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Acute Respiratory Distress Syndrome as a Post COVID issue

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Short Opinion

Acute Respiratory Distress Syndrome (ARDS) is characterised by the fast onset of non-cardiogenic pulmonary edema, which leads to respiratory failure and hypoxia. Over the last 25 years, efforts such as the ARDS and Prevention and Early Treatment of Acute Lung Injury (PETAL) Networks have proven commendable collaboration to improve ARDS therapy. However, progress has been slow, and ARDS continues to be a primary cause of death in perioperative and critical care settings. The high frequency of Coronavirus Disease 2019 (COVID-19) patients has recently highlighted the considerable morbidity and mortality of ARDS. The fact that present ARDS treatment is limited to preventive measures, such as the use of lung-protective ventilation, is a major roadblock to reducing ARDS mortality.

Therapeutic techniques that address the underlying inflammatory lung illness are the subject of extensive research, although they have yet to be tested in humans. Despite this, basic science and clinical research initiatives targeted at identifying novel therapeutic options and improving ARDS outcomes are still underway. We discuss evidence-based management methods for ARDS in this article, focusing on those that are being studied or heavily used in ARDS linked to COVID-19. A high prevalence of thromboembolic and hemorrhagic events has been described in critically unwell patients with acute respiratory distress syndrome (ARDS) coronavirus disease 2019 (COVID-19).

COVID-19 may affect the coagulation cascade, resulting in an imbalance in platelet function and coagulation and fibrinolysis regulating systems. Thromboembolic events, haemorrhage, and disseminated intravascular coagulation are among the clinical signs, which range from an increase in laboratory markers and subclinical microthrombi to thromboembolic events, haemorrhage, and disseminated intravascular coagulation. The tissue factor pathway, which induces endotoxin and tumour necrosis factor-mediated generation of interleukins and platelet activation, is the mechanism for activating the coagulation cascade in COVID-19 after an inflammatory stimulus. Inflammatory infiltrates in the endothelium region, as well as thrombocytopenia, may result from the large influx of activated platelets.

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