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Acute Respiratory Distress Syndrome: Risk of an Overloaded Diagnosis

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Abstract

Acute Respiratory Distress Syndrome (ARDS) develops when fluid accumulates in your lungs' small, elastic air sacs (alveoli). Because the fluid prevents your lungs from filling with enough air, less oxygen reaches your circulation. This deprives your organs of the oxygen they require to perform properly. ARDS is a potentially fatal form of respiratory failure that affects around 200,000 individuals in the United States each year, resulting in nearly 75,000 deaths. Globally, ARDS accounts for 10% of intensive care unit admissions, resulting in about 3 million ARDS patients each year.

Keywords: ARDS • Lungs • Ventilation approach • Alveoli • Respiratory failure

Introduction

Acute respiratory distress syndrome diagnosis

The Berlin definition has much higher mortality predictive validity than the previous American-European consensus conference definition. Clinician assessment of edoema origin and chest radiograph criteria may be less reliable in diagnosing ARDS. Mechanical ventilation remains the cornerstone of therapy, with the goal of minimising Ventilator-Induced Lung Harm (VILI). In patients at high risk for ARDS, aspirin proved ineffective in preventing the condition. Adjunctive therapies to further reduce VILI, such as prone positioning in patients with a PaO_2/FiO_2 ratio less than 150 mm Hg, were associated with a significant mortality benefit, although others (for example, extracorporeal carbon dioxide removal), are yet experimental. Pharmacologic therapy targeting pathophysiologic changes in ARDS, such as 2 agonists, statins, and keratinocyte growth factor [1].

Mechanical ventilation

A tailored mechanical ventilation approach for patients with ARDS based on lung physiology and morphology, ARDS aetiology, lung imaging, and biological characteristics may enhance ventilation practise and result. However, further research is needed before individualised mechanical breathing techniques can be used at the bedside. Ventilatory settings should be titrated based on close monitoring of targeted physiologic indicators and personalised goals. Although low tidal volume (vt) is a standard of treatment, further individualization of vt may demand the assessment of lung volume reserve.

Low driving pressures give an objective for physicians to modulate VT and maybe optimise Positive End-Expiratory Pressure (PEEP) while keeping plateau pressures below safety standards. Although esophageal pressure monitoring allows for the estimate of transpulmonary pressure, its clinical application at the bedside necessitates technical skill and precise physiologic interpretation. Mechanical power takes into account all ventilatory characteristics when optimising ventilation settings, although further research is needed to determine its therapeutic utility. Identification of recruitability in ARDS patients is critical for titrating and individualising PEEP. Clinicians should consider oxygen transport and dead space when defining gas exchange objectives for specific patients [2].

Researchers found two subtypes of Acute Respiratory Distress Syndrome (ARDS) with varied clinical trajectories and therapeutic responses using Latent Class Analysis (LCA) of clinical and protein indicators. We wanted to see if plasma metabolites differed between patients with LCA-derived hyperinflammatory and hypoinflammatory ARDS and we wanted to see if adding metabolic clusters to LCA phenotypes may help predict outcomes. We compared 970 metabolites between the two LCA-derived phenotypes in 93 ARDS patients with sepsis recruited in a multi-center prospective cohort of critically sick patients. There were 188 metabolites that differed in abundance between the two LCA-derived phenotypes. 82 metabolites remained substantially different after controlling for age, gender, confounding drugs, and concomitant liver and renal illness. Patients with hyper inflammatory ARDS showed lower levels of circulating lipids but higher amounts of pyruvate, lactate, and malate. Survival was strongly and independently linked with metabolic cluster and LCA-derived traits. Patients with hyper inflammatory ARDS may

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be suffering a glycolytic shift, resulting in dysregulated lipid metabolism. Metabolic profiling provides prognostic information in addition to LCA traits. Deeper biologic profiling may reveal critical distinctions in pathophysiology across ARDS patients, leading to novel targeted therapeutics [3].

COVID-19 lung complications

Since the advent of the COVID-19 pandemic, races have begun in academia and industry to uncover and develop disease modifying or preventative treatment options. The primary focus has been on pharmacological treatment of the immunological and respiratory systems, as well as vaccine development. The hyperinflammatory condition ("cytokine storm") reported in many COVID-19 cases implies a prognostically poor disease progression that may result in respiratory distress, multiple organ failure, shock, and death. Many critically sick patients are nonetheless at danger of serious, long-term morbidity or fatality [4]. The central nervous system extensively regulates the human immune and respiratory systems, and intervening in the signalling of these neural pathways may allow for targeted therapeutic management of excessive inflammation and pulmonary bronchoconstriction. Several invasive and non-invasive technologies are available and licenced for clinical use, but have not been well researched in the treatment of the cytokine storm in COVID-19 patients. This publication presents an overview of the nervous system's role in inflammation and respiration, as well as a rationale for investigating non-invasive neuromodulation to modify acute systemic inflammation and respiratory dysfunction caused by SARS-CoV-2 and potentially other viruses [4].

The model's performance was assessed using the Receiver Operating Characteristic Curve (ROC) analysis, the Hosmer-Lemeshow test, and the calibration curve [5]. As independent predictors, age, haemoglobin, heart failure, renal failure, Simplified Acute Physiology Score II (SAPS II), immune function impairment, Total Bilirubin (TBIL), and PaO_2/FiO_2 were discovered [6]. The prediction model's algorithm was: ln (Pr/(1+Pr)=-3.147+0.037 age-0.068 haemoglobin+0.522 heart failure (yes)+0.487 renal failure (yes)+0.029 SAPS II+0.697 immune function impairment (yes)+0.280 TBIL (abnormal)-0.006 (Pr represents the probability of death occurring). The model's AUC was 0.791 (0.766-0.816), and both internal and external validations validated the model's strong performance. A nomogram was created and validated to predict the probability of death in ARDS patients. It may aid clinicians in identifying ARDS patients at high risk of death, reducing mortality and improving ARDS survival [7].

Conclusion

We examine the rationale for tailored methods to mechanical ventilation for patients with ARDS, the role of lung imaging, phenotypic identification, physiologically based personalized ventilation systems, and a future research agenda in this review.

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