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A Short Commentary on COVID-19 related Acute Respiratory Distress Syndrome

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Description

Severe Acute Respiratory Distress Syndrome (ARDS) is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, which is represented by COVID-19 (ARDS). Its symptoms can be seen as a synthesis of the two conditions, viral pneumonia and ARDS. The rare disease COVID-19 was first identified in Wuhan, China, in December 2019 and has since spread throughout the world. Diffuse lung alveolar destruction results from ARDS. In the acute stage, the alveoli develop hyaline membranes, which are then followed by interstitial widening, oedema, and fibroblast proliferation in the organising stage [1].

The characteristic ARDS pathological features of diffuse lung alveolar destruction are brought on by COVID-19 ARDS. Consider how clearly the findings demonstrate that, despite the former having more severe hypoxaemia, respiratory system compliance was much higher in COVID-19-associated ARDS compared with classical aetiology ARDS. In keeping with this, we draw your attention to their finding that static compliance declined in patients with classical ARDS and in a pneumonia subset of patients with ARDS, however it remained unaltered in patients with COVID-19 ARDS [2].

ARDS caused by COVID-19 seems to have poorer outcomes than ARDS caused by other factors. Older age, the existence of concomitant conditions such hypertension, cardiovascular disease, and diabetes mellitus, reduced lymphocyte counts, renal damage, and elevated Ddimer levels are risk factors for poor outcomes. According to COVID-19 ARDS mortality statistics, deaths result from respiratory failure (53%), respiratory failure and cardiac failure (33%), myocardial injury and circulatory failure (7%), or death from unexplained causes.

The COVID19 pneumonia patients, however, exhibit an unusual type of the illness despite fitting the Berlin diagnosis of ARDS. In fact, the main phenomenon we are finding is a separation between the degree of hypoxemia and their rather well-preserved lung mechanics. It is now clear that patients with COVID-19 ARDS may experience thromboembolic consequences. There are documented cases of pulmonary embolism, and a recent study on the French experience with extracorporeal membrane oxygenation in COVID-19-induced ARDS revealed two oxygenator thromboses and 20% of pulmonary embolism occurrences.

Pneumonia and respiratory symptoms are both caused by COVID-19, and intensive care units require mechanical ventilation when respiration rates exceed 30 per minute. Following severe pneumonia brought on by COVID-19, emergency intubation is a crucial procedure, and it must be carried out by a qualified anesthesiologist in order to control the necessary settings in ventilator machines [3]. In almost 60% of cases, diffuse ground-glass opacity and alveolar consolidation were the most common patterns in COVID-19 ARDS. The lengthy median (IQR) time delay between the onset of symptoms and orotracheal intubation may help to explain this consolidation [4].

D-dimer concentrations have been found to be indicators of disease severity and predictors of mortality. Therefore, the drop in D-dimers is indicative of a decline in illness severity. In this instance, the prothrombotic condition transforms into a pro-hemorrhagic pattern as a result of the normal course of events, contrary to previous research that highlighted a potential transition to a DIC associated with death. In contrast, after two weeks, we noticed a return to normal of the viscoelastic parameters in the patients who lived. In our series, we were unable to detect a transition toward this state [5].

Similar to generic ARDS, the poor prognosis of cardiac injury in COVID-19 is a result of multisystem organ involvement. Myocardial damage is linked to markers of inflammation, iron metabolism, and thrombotic activity. In our data set, thorough cardiac imaging such as echocardiography is not accessible, and point-of-care ultrasonography is frequently substituted for formal cardiac imaging in COVID-19.

Patients with ARDS have lungs that are hypercoagulable, which causes fibrin to deposit in the intra-alveolar area. By triggering C-reactive protein (CRP), inflammation alters coagulation by increasing tissue factor exposure on monocytes and alveolar macrophages, which in turn encourages thrombin production and fibrin deposition. Fibrin deposition is further exacerbated by hepatic synthesis of fibrinogen, an acute phase protein, which is raised 2- to 10-fold in plasma during infection and is evident during pneumonia. Deposition of fibrin promotes fibrosis and inflammation while also harming lung surfactant. Increased levels of D-dimers and fibrin degradation products (FDP), which are linked to a higher risk of mortality, are a frequent observation in COVID-19 patients who need hospitalisation. A slight lengthening is visible in the prothrombin time and activated partial thromboplastin times. Patients with ARDS frequently experience disseminated intravascular coagulation (DIC), when fibrin and microthrombi are seen in the lungs and BAL.

Conflict of Interest

The author declares that there is no conflict of interest associated with this manuscript.

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