

Early Treatment of Severe Acute Respiratory Distress Syndrome



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KEYWORDS

- Acute respiratory distress syndrome • Acute respiratory failure • Hypoxia
- Hypoxemia • Severe ARDS

KEY POINTS

- Severe acute respiratory distress syndrome (ARDS) is a life-threatening condition characterized by acute bilateral pulmonary infiltrates occurring after a recognizable trigger and a PaO_2 to fraction of inspired oxygen (FiO_2) ratio of less than 100.
- Patients with all severities of ARDS should be managed with a low tidal volume strategy, safe plateau pressures, and fluid restriction as tolerated by hemodynamics.
- Patients with severe ARDS should receive early neuromuscular blockade and consideration for prone ventilation. Patients with severe ARDS not responding to therapy should be transferred to an ECMO center.

INTRODUCTION

ARDS is a rare but life-threatening syndrome characterized by acute bilateral inflammatory pulmonary infiltrates and severe hypoxia. US cases were estimated at 86 per 100,000 individuals, with 74,500 annual deaths in 2005.^{1–3} ARDS survival has improved due to advances in supportive care but mortality remains at 27% to 45% depending on the severity of ARDS. ARDS is classified into physiologic and prognostic categories of mild, moderate, and severe based on PaO_2 to FiO_2 (P/F) ratio (200–300, 100–200, and <100, respectively).⁴

ARDS may be triggered by pulmonary and nonpulmonary insults. It most commonly occurs in patients with acute critical illness due to sepsis, pneumonia, and trauma,

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where it is often accompanied by multiorgan dysfunction. Primary lung disease may be the initial or sole manifestation of acute severe ARDS in some cases, with pulmonary aspiration and near-drowning 2 common examples. This review discusses the clinical presentation of ARDS and provides an evidence-based approach to the early management of ARDS pertinent to emergency medicine physicians, with a focus on severe ARDS.

CLINICAL PRESENTATION AND ACUTE RESPIRATORY DISTRESS SYNDROME DEFINITION

Patients with ARDS exhibit hypoxemia associated with acute bilateral pulmonary infiltrates occurring within 1 week of a provoking insult. Intubation and mechanical ventilation with a high FiO_2 are often required to compensate for the large alveolar-arterial oxygen gradient. The former definition of ARDS and acute lung injury required exclusion of left atrial hypertension causing hydrostatic pulmonary edema. The revised criteria, however, removed this strict criteria, recognizing that inflammatory lung disease and elevated left atrial pressures are not mutually exclusive.⁴

The pathophysiology of ARDS includes increased pulmonary vascular permeability, loss of aerated lung, decreased lung compliance, and increase in physiologic dead space. The damaged capillaries allow protein-rich fluid to overwhelm the normal lymphatic drainage of the lung.⁵ Chest radiograph (CXR) frequently demonstrates diffuse and homogeneous infiltrates; however, CT scans often reveal a heterogeneous pattern of dependent consolidation.⁶

PATIENT EVALUATION

Hypoxia with acute bilateral infiltrates after a known trigger associated with ARDS is clinically diagnostic (**Table 1**). Usually a diagnosis of ARDS is determined with a good patient history, physical examination, and CXR data. Some patients develop ARDS during an emergency room course (eg, worsening sepsis, aspiration, and influenza), which can be overlooked without a high index of suspicion. Occasionally patients with ARDS present without a known trigger or an incomplete history, which makes a diagnosis of ARDS more difficult to confirm. Incomplete patient history and nonspecific time-consuming diagnostics are early hurdles in quickly identifying the inciting cause of ARDS for some atypical presentations and other causes for bilateral infiltrates should be considered (**Table 2**). Hydrostatic pulmonary edema commonly mimics ARDS and can be difficult to correctly identify. CXRs have limited

Sepsis	Pulmonary contusion
Aspiration	After upper airway obstruction
Infectious pneumonia	Stem cell transplant
Trauma	Drug reaction
Burn	Venous air embolism
Blood product transfusion	Amniotic fluid embolism
Cardiopulmonary bypass	Neurogenic pulmonary edema
Pancreatitis	Acute eosinophilic pneumonia
Drug overdose	Bronchiolitis obliterans organizing pneumonia
Near drowning	Smoke inhalation

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