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COVID-19 Pneumonia: A Complex Management Tailored on the Disease Stage

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Description

Despite falling in many respects under the Berlin definition of acute respiratory distress syndrome (ARDS) [1], COVID-19 pneumonia is not a "Typical" ARDS. The same disease actually presents itself with impressive non-uniformity. Two disease types or stages can be identified: the early, "non-ARDS", and the advanced, "ARDS" [2-5]. The staging of the disease is crucial to decide the right therapeutic approach. An important role in the transition from one stage to the other is played by the activation of the coagulative cascade triggered by inflammation and the pulmonary micro and macro thromboembolism, both contributing to the worsening evolution of the syndrome [6,7].

Discussion

COVID-19 requires a complex management tailored on the disease stage or type. Thus, it is necessary to identify parameters able to correctly classify the disease stage and to avoid the evolution from one stage to another. The following parameters should be considered:

i) Signs: Tachypnea without dyspnea in the early stage; dyspnea without tachypnea in the advanced disease. The persistence of tachypnea and high transpulmonary negative pressures despite satisfactory SaO2 has been reached are indicators of a possible p-SILI (patient self-inficted lung injury) and therefore of a worsening of lung mechanics;

ii) Distribution of alveolar edema: the assessment should be quantitative (sub-pleural distribution in the early stage, diffused in the advanced stage) and qualitative terms (sporadic B-lines in the early stage on lung ultrasound, "white lung" with signs of pulmonary thickening in the advanced stage);

iii) Different response to therapy: good response to oxygen therapy (PaO2 enhancement) and good response to low PEEP delivery (P/F improvement) in the early stage, poor in the advanced stage. The early stage (non-ARDS) is characterized by high lung compliance and low alveolar recruitment and a mismatch between lung damage, P/F ratio and mild symptoms is observed. In this stage the use of high PEEP could be harmful both for the hemodynamic and ventilatory profile (it can worse the V/Q ratio and the shunt effect leading to hypoxia[8]).

iv) Markers for prediction of intraparenchymal thrombosis could guide the choice of antithrombotic therapy: 1) high D-dimer concentrations, 2) lung damage based on the aforementioned parameters, 3) ETCO2/PaCO2 ratio <1 evaluated by capnography which indicates elevated shunt and dead space (areas of lung ventilated and not perfused).

Conclusion

Based on the aforementioned parameters, in the early stage (non-ARDS) the clinician should evaluate the increase of PaO2, by boosting FIO2 (through Venturi mask, reservoir), since lungs promptly respond to oxygen therapy with PaO2 enhancement. In the advanced stage (ARDS), when pulmonary thrombosis occurs, PaO2 does not increase despite boosting FIO2, since dead space and shunt fraction rise. Due to high lung compliance and low alveolar recruitment, P/F ratio rises with low PEEP in the early stage.

Understanding the correct COVID-19 stage is crucial in order to establish the appropriate treatment, also avoiding iatrogenic complications.

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