2016

ARDS: Acute Respiratory Distress Syndrome

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Acute Respiratory Distress Syndrome: ARDS

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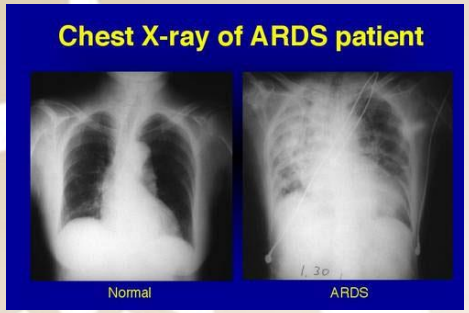
Introduction

In the 1960's, military physicians discovered a distinct hypoxic condition involving both lungs simultaneously (Modrykamien & Gupta, 2015). In Vietnam, this condition was referred to by physicians as "shock lung". Meanwhile, civilian physicians who encountered this condition termed it adult respiratory distress syndrome. The term was later modified to acute respiratory distress syndrome (ARDS) after determining that similar cases existed among all age groups (Modrykamien & Gupta, 2015).

ARDS develops after insult to the lung tissue. There are many conditions that can precipitate such an injury. Some of the more common precipitating factors include:

- Sepsis
- Aspiration
- Pneumonia
- Pancreatitis
- Trauma (Hansen-Flaschen &Siegel, 2016)

Despite advances in treatment of ARDS, incidence and mortality remain high. In the United States, ARDS has an estimated incidence of 190,000 cases per year and a mortality rate of 26% to 58% (Modrykamien & Gupta, 2015). In a recent study, ARDS represented 10.4% of total intensive care unit (ICU) admissions and 23.4% of all patients requiring mechanical ventilation. (Bellani et al., 2016).



(Image retrieved from https://www.homeofpoi.com/us/lessons/teach/Firebreathers-Lung-or-ARDS-11x52x198)

Signs and Symptoms

In order to meet the Berlin definition of ARDS, certain criteria must be met, including:

- an acute onset of hypoxemia
- radiographic imaging showing bilateral pulmonary infiltrates
- the event is not cardiogenic in nature (Hansen-Flaschen &Siegel, 2016).

The signs and symptoms of ARDS are related to these findings and include:

- dyspnea
- cyanosis
- diffuse crackles (Hansen-Flaschen &Siegel, 2016)
- arterial blood gas revealing hypoxemia
- eventual respiratory failure (Hansen-Flaschen &Siegel, 2016).

In addition to the symptoms related to respiratory distress and subsequent respiratory failure, ARDS patients will typically display symptoms of the precipitating disease process as well.

ARDS Pathophysiology

The pathophysiology of ARDS primarily involves an initial lung injury followed by mechanisms that increase pulmonary microvascular permeability and perpetuate further injury. The innate immune system and the inflammation process are key factors in this process (Fujishima, 2014).

Tissue Injury

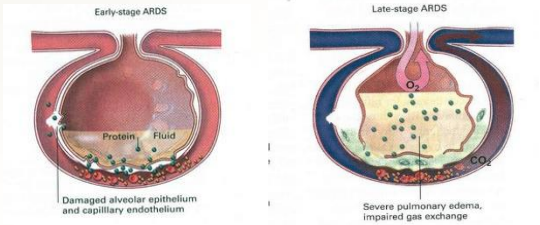
Normally, physical barriers including endothelial and epithelial cell layers, the basement membrane, and the extracellular matrix help healthy lungs maintain dry alveoli by regulating the passage of fluid from the pulmonary capillaries to the alveoli (Fujishima, 2014). Tissue injury disrupts this process 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 flood of exudate into the airspace perpetuates tissue injury leading to a further release of pro-inflammatory cytokines (Hansen-Flaschen & Siegel, 2016).

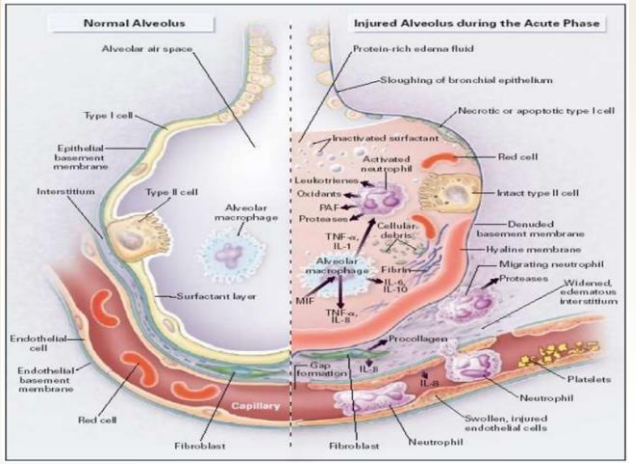
The Innate Immune System and Inflammation

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 macrophages 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 cytotoxic molecules cause tissue necrosis as well as the induction of distal cell death, including the destruction of type II epithelial cells. This is of particular importance since type II epithelial cells are responsible for making 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-1*B*, TNF-*a*, IL-6, and IL-8. Also of note is 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).



(Images retrieved from https://quizlet.com/20888959/pathophysiology-26-pulmonary-objectives-2356-flash-cards/)



(Image retrieved from http://www.slideshare.net/subodhchaturvedi1/ventillation-strategies-in-ards)

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 & Quintel, 2016). The resulting shunt from the increased gasless areas and reduced lung size are the cause of the characteristic refractory hypoxemia seen in ARDS (Gattinoni & Quintel, 2016).

Decreased Lung Compliance

Decreased lung compliance is a result of the activation of the coagulation cascade during the acute inflammatory process. It is triggered by the release of tissue factor from the damaged lungs (Marino, 2014). During the process, fibrin deposits form in the lungs and can undergo remodeling to produce pulmonary fibrosis (Marino, 2014). The fibrous tissue, combined with collapsed alveoli, decrease the compliance of the lungs and result in progressive respiratory insufficiency.

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 destruction, airway collapse, and hypercarbia (Siegel, 2016). Although cor pulmonale is rare, right ventricular dysfunction associated with pulmonary hypertension increases the mortality of ARDS (Nagelhout & Plaus, 2014).

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 therapy and equipment seen in the ICU, including, advanced ventilator management, pronation beds, 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.

Conclusion

As of now, the only treatment for ARDS is supportive care aimed at improving gas exchange. The standard of care is lung protective ventilation strategies including low-tidal volumes and utilization of positive end expiratory pressure (PEEP). As well as the use of more aggressive strategies including neuromuscular blockers and prone positioning. Although these therapies are currently the only treatments shown to improve mortality, efforts are being made to identify treatments aimed at modulating the inflammatory response.

It is the responsibility of the nurse, and the rest of the treatment team, to be aware of the disease process, associated complications, and the most up to date therapy options. As a team, the various disciplines can utilize their expertise to optimize the patients' ventilation status while treating the underlying cause. With a deeper understanding of the pathophysiologic process of ARDS, clinicians can better implement treatment plans to improve patient mortality.

References

Bellani, G., Laffey, J. G., Pham, T., Fan, E., Brouchard, L., Esteban, A., . . . Pesenti, A. (2016). Epidemiology, patterns of care, and mortality,for patients with acute respiratory distress syndrome in intensive care units in 50 countries. *JAMA: Journal of the American Medical Association, 315*(8). http://dx.doi.org.ezproxy.otterbein.edu/10.1001/jama.2016.0291

Chiumello, D., Coppola, S., Froio, S., & Gotti, M. (2016). What's next after ARDS: Long-term outcomes. *Respiratory Care, 61*(5). http://dx.doi.org.ezproxy.otterbein.edu/10.4187/respcare.04644

Fujishima, S. (2014). Pathophysiology and biomarkers of acute respiratory distress syndrome. *J Intensive Care, 2*(1). http://dx.doi.org/10.1186/2052-0492-2-32

Gattinoni, L., & Quintel, M. (2016). How ARDS should be treated. *Crit Care, 20*. http://dx.doi.org/10.1186/s13054-016-1268-7

Han, S., & Mallampalli, R. K. (2015). The acute respiratory distress syndrome: From mechanism to translation. *J Immunol, 194*(11). http://dx.doi.org/10.4049/jimmunol.1402513

Hansen-Flaschen, J., & Siegel, M. D. (2016, March 15). Acute respiratory distress syndrome: Clinical features and diagnosis in adults. Retrieved May 30, 2016, from http://www.uptodate.com/contents/acute-respiratory-distress-syndrome-clinical-features-and-diagnosis-in adults?source=search_result&search=ards&selectedTitle=1%7E150

Marino, P. L. (2014). *The ICU book* (4th ed.). Philadelphia, PA: Wolters Kluwer.

Modrykamien, A. M., & Gupta, P. (2015). The acute respiratory distress syndrome. *Baylor University Medical Center Proceedings, 28*(2). Retrieved from http://web.b.ebscohost.com.ezproxy.otterbein.edu/ehost/detail/detail?sid=89caa535-f82e-4bad-a29c-af5875dfb142%40 sessionmgr105&vid=4&hid=128&bdata=JnNpdGU9ZWhvc3Qtb G2ZSZzY29wZT1zaXRl#AN=109789201&db=rzh

Nagelhout, J. J., & Plaus, K. L. (2014). *Nurse anesthesia* (5th ed.). St. Louis, MO: Elsevier.

Siegel, M. D. (2016, March 15). Acute respiratory distress syndrome: Epidemiology, pathophysiology, pathology, and etiology in adults. Retrieved May 30, 2016, from http://www.uptodate.com/contents/acute-respiratory-distress-syndrome-epidemiology-pathophysiology-pathology-and-etiology-in-adults?source=search_ result&search=ards&selectedTitle=2%7E150

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