COPD: EMPHYSEMA

Nicole Nuxol
nicole.nuxol@otterbein.edu

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

Part of the Nursing Commons

Recommended Citation
https://digitalcommons.otterbein.edu/stu_msn/208

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 Master of Science in Nursing (MSN) Student Scholarship by an authorized administrator of Digital Commons @ Otterbein. For more information, please contact shickey@otterbein.edu.
COPD: Emphysema
Nicole Nuxol, RN BSN
Otterbein University, Westerville, Ohio

Introduction
Working in the Critical Care Unit at Grant Medical Center, many of our patients admitted to the unit have a history of Chronic Obstructive Pulmonary Disease (COPD), and the disease can complicate their course of treatment. Working with this patient population, it is common for these patients to experience prolonged time on the ventilator and increased length of stay. Many patients have multiple admissions throughout the year with COPD exacerbation, thus decreasing lung function and increasing risk for mortality.

Chronic Obstructive Pulmonary Disease is the third leading cause of death in the United States and is affecting more than 32 million Americans (Mosenifar, 2017). The presence of the disease continues to rise and many people are unaware they even have the disease. Prevention is essential in decreasing the incidence of the disease and Advanced Practice Nurses (APN) play a vital role. Chronic Obstructive Pulmonary Disease (COPD) is a lung disease characterized by progressive airflow limitation resulting from small- and medium-diameter airway disease and parenchymal destruction (Burt & Corbridge, 2011). The inflammation further weakens and destroys the airway. This decrease in airflow, in addition to the alveoli and narrowing of the airway (Burt & Corbridge, 2011). There are two types of COPD, chronic bronchitis, and emphysema. Emphysema is abnormal enlargement of airspaces distal to the terminal bronchioles, accompanied by destruction of the alveolar walls and without obvious fibrosis (Bokas, 2016).

Being able to understand the pathophysiology of emphysema will allow for providers to accurately assess patients and diagnose the disease. Educating patients and family members about the disease and providing preventative education such as risk factors to the disease will help prevent exacerbation of disease.

Pathophysiology

There are three classifications for emphysema. They are classified based off of the region of injury. The three types include; centriacinar, panacinar, and paraseptal (King Han, Dransfield, & Martinez, 2017).

• Centriacinar emphysema is mainly located in the upper lung zone and is the most common type of emphysema (Boka, 2016). This emphysema is specific to the proximal respiratory bronchioles with focal destruction (Boka, 2016). Long term cigarette smoking and dust inhalation are the most common causes of this type.

• Panacinar emphysema is mainly located in the lower half of the lungs. This type of emphysema is usually present in individuals who are alpha-1-antitrypsin deficient (King Han, Dransfield, & Martinez, 2017). In this type of emphysema, the entire alveolus is destroyed (Boka, 2016).

• Paraseptal emphysema is mainly in the distal airway structures, alveolar ducts, and alveolar sacs (Boka, 2016). The process of destruction occurs near the septae of the lungs or pleura and is at risk for spontaneous pneumothorax (Boka, 2016).

Signs & Symptoms

There are two types of emphysema, primary and secondary emphysema. Primary emphysema has a genetic component. Primary emphysema is an autosomal recessive trait of alpha-1 antitrypsin deficiency. Alpha-1 antitrypsin is a proteinase inhibitor and is made in the liver. This gene is a mutation in the SERPIN1 gene located in the 14th chromosome. This protein helps protect the lung from destruction. If not enough is made, then damage can occur and lead to emphysema (Bagurure Anariba, 2017). Secondary emphysema usually occurs by environmental factors, mainly from cigarette smoke, occupational and air pollutants (Boka, 2016). Below discusses the pathophysiology behind emphysema.

• Foreign antigens infiltrate the lung epithelial cell barrier (Boka, 2016). In this type of emphysema, the entire alveolus is destroyed (Boka, 2016). Proteases and free radicals are released from macrophages, which destroys the lungs epithelial barrier (Boka, 2016). Proteolytic enzymes are released mainly by macrophages, which destroys the lungs epithelial basement membrane (Boka, 2016). T lymphocytes in the alveoli are mainly CD8. These cells release chemotactic factors that recruit more cells such as cytotoxins. This leads to structural change to the lungs (Boka, 2016). Oxidative stress (produced from cigarette smoke and released from inflammatory cells) and protease (produced by inflammatory, macrophage, and epithelial cells) production further increases the inflammation (Boka, 2016). Proteolytic anti-proteinase imbalance causes bronchiolar edema and breakdown of lung elastin (Boka, 2016). Thus, causing elastic fiber damage to the parenchyma, known as emphysema (Burt & Corbridge, 2011). Shortness of breath, especially during light exercise or climbing steps

Significance of Pathophysiology

Understanding the importance of the pathophysiology and how it affects the individual will help providers to understand the disease of emphysema. Below is the significance of the pathophysiology of emphysema.

• Inflammation decreases the elastic recoil of the lung tissues and destroys alveolar attachments to small airways (Burt & Corbridge, 2011).
• Reduction in alveoli with septal wall destruction reduces gas exchange and causes the patient’s collapse during expiration, thus limiting airflow (Berg, Joanne, & Wright, 2016).
• Bronchial inflammation increases pulmonary secretions, muscle contraction, loss of alveolar structure and airway edema causes airway narrowing or obstruction (Burt & Corbridge, 2013). When the airway is narrowed or obstructed, inspiratory flow is decreased causing hyperventilation of lung from small shallow breaths. (Burt & Corbridge, 2013)
• Dysnea occurs because the lungs are being overworked (Burt & Corbridge, 2013). Individuals will have the appearance of “barrel-chested” due to too much air being trapped in the lungs (The Cleveland Clinic Foundation, 2017). Further parenchymal destruction impedes gas exchange and leads to hypoxia. (Burt & Corbridge, 2013)

Conclusion

APNs can play an important role in the management of emphysema. It is crucial for APNs to understand the pathophysiology of the disease so they can properly explain the significance it plays in helping individuals to better diagnose, treat, and manage patients. Educating individuals about the disease and staying up to date on treatment options will benefit both the APN and patient.

References
Martinez, R., & King Han, D. (2017). In this type of emphysema, the entire alveolus is destroyed (Boka, 2016). Proteases and free radicals are released from macrophages, which destroys the lungs epithelial barrier (Boka, 2016). Proteolytic enzymes are released mainly by macrophages, which destroys the lungs epithelial basement membrane (Boka, 2016). T lymphocytes in the alveoli are mainly CD8. These cells release chemotactic factors that recruit more cells such as cytotoxins. This leads to structural change to the lungs (Boka, 2016). Oxidative stress (produced from cigarette smoke and released from inflammatory cells) and protease (produced by inflammatory, macrophage, and epithelial cells) production further increases the inflammation (Boka, 2016). Proteolytic anti-proteinase imbalance causes bronchiolar edema and breakdown of lung elastin (Boka, 2016). Thus, causing elastic fiber damage to the parenchyma, known as emphysema (Burt & Corbridge, 2011). Shortness of breath, especially during light exercise or climbing steps

Significance of Pathophysiology

Understanding the importance of the pathophysiology and how it affects the individual will help providers to understand the disease of emphysema. Below is the significance of the pathophysiology of emphysema.

• Inflammation decreases the elastic recoil of the lung tissues and destroys alveolar attachments to small airways (Burt & Corbridge, 2011).
• Reduction in alveoli with septal wall destruction reduces gas exchange and causes the patient’s collapse during expiration, thus limiting airflow (Berg, Joanne, & Wright, 2016).
• Bronchial inflammation increases pulmonary secretions, muscle contraction, loss of alveolar structure and airway edema causes airway narrowing or obstruction (Burt & Corbridge, 2013). When the airway is narrowed or obstructed, inspiratory flow is decreased causing hyperventilation of lung from small shallow breaths. (Burt & Corbridge, 2013)
• Dysnea occurs because the lungs are being overworked (Burt & Corbridge, 2013). Individuals will have the appearance of “barrel-chested” due to too much air being trapped in the lungs (The Cleveland Clinic Foundation, 2017). Further parenchymal destruction impedes gas exchange and leads to hypoxia. (Burt & Corbridge, 2013)

Conclusion

APNs can play an important role in the management of emphysema. It is crucial for APNs to understand the pathophysiology of the disease so they can properly explain the significance it plays in helping individuals to better diagnose, treat, and manage patients. Educating individuals about the disease and staying up to date on treatment options will benefit both the APN and patient.