8-2019

Pathophysiology of ARDS

Brittney Nessle
brittney.nessle@otterbein.edu

The Pathophysiology of ARDS

Brittney Nessle, RN, BSN

Otterbein University, Westerville, Ohio

ARDS Patient Presentation

Introduction

Acute respiratory distress syndrome (ARDS) is a progressive life-threatening disease process, having an occurrence of up to 200,000 cases annually in the United States (Schreiber, 2018). The definition of ARDS has changed throughout the years. In 2011, the European Society of Intensive Care Medicine officially proposed the now adopted Berlin ARDS definition. The components within the definition include timing, chest radiography, the origin of edema, and severity of hypoxemia (Modrykamien & Gupta, 2015). There are over 50 different conditions that result in injury to the alveolar-capillary membrane that can lead to ARDS and produce several severe complications, including death. The diagnoses of ARDS can be difficult to determine, thus subsequently ending in a 30-50% mortality rate (Schreiber, 2018).

ARDS is a common finding in the Intensive Care Unit (ICU) at within hospitals in Columbus, Ohio, and in Intensive Care Units worldwide. It is essential for the nurses who take direct care of these patients to fully understand the mechanisms behind this complication, as early detection and treatment are imperative.

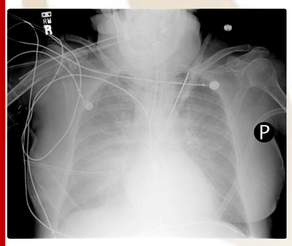
The purpose of this poster is to explain the presentation and pathophysiology behind ARDS and emphasize the nurse's role within prevention and treatment options.

Scenario

An 85 year old, obese, African American, male presented to the Emergency Department in Columbus, Ohio with shortness of breath, a productive cough, and periods of dizziness for over a week. The nurse assessed the patient as pale and diaphoretic. The patient did claim a history of pancreatitis, COPD, diabetes, hypertension, 30 years of smoking two packs of cigarettes a day, current everyday alcohol drinker, and receiving radiation therapy for esophageal cancer. Patient states that he has been having trouble swallowing certain foods since radiation.

Vital signs were obtained by the nurse, and the patient's heart rate revealed 130 beats per minute, oral temperature was 102.3 degrees Fahrenheit, blood pressure 90/42, oxygen saturation 85%, and respirations 38. The patient did appear to be using accessory muscles while breathing, therefore a nasal cannula was placed on 3L to obtain an oxygen saturation above 90%. The nurse quickly established IV access and obtained routine labs. A chest x-ray discovered bilateral pulmonary infiltrates (see image in bottom left corner). The arterial blood gas (ABG) showed: pH 7.12, pCO2 60, HCO3 26 determining the patient was experiencing respiratory acidosis. The patient's work of breathing started to increase after several hours, therefore the patient was rapidly placed on a mechanical ventilator and sent to the ICU for further management.

The patient was determined to be septic due to pneumonia from aspiration and in the proliferative phase of ARDS. Sepsis is the most common cause of ARDS (Modrykamien & Gupta, 2015). Over the next ten days in the ICU the medical team worked diligently to save this patient's quality of life (see nursing implication column). The nurses monitored the patient's vital signs, labs, and fluid status. The nurses flipped the patient from supine position to prone positioning. Approximately, two-thirds of patients with ARDS demonstrate better oxygenation and ventilation from prone positioning (Drahnak & Custer, 2015). However, the disease progressed, and the patient passed away. Mortality in patients experiencing sepsis with ARDS is 40.6%. The male gender, African American race, high body-mass index, and history of alcoholism are associated with a higher mortality rate (Modrykamien & Gupta, 2015).



Chest x-ray of patient showing bilateral pulmonary infiltrates

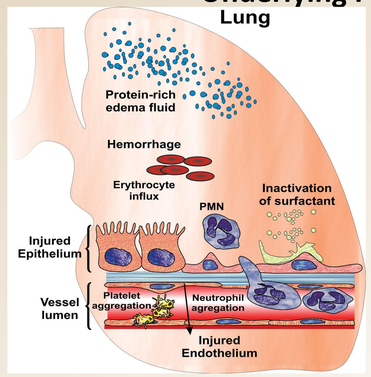
Image citation: Horlander, K. T. (2016). Imaging in Acute Respiratory Distress Syndrome. Retrieved July 19, 2019, from https://emedicine.medscape.com/article/362571-overview#a2

Clinical Manifestations

Phases	Exudative or Inflammatory	Proliferative	Fibrotic
Timeline	Within 72 Hours	4 to 21 Days	14 to 21 Days
Clinical Manifestations	▪ Dyspnea ▪ Tachypnea ▪ Tachycardia ▪ Pallor ▪ Mild Hypoxia ▪ Respiratory Alkalosis ▪ Bilateral pulmonary infiltrates on chest x-ray ▪ Diaphoresis	▪ Increased work of breathing ▪ Dyspnea ▪ Tachypnea ▪ Tachycardia ▪ Refractory hypoxemia ▪ Respiratory alkalosis ▪ Increased bilateral pulmonary infiltrates on chest x-ray ▪ Pulmonary hypertension ▪ Right heart failure ▪ Pulmonary fibrosis ▪ Hypercarbia	▪ Profound respiratory distress ▪ Critical refractory hypoxemia ▪ Cyanosis ▪ Mental status change ▪ White lung on chest x-ray ▪ Pulmonary hypertension ▪ Right heart failure ▪ Dense fibrotic tissue ▪ Hypercarbia ▪ Death

(Schreiber, 2018, p. 60)

Underlying Pathophysiology



Early stage of ARDS in the exudative phase is associated with diffuse alveolar damage, neutrophil infiltrate, hemorrhage and the accumulation of a protein-rich pulmonary edema

Image citation: Fudala, R., Krupa, A., Stankowska, T. (2010). Does activation of the FcγRIIa play a role in the Pathogenesis of the acute lung injury/acute respiratory distress syndrome? *Clinical Science,* 118(8). Retrieved from http://www.clinsci.org/content/118/8/519

ARDS is a progressive disease that can be categorized into three different phases according to the now adopted Berlin definition (Butt et al, 2016). See algorithm below for a quick version of significant pathophysiology events.

1. The first phase called the acute exudative phase begins one to seven days after the initial lung injury. After the injury occurs, the body releases a systemic immune response and triggers inflammatory mediators called cytokines. This response causes the pulmonary vasculature to become permeable and release extra fluid into the lungs. Fluid in the lungs increase, leading to other issues. Non-cardiogenic pulmonary edema occurs within the alveoli, causing the blood to leave the heart without being oxygenated (hypoxemia). At this point, increasing the oxygen dose does not improve overall oxygenation status (refractory hypoxemia) causing a lack of oxygen to the tissues (hypoxia). The surfactant in the lungs decrease and the alveolae begin to collapse causing compromised gas exchange. These events cause the patient's work of breathing to increase. Some patients can recover during this phase, but others will progress to the next phase.
2. The second phase called the proliferative phase happens during day seven to 14 after the initial injury. The compromised gas exchange gets worse, causing increasing refractory hypoxemia with tissue injury and scarring. As the body attempts to compromise, the vasculature constricts resulting in pulmonary hypertension and right sided heart failure.
3. The final phase of ARDS happens during days 14-21. It is called the fibrotic phase because the lungs are grossly compromised by thick pulmonary fibrosis. All previous mechanisms worsen and the patient begins to have multi organ failure. It is during this phase that the patient may require mechanical ventilation for a long period, and may never be able to recover (Schreiber, 2018).

Dyspnea and hypoxemia with poor response to oxygen supplementation

↓

Hyperventilation and respiratory alkalosis

↓

Decreased tissue perfusion, metabolic acidosis, and organ dysfunction

↓

Increased work of breathing, decreased tidal volume, and hypoventilation

↓

Hypercapnia, respiratory acidosis, and worsening hypoxemia

↓

Decreased cardiac output, hypotension, death

(McCance & Huether, 2018, p. 3732)

Significance of Pathophysiology

It is evident that early recognition of ARDS is crucial. If ARDS is detected and treated in the early phase, then that could prevent the disease from progressing and ultimately avoid death. Diagnosis is made due to evidence of lung injury, physical assessment, ABG analysis, and chest radiographs (McCance & Huether, 2018). It is critical that health care providers understand the underlying pathophysiology and diagnosis to pinpoint signs and symptoms of ARDS.

Implications for Nursing Care

- Frequent respiratory assessment
- Frequent evaluation of vital signs, especially respiratory rate, oxygen saturation
- Arterial blood gas interpretation
- Aseptic techniques and infection with any nursing care
- Assess for cardiac dysrhythmias and signs for decreased cardiac output
- Monitor routine labs and replace electrolyte imbalances
- Medication therapy may include but not limited to: antibiotics, bronchodilators, vasopressors, diuretics, anxiolytics, analgesics, sedatives, and neuro-muscular blockade.
- Manage mechanical ventilator with higher PEEP (Del Sorbo et al, 2017)
- Frequent aseptic oral care while on ventilator
- Dietary consult and manage possible tube feeding
- Blood product administration techniques
- Daily weight
- Strict input and output
- Prone positioning
- Elevating head of bed
- Managing secretions adequately
- Emotional support to manage fear and anxiety for patient and family involved
- Education on risk factors, including but not limited to smoking and alcohol sensation, and vaccinations

(Schreiber, 2018, p. 60)

Conclusion

Acute respiratory distress syndrome is a critical respiratory disease that progresses quickly through three different phases. If prevented or recognized early, the patient will experience a promising outcome. Additionally, there are current studies investigating new ways and strategies to treat ARDS. Some possibilities being tested include gene therapy, prophylactic immunotherapy, surfactant replacement, various immunotherapy mediators, stem cell replacements, among several other therapies (McCance & Huether, 2018). It is imperative that all heath care providers understand signs, nursing implications for early detection and ongoing advancements for the best treatment available.

References Cited

Butt, Y., Kurdowska, A., & Allen, T. C. (2016). Acute lung injury. *Archives of Pathology and Laboratory Medicine*, *140*(4), 345-350. https://doi.org/10.5858/arpa.2015-0519-RA

Del Sorbo, L., Goligher, E. C., McAuley, D. F., Rubenfeld, G. D., Brochard, L. J., Gattinoni, L., ... Fan, E. (2017). Mechanical ventilation in adults with acute respiratory distress syndrome. *Annals of the American Thoracic Society*, *14*, S261-S270. https://doi.org/10.1513/AnnalsATS.201704-345OT

Drahnak, D. M., & Custer, N. (2015). Prone positioning of patients with acute respiratory distress syndrome. *Critical Care Nurse*, *35*(6), 27-37. https://doi.org/10.4037/cnn2015753

Fudala, R., Krupa, A., Stankowska, T. (2010). Does activation of the FcγRIIa play a role in the Pathogenesis of the acute lung injury/acute respiratory distress syndrome? *Clinical Science,* 118(8). Retrieved from http://www.clinsci.org/content/118/8/519

Horlander, K. T. (2016). Imaging in Acute Respiratory Distress Syndrome. Retrieved July 19, 2019, from https://emedicine.medscape.com/article/362571-overview#a2

McCance, K. L., & Huether, S. E. (2018). *Pathophysiology: The Biologic Basis for Disease in Adults and Children* (8th ed.). St. Louis, MO: Elsevier/Mosby.

Modrykamien, A. M., & Gupta, P. (2015). The acute respiratory distress syndrome. *Baylor University Medical Center Proceedings*, *28*(2), 163-171.

Schreiber, M. L. (2018). Evidence-based practice. acute respiratory distress syndrome. *Medsurg Nursing*, *27*(1), 59-65.

Additional Sources

American Lung Association. (n.d.). Acute Respiratory Distress Syndrome. Retrieved July 25, 2019, from https://www.lung.org/lung-health-and-diseases/lung-disease-lookup/ards/

ARDS Global. (2019). ARDS Foundation. Retrieved from http://ardsglobal.org/

National Heart, Lung, and Blood Institute. (n.d). ARDS. Retrieved from https://www.nhlbi.nih.gov/health-topics/ards

OTTERBEIN UNIVERSITY