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Broad Perspective on Acute Respiratory Distress Syndrome and Pathogenesis

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Perspective

Acute Respiratory Distress Syndrome (ARDS) is a noncardiogenic pulmonary edema-induced illness of acute respiratory failure. Despite five decades of basic and clinical research, no effective medication exists for this illness, and treatment is mostly supportive. To increase our understanding of ARDS and reduce mortality, researchers must investigate the molecular and physiologic pathways that cause it. The purpose of this review is to summarise what we now know about the pathogenesis and pathophysiology of ARDS. To begin, we'll go over how pulmonary edoema fluid builds up in ARDS patients as a result of lung inflammation and increased alveolar endothelial and epithelial permeabilities. Following that, we'll go through how pulmonary edoema fluid is routinely removed in a healthy lung and how these pathways are interrupted in ARDS. Finally, we'll go over how clinical trials and preclinical investigations of novel therapeutic agents have helped us learn more about this syndrome, with a focus on the use of mesenchymal stromal cells in the treatment of ARDS.

For patients with severe Acute Respiratory Distress Syndrome (ARDS), prone placement is now considered one of the most effective techniques. The pathologic understanding of the prone posture has evolved in lockstep with the development of ARDS. The improvement in oxygenation owing to perfusion redistribution was the focus of the prone posture at first. The mechanics underlying the prone position, on the other hand, are more complicated. Indeed, the prone position's beneficial effects on oxygenation and CO_2 clearance can be attributed to more uniform inflation-ventilation, a lung/thoracic shape mismatch, and a change in chest wall elegance.

Over the last two decades, five large trials have attempted to demonstrate the usefulness of the prone position, beginning with various theories, hypotheses, and designs and eventually finding its definite place among the various ARDS supportive regimens. The Acute Respiratory Distress Syndrome (ARDS) has been recognised as a serious clinical concern in respiratory medicine from its first description. ARDS produced about 300 indexed articles between July 2015 and July 2016. As an arbitrary overview of clinical relevance, this review summarises only eight of them: Risk factors, prevention, and therapy are all covered in the definition and epidemiology section. Although precise application of definition criteria is essential, the many resource-setting situations encourage spatial diversity and disparate outcome data. ARDS is underdiagnosed, according to a major worldwide multicenter prospective cohort study including 50 nations on five continents, and there is room for improvement in its care.

In addition, epidemiological data from low-income countries suggests that the present definition of ARDS needs to be revised in order to enhance its detection and global clinical outcome. Although precise application of definition criteria is essential, the many resource-setting situations encourage spatial diversity and disparate outcome data. ARDS is underdiagnosed, according to a major worldwide multicenter prospective cohort study including 50 nations on five continents, and there is room for improvement in its care.

In addition, epidemiological data from low-income countries suggests that the present definition of ARDS needs to be overhauled in order to enhance its detection and global clinical outcome. Acute Respiratory Distress Syndrome (ARDS) is a noncardiogenic pulmonary edoema that causes dyspnea, tachypnea, and hypoxemia to worsen rapidly. The presence of new or worsening respiratory symptoms within one week of a known injury, significant hypoxemia, bilateral lung opacities on radiography, and the inability to explain respiratory failure by heart failure or fluid overload are all diagnostic criteria. ARDS is hypothesised to develop when a pulmonary or extrapulmonary insult triggers the production of inflammatory mediators, encouraging the buildup of inflammatory cells in the alveoli and disruption of lung microcirculation. Inflammatory cells cause pulmonary edoema, hyaline membrane development, decreased lung compliance, and impaired gas exchange by damaging the vascular endothelium and alveolar epithelium. The majority of cases are linked to pneumonia or sepsis. One out of every ten admissions to intensive care units and one out of every four mechanical ventilations is due to ARDS.

Patients with severe ARDS have an in-hospital death rate of 46% to 60%. ARDS must frequently be distinguished from pneumonia and congestive heart failure, both of which show indications of fluid overload. Ventilation system, prophylaxis for stress ulcers and venous thromboembolism, nutritional assistance, and therapy of the underlying damage are all part of ARDS treatment. Low tidal volume and strong positive end-expiratory pressure help patients get better results. For some mild and all severe instances, prone positioning is advised. A spontaneous breathing trial is recommended as patients with ARDS improve and the underlying illness improves to determine whether they are eligible for ventilator weaning.

Patients who survive ARDS are at risk for reduced functional capacity, mental illness, and a lower quality of life; these patients benefit from continued treatment from a primary care physician. Pulmonary infiltrates on CXR and CT, as well as decreased lung compliance, are the hallmarks. ARDS develops within a week after being exposed to a precipitating event, which is most commonly pneumonia, shock, aspiration of gastric contents, sepsis, or trauma. The illness is usually inhomogeneous on CT imaging, with gravity infiltrates coexisting with patches of normal density as well as hyper aerated parenchyma. ARDS linked with septic shock and intensive care illnesses has a significant mortality rate (30-60%).

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