

Adult Respiratory Distress Syndrome: Mechanisms of Lung Injury and Advances in Critical Care Management

Sarah Collins*

Department of Respiratory Medicine, University of Oxford, United Kingdom

Editorial

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*For Correspondence

Sarah Collins, Department of Respiratory Medicine, University of Oxford, United Kingdom

E-mail: sarah.collins@ox.ac.uk

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INTRODUCTION

Acute Respiratory Distress Syndrome (ARDS) is a severe form of acute lung injury that leads to respiratory failure. First described in 1967, ARDS continues to pose a significant challenge in critical care due to its complex pathophysiology and lack of definitive treatment. The syndrome is characterized by rapid onset of hypoxemia, bilateral pulmonary infiltrates, and decreased lung compliance[1].

ARDS can result from direct lung injuries such as pneumonia, aspiration, and inhalation injury, or indirect causes such as sepsis, trauma, and pancreatitis. The incidence of ARDS varies globally but remains a leading cause of mortality in intensive care units (ICUs). Understanding its mechanisms and improving management strategies are crucial for enhancing patient survival.

METHODOLOGY

This article is based on a comprehensive narrative review of clinical studies, systematic reviews, and international guidelines. Sources included peer-reviewed journals, randomized controlled trials, and ICU-based observational studies.

The methodology focused on:

- Evaluating pathophysiological mechanisms of ARDS
- Reviewing diagnostic criteria and clinical features
- Analyzing recent advances in management strategies
- Comparing outcomes of different treatment approaches

DISCUSSION

Pathophysiology and Mechanisms of Lung Injury

The pathogenesis of ARDS is driven by an intense inflammatory response that damages the alveolar-capillary membrane. This results in increased permeability, allowing protein-rich fluid to accumulate in the alveoli. The disruption of this barrier impairs oxygen exchange and leads to hypoxemia[2,3].

Neutrophils are central to the inflammatory process. They release cytokines and proteolytic enzymes, which contribute to tissue injury. Key inflammatory mediators include interleukin-1, interleukin-6, and tumor necrosis factor-alpha. These mediators amplify the inflammatory cascade and perpetuate lung damage.

ARDS progresses through three phases:

Exudative Phase: Characterized by edema, inflammation, and hyaline membrane formation.

Proliferative Phase: Marked by cellular repair and resolution of edema.

Fibrotic Phase: In some patients, fibrosis leads to permanent lung damage.

Clinical Features and Diagnosis

Patients with ARDS typically present with acute onset of dyspnea, tachypnea, and hypoxemia. Oxygen therapy alone is often insufficient, necessitating mechanical ventilation.

Diagnosis is based on the Berlin criteria, which include:

Acute onset within one week

Bilateral opacities on chest imaging

Respiratory failure not explained by cardiac causes

Severity based on PaO₂/FiO₂ ratio

Advances in Critical Care Management

Mechanical Ventilation

Lung-protective ventilation using low tidal volumes is the cornerstone of ARDS management. This approach minimizes ventilator-induced lung injury.

Positive End-Expiratory Pressure (PEEP)

PEEP helps prevent alveolar collapse and improves oxygenation.

Prone Positioning

Prone ventilation improves ventilation-perfusion matching and has been shown to reduce mortality in severe ARDS.

Fluid Management

Conservative fluid strategies help reduce pulmonary edema without compromising organ perfusion.

Extracorporeal Membrane Oxygenation (ECMO)

ECMO provides respiratory support in severe cases unresponsive to conventional therapy.

Prognosis and Challenges

Despite advances, ARDS mortality remains high. Survivors often experience long-term complications such as reduced lung function and psychological issues. Early intervention and standardized care protocols are essential.

CONCLUSION

ARDS is a complex syndrome requiring prompt diagnosis and advanced supportive care. Continued research into targeted therapies and personalized medicine is essential to improve patient outcomes[4,5].

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