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Acute Respiratory Distress Syndrome

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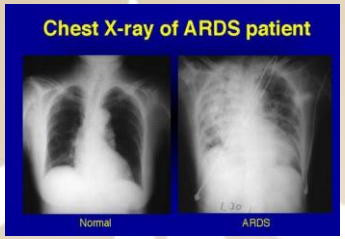
Introduction

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 (Michaels, Hill, Long, Young, Sperley, Shanks, & Morgan, 2013). ARDS is characterized by acute, widespread pulmonary inflammation due to infection (viral or bacterial), trauma, and/or inhaled toxins (Aokage, Palmer, Ichiba, & Takeda, 2015).

Approximately 150,000 patients are diagnosed with ARDS each year in the U.S. with reported mortality rates varying from 20%-40% (Butt, Kurdowska, & Allen, 2016; Drahnak & Custer, 2015). Risk factors for developing ARDS following an acute lung injury include age, males, African American, history of alcoholism, smoking, obesity, and diabetes (Modrykamien & Gupta, 2015).

Diagnosis of ARDS is based on acute respiratory failure associated with extensive pulmonary infiltrates not caused by fluid overload or cardiac failure (Butt, et al, 2016). Most patients who develop ARDS require mechanical ventilation, and the actual acute respiratory distress syndrome typically develops within 2-5 days of hospitalization for an acute lung injury (Butt, et, al, 2016). The radiographic chest image of a patient with ARDS is characterized by a "whiteout" of the lungs, or patchy alveolar opacities, suggesting alveolar edema (Hammer & McPhee, 2014, pp243). The 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.



Chest X-ray, Retrieved from https://www.homeofpoi.com/lessons_all/media/298-158-fig6.jpg

Alveoli in ARDS, Retrieved from https://i.ytimg.com/vi/lOj-4oH9ROY/hqdefault.jpg

There are not currently any biomarker molecules that are useful in the predicted severity of ARDS, and the PaO2/FiO2 ratio is used as a clinical indicator of severity (Butt, et al, 2016). Fuller, Granton, & McConachie (2015, pp363) describe the levels of severity of acute respiratory distress syndrome in three categories, based on the level of hypoxemia.

- Mild- PaO2/FiO2 >200 or <300mmHg with positive end expiratory pressure (PEEP) >5cm H2O
- Moderate- PaO2/FiO2 >100 or <200mmHg with PEEP >5cm H2O
- Severe- PaO2/FiO2 <100mmHg with PEEP >5cm H2O

The pathophysiology of acute respiratory syndrome is complex, and can result from a number of different insults. No matter the mechanism of injury, the common consequence is increased permeability pulmonary edema (Hammer & McPhee, 2014, p 243). A healthy, functioning lung is able to regulate a delicate balance between dry, patent alveoli and a very small amount of interstitial fluid. Any injury to the lung can upset this balance, resulting in impaired gas exchange, decreased lung compliance, and an increase in pulmonary arterial pressures, all which can lead acute respiratory failure (Siegel, 2016).

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 part of the "first line of defense" (Butt, et al, 2016). Pattern recognition receptors identify endogenous pathogen-associated molecular patterns (PAMPs) and non-endogenous 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 granular enzymes, bioactive lipids, complement and reactive oxygen metabolites. The cytotoxic mediators release platelet activation factor, and lead to formation of microthrombi in pulmonary vasculature. All of these cytotoxins can also lead to tissue necrosis, apoptosis, and autophagy and essentially, damage to the alveolar epithelium and the pulmonary capillary endothelium (Fujishima, 2014; Siegel, 2016). The image below illustrates the process of ARDS pathology on the alveolus.

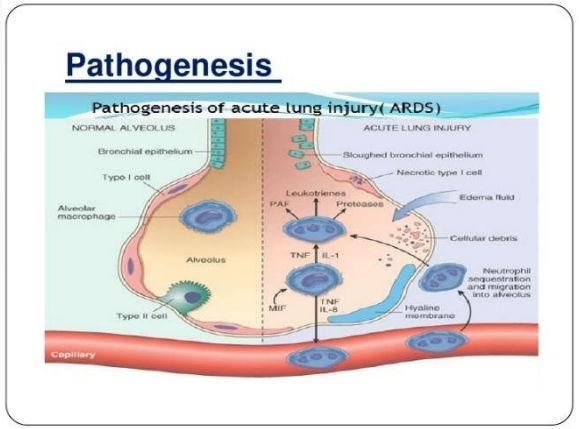
Injury to the alveolar epithelium and pulmonary capillary endothelium causes increased alveolar permeability, leading to alveolar and interstitial edema (Drahnak & Custer, 2015). The damaged capillary endothelium allows larger molecules and proteins to permeate out of the vasculature, and when the oncotic pressure favoring fluid resorption is lost, the protein dense fluid can flood 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 autophagic 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 referred to as exudative, early, or acute phase, develops 1-7 days after the initial injury/insult to the lungs (Choi, 2009). This early phase is characterized by uncontrolled inflammatory changes to the alveolocapillary membrane, and an influx of protein dense fluid and blood, sometimes referred to as hemorrhagic exudate, into the alveoli. Fibrin and plasma proteins develop a hyaline membrane on the alveolar walls that can impede gas exchange and decrease lung compliance (Aokage, Palmer, Ichiba, & Takeda, 2015).

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

The fibrosis (late) stage is the third phase of the ARDS process, and is characterized by interstitial fibrosis, continued proliferation of type II cells, and chronic inflammation (Choi, 2009).

Treatment for acute respiratory distress syndrome can be as complex as the disease itself. Mechanical ventilation using low tidal volumes, lung protective and recruitment maneuvers, inhaled nitric oxide, prone positioning, and the use of corticosteroids are the most utilized treatments (Michaels, et al, 2013; Zampieri, Mendes, Ranzani, Taniguchi, Azevedo, Costa, & Park, 2013). In the event of refractory hypoxemia and respiratory failure despite the therapies mentioned above, extracorporeal membrane oxygenation (ECMO) may be utilized (Turner, Rehder, Carmichael, Ozment, Al-Hegelan, Willifort, Peters, Noble, & Cheifetz, 2011).



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Significance of pathophysiology and implications for nursing practice

Acute respiratory distress syndrome is a complex and often fatal complication of acute lung injury. The predisposing factors to the development of ARDS vary, and include direct injury such as pneumonia, aspiration of gastric contents, pulmonary contusion or vasculitis, and inhalation injury; or indirect methods of lung injury such as pancreatitis, drug overdose, and nonpulmonary sepsis (Modrykamien & Gupta, 2015). Understanding the underlying pathophysiology of why a patient has developed pulmonary edema because of pancreatitis and sepsis may be difficult to "wrap your head around". That is why it is so important for medical and nursing staff to understand what is happening at the cellular level, so as to understand why the body is reacting in such a way, and why certain therapies, medications, and interventions are chosen to treat ARDS. Health care providers must be vigilant in their critical thinking to properly care of an ARDS patient. Physicians, mid-level providers, bedside nurses, and respiratory therapists must understand the signs and symptoms of ARDS, as well as the progression of disease, and be able to anticipate what a patient's needs may become. Thorough monitoring of vital signs, ventilator requirements, lab values, and radiographical imaging is crucial. Potential complications must be anticipated, such as pressure ulcers, poor nutrition and wound healing, muscle wasting from prolonged bedrest and possible use of neuromuscular blockades, and altered mental status from sedation medications and/or hypoxemia. If prone therapy is used, staff must ensure patient safety, and making sure all the invasive tubes, lines, etc. stay in place when repositioning.

Conclusion

Acute respiratory distress syndrome is a life threatening condition that requires aggressive treatment with close monitoring. Successful treatment of ARDS requires expert 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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