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Short Notes

SARS-CoV2 Happy Hipoxemia: is it really happy?

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INTRODUCTION

A significant number of patients affected by COVID-19 present a pattern of severe respiratory failure, with heterogeneous characteristics of acute respiratory distress syndrome (ARDS) [1]. Many of them show a marked alteration in the ventilation-perfusion ratio (V/Q) and, consequently, an increase in the shunt and intrapulmonary dead space with impaired oxygenation; on the other hand, different reports [2-4], show the presence of thrombosis and diffuse alveolar damage, which translates into an increase in dead space and shunt too, which reduces CO_2 elimination and, probably, an increase in respiratory rate. In some patients, relatively preserved compliance values have been observed and, additionally, a reduction in the hypoxic vasoconstriction mechanism has been postulated as a consequence of an alteration in the carotid bodies function [5]. Thus, we would be in the presence of patients showing hypoxemia and hypercapnia with no more functional alteration than an increase in respiratory rate. This is what was called happy hypoxemia. This clinical picture usually presents greater severity around 7-10 days from the onset of symptoms.

Considering that the respiratory drive control is regulated by the bulbar center, whose function depends on the pH and CO_2 levels, the evolution of the clinical picture may show more accentuated respiratory efforts, with increased use of accessory muscles and marked amplitude in the changes of pleural pressure. This scenario, together with the combination of high respiratory rates (due to hypercapnia or fever) and vigorous diaphragmatic contractions can generate changes in the levels of stress and pulmonary strain, generating changes in transpulmonary pressure and alveolar edema. Patient self-inflicted injury may occur at this stage, facing with "air hungry" will make sudden, deep and violent respiratory movements. At this point dyspnea would be inevitable, since the relationship between the work of breathing and the volume gain obtained is unbalanced.

The effects of non-invasive mechanical ventilation (NIV) in patients with pulmonary edema are well known; however, in the context of COVID-19, it has been suggested that high-flow nasal cannula (HFNC) or the use of Helmet might have more beneficial effects in the early phases of hypoxemic respiratory failure, although conflicting results have been reported [6-9]. Prone positioning has also been proposed, with the aim of improving the V/Q ratio and, consequently, oxygenation, CO_2 elimination and avoiding intubation and invasive mechanical ventilation (IMV) as an adjuvant therapy with favorable outcomes [10,11]. Certainly, this may represent a point of concern since not all patients with hypoxemic respiratory failure tolerate this position and, on the other hand, the application of HFNC and prone positioning, if well tolerated, could mask impaired function due to lung disease and delay the onset of IMV. The precise moment to discontinue this therapy and begin IMV is up to be elucidated. In this sense, a "plea" has been proposed not to systematically intubate patients with severe hypoxemia, allowing a SpO_2 close to 88%, the use of nasal cannula (6L/min) or HFNC, and allowing patients to adopt the prone position [12].

Tobin et al. [13], mention that these patients may present not so severe hypoxemia and, therefore, not present dyspnea. Other studies have shown that dyspnea is a consequence of the action of pulmonary receptors that capture the relationship between the work of breathing and the volume of air achieved [14,15], and not the concentration of CO_2 and oxaemia. These findings agree with what was mentioned by Gattinoni et al., about the possibility of finding a compliance close to normal that would explain why some of these patients do not present dyspnea [16].

In short, much has been published since the start of the pandemic, which raises the need for a critical reading and a return to the physiological bases of the mechanisms of gas exchange, work of breathing, and dyspnea. Taking into account that the disease is accompanied by a significant inflammatory process, the course of the clinical evolution of the disease, together with the complementary tests and the evolution of the acute phase reactants curve (PCR, D-dimer, LDH, ferritin; although they are not specific parameters), it could be useful to address respiratory failure secondary to SARS-CoV2. The definitive sequelae of this disease are still unknown; therefore, future studies will be necessary to elucidate the whole picture.

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