QUICK LESSON

Acute Respiratory Distress Syndrome (ARDS)

Description/Etiology

Acute respiratory distress syndrome (ARDS; also known as noncardiogenic pulmonary edema) is an inflammatory lung condition that develops acutely—usually in 24–48hours —after an inciting event, such as infection (e.g., pneumonia), sepsis, or injury (e.g., severe nonthoracic trauma, lung contusion). ARDS is considered a medical emergency.

The pathophysiology of ARDS involves three phases: exudative, fibrosing alveolitis, and resolution, or recovery. The exudative phase is caused by the activation of cytokines (i.e., inflammatory molecules that send signals to other cells), which subsequently damage respiratory cells. Alveolar inflammation and damage lead to increased pulmonary capillary permeability and profound hypoxia; blood oxygen levels can remain dangerously low in spite of supplemental oxygen delivered by mechanical ventilation. The fibrosing alveolitis phase, which occurs 1–2 weeks after the inciting event, is characterized by residual hypoxia, pulmonary dead space, and decreased lung compliance. The resolution phase follows and can last 6–12 months. ARDS often occurs with failure of other organs, and the resulting end-organ damage is thought to be responsible for subsequent death in many patients. Potential complications of ARDS include pulmonary fibrosis, multiple organ failure, ventilator-associated pneumonia, permanent lung disease, oxygen toxicity, barotrauma, superinfection, pneumothorax (i.e., collapsed lung), and death. Survivors of ARDS are at risk for long-term functional disability, depression, anxiety, neurocognitive impairment, posttraumatic stress disorder, exercise limitations, and diminished quality of life.

According to the Berlin definition of ARDS (which was proposed in 2012 by an expert panel convened by the European Society of Intensive Care Medicine and endorsed by the American Thoracic Society and the Society of Critical Care Medicine), the condition is diagnosed in patients who meet the following criteria:

- > Onset within 1 week of a known clinical insult or new or worsening respiratory signs and symptoms
- > Bilateral opacities that are not fully explained by effusions, lobar/lung collapse, or nodules on chest X-ray or CT scan
- > Respiratory failure that is not fully explained by cardiac failure or fluid overload
- > Impaired gas exchange
 - Mild ARDS: P/F ratio (i.e., a measure of the *p* artial pressure of oxygen in arterial blood [PaO₂] divided by the *f* raction of inspired oxygen [FiO₂]) of 200–300 mm Hg with positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) $\geq 5 \text{ cm H}_2\text{O}$
 - Moderate ARDS: P/F ratio of 100–199 mm Hg with PEEP \ge 5 cm H₂O
- Severe ARDS: P/F ratio $\leq 100 \text{ mm Hg with PEEP} \geq 5 \text{ cm H}_2\text{O}$

Treatment involves maintaining respiratory function with tracheal intubation and positive-pressure mechanical ventilation (MV), and identifying and treating the causative condition. Patients who do not improve with MV can require extracorporeal membrane oxygenation (ECMO; i.e., use of an intravascular device to circulate blood through an artificial lung).

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ICD-10 J80

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Facts and Figures

ARDS accounts for 10.4% of ICU admissions. An estimated 23% of patients requiring mechanical ventilation have ARDS. The in-hospital mortality rate is 35% for patients with mild ARDS, 40% for patients with moderate ARDS, and 46% for patients with severe ARDS. Sepsis is the most common cause of the syndrome and is associated with a particularly poor prognosis.

Risk Factors

Patients with severe pulmonary or extrapulmonary infection are at risk of developing ARDS. Other risk factors include aspiration of gastric contents, shock, thoracic or nonthoracic trauma, fat or air embolism, pancreatitis, major surgery, cardiopulmonary bypass, disseminated intravascular coagulopathy, burns, inhalation of toxic fumes, near-drowning, sepsis, drug overdose and receiving multiple blood transfusions. Patients who are black, are male, or have a history of alcoholism or tobacco use also have an increased risk of developing ARDS. Diffuse alveolar damage in ARDS is associated with increased risk of death.

Signs and Symptoms/Clinical Presentation

Signs and symptoms of ARDS include shortness of breath, low blood pressure or shock, rapid and labored breathing, tachycardia, hyperventilation, agitation, cough, joint stiffness, lethargy followed by obtundation (i.e., diminished sense of awareness), pain, and apnea. Survivors of ARDS can have persistent pulmonary and/or nonpulmonary disability such as memory loss, cognitive impairment, brain atrophy, pulmonary fibrosis, and restrictive lung disease (i.e., restricted lung expansion that results in inadequate ventilation and/or oxygenation).

Assessment

> Physical Findings of Particular Interest

- Physical findings frequently include nasal flaring, moist and cyanotic skin, tachycardia, hyperventilation, flat neck veins, lethargy or obtundation, crackles at the lung bases, and low blood pressure
- > Laboratory Tests
 - ABGs identify hypoxemia, hypoxia, and hypocapnia ($\downarrow CO_2$; early onset); hypercapnia ($\uparrow CO_2$; later onset); and low P/F ratio in patients with ARDS
 - Other laboratory tests include CBC; blood chemistry testing for anemia and electrolyte disturbances; BUN; creatinine; BNP level to rule out cardiogenic pulmonary edema, sputum, blood, and urine cultures to assess for infection and sepsis; and cytologic examination of bronchoalveolar lavage samples obtained via bronchoscopy to assess for lung infection

> Other Diagnostic Tests/Studies

- Pulmonary artery catheterization is performed to measure pulmonary artery wedge pressure (PAWP)
- Pulse oximetry is important to monitor hypoxemia
- Chest X-ray is performed to assess for diffuse lung infiltrates
- Echocardiogram to assess for cardiogenic pulmonary edema if BNP level is not conclusive

Treatment Goals

> Provide Emergency Care, as Appropriate, and Promote Optimal Respiratory Function

- Assess and assist with resuscitation, as appropriate, and frequently monitor vital signs, oxygenation, serum electrolytes, ventilator settings, ABG results, pulmonary capillary wedge pressure (PCWP), and for hypoxemia, neurologic and mental status changes, and arrhythmias
- -Immediately report abnormalities to the treating clinician
- Administer and monitor prescribed treatment, including mechanical ventilation, high doses of oxygen (oxygen saturation > 90%), and PEEP of 5–10 cm H₂O, depending on FiO₂ level. To prevent ventilatory-induced lung injury, utilize a low tidal volume (6 mL/kg body weight) and plateau pressure ventilator strategy, as ordered
- Administer prescribed medications, including sedatives and neuromuscular blocking agents (e.g., tubocurarine, pancuronium bromide) during mechanical ventilation; sodium bicarbonate for severe metabolic acidosis; heparin for deep venous thrombosis (DVT) prophylaxis; DOPamine or DOBUTamine to maintain cardiac output; and antibiotics for pulmonary or extrapulmonary infections
- If ECMO is ordered, monitor insertion site for inflammation, infection, and bleeding; frequently assess for signs of ischemia and thrombosis; and measure diameter of the limb used for insertion for bilateral comparison

• Assess patient/family member anxiety level and coping ability; provide emotional support and educate about ARDS etiology and pathophysiology, potential complications, treatment risks and benefits, and individualized prognosis. As appropriate, request referral to a clergyperson and/or a mental health clinician for counseling on coping strategies and to a social worker for identification of resources for in-home care, hospice, and support groups

Food for Thought

- > Among the interventions being studied for their efficacy in ARDS are high-frequency oscillatory ventilation (HFOV), antioxidants, nitric oxide, cytokine inhibitors, and surfactant replacement
- > Compared with supine positioning, use of prone positioning is effective in improving oxygenation in 60–75% patients. Possible mechanisms by which prone positioning benefits patients with ARDS include expanding dependent lung areas thereby opening collapsed alveoli and increasing ventilation capacity—and reducing the work of breathing
- Investigators who conducted a meta-analysis of 11 randomized controlled trials that included a total of 2,246 adult patients calculated that ventilation in the prone position was associated with a 23% reduction in overall mortality, but was associated with a 49% increased risk of developing pressure injuries and a 55% increased risk of developing major airway complications (Lee et al., 2014)
- Statins can curb inflammatory responses and researchers in previous observational studies have reported that these drugs improve clinical outcomes in patients with ARDS. Researchers in a randomized, controlled trial, however, found that rosuvastatin did not improve the 60-dayin-hospital mortality rate of patients with sepsis-associated ARDS and might have contributed to liver and kidney dysfunction (Truwit et al., 2014)
- > Patients with diabetes mellitus (DM) are less likely to develop ARDS. It is theorized that the hyperglycemia inherent in patients with DM impairs neutrophil function; neutrophils are thought to play a role in the development of ARDS (Gibbons, 2015)

Red Flags

> Use of corticosteroids is not recommended for patients with persistent ARDS; patients with ARDS and type A H1N1 influenza infection who are treated with corticosteroids are at increased risk of healthcare-associated pneumonia and death

What Do I Need to Tell the Patient/Patient's Family?

- > Educate about the importance of continued medical surveillance after discharge to home and seeking immediate medical attention for new or worsening signs and symptoms
- > Recommend sources for additional information and support such as the ARDS Support Center at http://ardsglobal.org/

Note

> Recent review of the literature has found no updated research evidence on this topic since previous publication on November 4, 2016

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