonary syndrome, could explain most of the COVID-19-related symptoms, leading us to propose the acronym AVDS for 'acute vascular distress syndrome'.[3] In this hypothesis, we may observe increased cardiac output, decreased PVR and increased Qs/Qt leading to a low PaO_2/FiO_2 ratio. To the best of our knowledge, the study by Caravita *et al.*[1] is the first to describe such a haemodynamic profile. Nevertheless, as static lung compliance and PaO_2/FiO_2 ratio are similar in the two groups, we suggest that the results may not be explained only by a blunted hypoxic vasoconstriction (in this case, a lower PaO_2/FiO_2 ratio in COVID-19 is expected, which is not the case). As the PaO_2/FiO_2 ratio is similar in the two groups, we suggest that the main mechanism to explain the increased cardiac output with decreased PVR is an intrapulmonary shunt comparable to that observed in the hepatopulmonary syndrome. However, when COVID-19 progresses and affects more the lung parenchyma than the lung vessels (moderate to severe ARDS), the worsened lung lesions hide the intrapulmonary shunt by inducing a certain degree of pulmonary hypoxic vasoconstriction. This hypothesis can account for all the specific symptoms observed in this disease: the happy hypoxia in spontaneous breathing patients,[4] the usefulness of prone positioning,[5] the limited efficacy of inhaled nitric oxide, the potential effect of almitrine and the PaO_2/FiO_2 improvement despite chest radiological worsening (Mahjoub Y., unpublished data).

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