Nursing Student Class Projects (Formerly MSN)

Summer 2016

Acute Respiratory Distress Syndrome

Jessica L. Kaufman
Otterbein University, jessica.kaufman@otterbein.edu

Follow this and additional works at: https://digitalcommons.otterbein.edu/stu_msn

Part of the Medical Sciences Commons, and the Nursing Commons

Recommended Citation
Kaufman, Jessica L., "Acute Respiratory Distress Syndrome" (2016). Nursing Student Class Projects (Formerly MSN). 137.
https://digitalcommons.otterbein.edu/stu_msn/137

This Project is brought to you for free and open access by the Student Research & Creative Work at Digital Commons @ Otterbein. It has been accepted for inclusion in Nursing Student Class Projects (Formerly MSN) by an authorized administrator of Digital Commons @ Otterbein. For more information, please contact digitalcommons07@otterbein.edu.
Acute Respiratory Distress Syndrome
Jessica Kaufman, RN, BSN
Otterbein University, Westerville, Ohio

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 identify pathogen-associated molecular patterns (PAMPs) and non-enzymatically 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, 2015). 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 venules. All of these cytokines can also lead to tissue necrosis, apoptosis, and autophagy and essentially, damage to the alveolar epithelium and the pulmonary capillary endothelium (Ichiba, 2014; Siegel, 2015). 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 penetrate out of the vasculature, and when the oncotic pressure favoring fluid resorption is lost, the protein-dense fluid can flood the interstitial space (Siegel, 2015). 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 autophagocytosis (Siegel, 2015). The influx of fluid into the interstitium leads to a decrease in surfactant production causing an increase in alveolar wall thickness and a decrease in lung compliance (Chez, 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 or insult to the lungs (Chez, 2009). This early phase is characterized by uncontrolled inflammatory changes to the alveolar capillary membrane, and fluid and protein-dense blood, sometimes referred to as hematothorax, exudate, into the alveoli. Fibron and plasma proteins develop a fibrous membrane on the alveolar walls that impedes gas exchange and decreases lung compliance (Chez, Karger, Libbey, & Siegel, 2015). The second phase of ARDS is the proliferative (or organizing) phase, which develops 5-7 days after the initial insult, and can be characterized by the continued inflammation of the interstium and proliferation of type II alveolar cells (Chez, 2009). Damage to type II alveolar cells also makes surfactant inactive, contributing even more to atelectasis and decreased lung compliance (Drahnak & Custer, 2015). At the proliferative stage, granulation tissue becomes part of the alveolar septum and leads to organized fibrosis (Butt et al., 2016). The fibrosis stage is the final stage of the ARDS process, and is characterized by interstitial fibrosis, continued proliferation of type II cells, and chronic inflammation (Chez, 2009).

Treatments for acute respiratory distress syndrome can be as complex as the disease itself. Mechanical ventilation using low tidal volumes, lung protective ventilation, and the use of prone positioning are the most utilized treatments (Michaud, et al., 2014; Zampieri, Mendes, Ronai, Taniguchi, Alves, & Costa, 2015). In the event of refractory hypoxemia and respiratory failure despite the therapies mentioned above, extracorporeal membrane oxygenation (ECMO) may be utilized (Turner, Notter, Carmichael, et al., 2011; Zampieri, Mendes, Ronai, Taniguchi, Alves, & Costa, 2015). Extracorporeal membrane oxygenation for severe respiratory failure secondary to 2009 H1N1 influenza A Respiratory Care 59(5), pp140-148). Extracorporeal membrane oxygenation for severe respiratory failure in adult patients: A systematic review and meta-analysis of current evidence. Journal of Critical Care, 31(2), pp139-149.

References


Additional Sources


Significance of pathophysiology and implications for nursing practice
Acute respiratory distress syndrome is a life-threatening condition that requires aggressive treatment with close monitoring. Successful treatment depends on the knowledge from physicians, advanced practice nurses, bedside nurses, and respiratory therapy; all of whom must understand the complex underlying pathology and critical nature of this condition.