ARDS: Acute Respiratory Distress Syndrome

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ARDS Pathophysiology

The pathophysiology of ARDS primarily involves an initial lung injury followed by mechanisms that increase fluid from the pulmonary microcirculation to the alveoli (Fujishima, 2014).

Tissue Injury

Normally, physical barriers including endothelial and epithelial cell layers, the basement membrane, and the extracellular matrix help lungs maintain dry alveoli by regulating the passage of fluid from the pulmonary capillaries to the alveoli (Fujishima, 2014). Tissue injury disrupts this process and results in severe negative consequences, including:

• Destruction of the microvascular make-up allows leakage of blood products and proteins from the capillaries to the alveoli (Fujishima, 2014).
• With proteins leaking out of the capillaries, the oncotic gradient no longer favors reabsorption of fluid into the intravascular space (Hansen-Flaschen & Siegel, 2016).
• The blood of exudate into the airspaces perpetuates tissue injury leading to a further release of pro-inflammatory cytokines (Hansen-Flaschen & Siegel, 2016).

The Innate Immune System and Inflammation

The innate immune system plays a major role in the dysregulation of the inter- and intracellular molecules that increase vascular permeability and subsequent tissue damage, particularly the stimulation of alveolar macrophages and neutrophils. Once stimulated, macrophages recruit neutrophils and circulating macropoles to the site of injury where they perpetuate the inflammatory response by promoting the release of biochemical mediators such as proteases, reactive oxygen species, eicosanoids, phospholipids, and cytokines (Han & Mallampalli, 2015).

These cytokotic molecules cause tissue necrosis as well as the induction of detail cell death, including the destruction of type II epithelial cells. This is of particular importance since type II epithelial cells are responsible for maintaining surfactant, a substance that lines the alveoli, lowers surface tension and prevents alveolar collapse (Han & Mallampalli, 2015). Some of the inflammatory cytokines that are shown to be elevated in ARDS patients include IL-1β, TNF-α, IL-6, and IL-8. Also of note in the research into pattern recognition receptors (PRRs) and their involvement in the development of ARDS. Particularly the toll-like receptor (TLR) signaling pathway. It’s believed that hyaluronan, an extracellular matrix of glycosaminoglycan produced after tissue injury, initiates the inflammatory response in ARDS through TLR2 and TLR4 (Han & Mallampalli, 2015).

Significance of Pathophysiology

Impaired Gas Exchange

Damage to the endothelium allows a type of exudate into the lung parenchyma and eventually the alveolar airspace (Marino, 2014). The increased lung weight causes dependent zones of the lungs to collapse, leaving only non-dependent areas to remain open for ventilation (Gattinoni & Quental, 2016). The resulting short from the increased gasless areas and reduced lung size are the cause of the characteristic refractory hypoxemia seen in ARDS (Gattinoni & Quental, 2016).

Decreased Lung Compliance

Increased pulmonary artery pressure increases the mortality of ARDS (Naggert & Plans, 2014).

Increased Pulmonary Artery Pressure

Increased pulmonary artery pressure is another significant consequence of the ARDS pathophysiology. It may be caused by hypoxic vasoconstriction, positive pressure ventilation, parenchymal distortion, airway collapse, and hypercarbia (Siegel, 2016). Although cor pulmonale is rare, right ventricular dysfunction associated with pulmonary hypertension increases the mortality of ARDS (Naggert & Plans, 2014).

Conclusion

In the responsibility of the name, and the cost of the treatment team, to ensure the patient’s ventilation status while treating the underlying cause. With a deeper understanding of the pathophysiological process of ARDS, clinicians can better implement treatment plans to improve patient mortality.

Nursing Implications

These patients are typically going to require intensive care. It is the duty of the ICU nursing staff to understand the progression, treatment, and potential complications of this disease. The ARDS patient can be incredibly complex, requiring some of the most advanced nursing top skills apparent seen in the ICU, including, advanced ventilator management, pressure holes, advanced hemodynamic monitoring, vasoactive medications, and neuromuscular blocking agents (Modrykamien & Gupta, 2015). Of course, the ICU nurse cannot be expected to manage these patients alone. They must be adept in working as part of an interdisciplinary team, able to collaborate with physicians, respiratory therapists, and other members of the treatment team to ensure optimal therapy.

References


