Summer 2016

Acute Respiratory Distress Syndrome

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Recommended Citation
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Introduction
Acute respiratory distress syndrome (ARDS) is defined as lung failure with a ratio of partial pressure oxygen (PaO₂) to fraction of inspired oxygen (FiO₂) <100 (Michaelis, Hill, Long, Young, Sperly, Shanks, & Morgan, 2013). ARDS is characterized by acute, widespread pulmonary dysfunction due to injury (viral or bacterial), trauma, and/or inhaling toxins (Akojie, Palm, Ichikawa, & Takekawa, 2015).

Approximately 120,000 patients are diagnosed with ARDS each year in the U.S. with reported mortality rates varying from 20%-40% (Butt, Kardoukas, & Allen, 2015; Draheim & Custer, 2015). Risk factors for developing ARDS following an acute lung injury include age, males, African American, history of smoking, obesity, and diabetes (Modrykamien, 2015).

Diagnosis of ARDS is based on acute respiratory failure associated with extensive pulmonary infiltrates not inflamed by fluid from congestive heart failure (Butt, et al., 2015). Most patients who develop ARDS require mechanical ventilation, and the acute scorable acute respiratory distress syndrome typically develops within 3-5 days of hospitalization for an acute lung injury (Butt, et al., 2015). The radiographic chest image of a patient with ARDS is characterized by a "whiteout" of the lung, or patchy alveolar opacities, indicating alveolar hemorrhage (Hammer & McPhee, 2014). This image on the left below compares a normal chest x ray to that of a patient with acute respiratory distress syndrome. Notice in the image the "whiteout" appearance of right lung in the ARDS patient. The image on the right provides a "close-up" look at the alveolar edema.

Pathophysiology
After the initial injury or insult to the lung, the body's innate immune response is activated. An emerging theory in ARDS development is pattern recognition receptors (PRRs), which are essential components of the body's innate immune system, and can be described as the "first line of defense" (Butt, et al., 2016). Pattern recognition receptors readily identify pathogen-associated molecular patterns (PAMPs) and non-enzymatic damage-associated molecular patterns (DAMPs), then initiate the inflammatory signaling cascade of pro-inflammatory cytokines (Butt, et al., 2016). Pro-inflammatory cytokines, including tumor necrosis factor, and interleukin-1, interleukin 6, and interleukin-8, are released, which recruit neutrophils to the lungs (Siegel, 2016). Neutrophils invade the pulmonary tissue and release cytotoxic mediators, which include granule enzymes, bioactive molecules, complement and reactive oxygen metabolites. The mitogenic releases platelet activation factor, and lead to formation of microthrombi in pulmonary vasculature. All of these cytokines can also lead to tissue neomore, apoptosis, and autophagy and essentially, damage to the alveolar epithelium and the pulmonary capillary endothelium (Fujihira, 2014; Siegel, 2016).

The image below illustrates the process of ARDS pathology on the alveolus. Injury to the alveolar epithelium and pulmonary capillary endothelium causes increased alveolar permeability, leading to alveolar and interstitial edema (Draheim & Custer, 2015). The damaged capillary endothelium allows larger molecules and proteins to permeate out of the vasculature, and when the osmore pressure favoring fluid resorption is lost, the protein-dense fluid can fill the interstitial space (Siegel, 2016). This increase in interstitial fluid, combined with damage to the alveolar epithelium causes the air spaces to be filled with the protein-dense fluid and debris from autophagocytic cells (Siegel, 2016). The influx of fluid into the interstitium leads to a decrease in surfactant production, causing an increase in atelectasis and a decrease in lung compliance (Choi, 2009).

Patients who develop acute respiratory distress syndrome can be expected to move through a continuum of three phases. The first phase, often equated to exudative, early, or acute phase, develops 1-7 days after the initial injury/isult to the lungs (Choi, 2009). This phase is characterized by uncontrolled inflammatory shock to the alveolar capillary membrane, and leakage of protein dense fluid and blood, sometimes referred to as hemothorax edema, into the alveoli. Fibrin and plasma proteins develop a fibrin membrane on the alveolar walls that can impede gas exchange and decrease lung compliance (Akojie, Palm, Ichikawa, & Takekawa, 2015).

The second phase of ARDS is the proliferative (or organizing) phase, which develops 5-7 days after the onset, and can be characterized by the continued inflammation of the interstitial and proliferation of type II alveolar cells (Akojie, et al., 2015). Damage to type II alveolar cells also makes surfactant insufficient, contributing even more to atelectasis and decreased lung compliance (Draheim & Custer, 2015). As the proliferative phase continues, granulation tissue becomes part of the alveolar septum and leads to organ failure (Butt, et al., 2016).

The fibrous (stage 3) phase is the organizing phase of ARDS process, and is characterized by interstitial fibrosis, continued proliferation of type I cells, and chronic injury (Choi, 2009).

Treatment for acute respiratory distress syndrome can be complex as the disease itself. Mechanical ventilation using low tidal volumes, lung protective ventilation, prone position, high positive end expiratory pressures is the initial treatment for patients with ARDS (Michaels, et al., 2013). Prone positioning of patients with acute respiratory distress syndrome typically develops within 2-3 days of hospitalization for an acute lung injury (Butt, et al., 2015). The radiographic chest image of a patient with ARDS is characterized by a "whiteout" of the lung, or patchy alveolar opacities, indicating alveolar hemorrhage (Hammer & McPhee, 2014). This image on the left below compares a normal chest x ray to that of a patient with acute respiratory distress syndrome. Notice in the image the "whiteout" appearance of right lung in the ARDS patient. The image on the right provides a "close-up" look at the alveolar edema.

Pathogenesis
Acute respiratory distress syndrome is a life threatening condition that requires aggressive treatment with close monitoring. Successful treatment requires a multidisciplinary approach with knowledge from physicians, advanced practice nurses, bedside nurses, and respiratory therapists; all of whom must understand the complex underlying pathophysiology and critical nature of this condition.


References

Additional Sources


Conclusion
Acute respiratory distress syndrome is a life threatening condition that requires aggressive treatment with close monitoring. Successful treatment requires a multidisciplinary approach with knowledge from physicians, advanced practice nurses, bedside nurses, and respiratory therapists; all of whom must understand the complex underlying pathophysiology and critical nature of this condition.


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