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Pathophysiology

Emphysema: The Silent Killer

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Scholarly Paper

Introduction

A slow, painful death is not wanted for any patient; especially not when the cause of the death is, in most cases, preventable. Approximately 3.1 million Americans have emphysema (National Vital Statistics, 2017). COPD (which encompasses emphysema) is the third leading cause of death in the United States (Hinkle, 2018It is difficult to determine exactly how many of those people die directly from Emphysema because most people die from an infection that is secondary to emphysema, or an infection that the patient was made susceptible to as a result of emphysema (National Vital Statistics, 2017). Regardless of the exact number of deaths, the damage to the lungs in the course of emphysema is permanent and causes pain and suffering to both the patient and the patient's loved ones. The disease will progress until the patient dies (Hinkle, 2018). Most cases of emphysema are caused by lifestyle choices, and could have been prevented with changes in living and environment (Capriotti, 2016). In 2014, 29.7 % of known COPD patients came to the emergency department with an exacerbation of emphysema (Hasegawa, 2014). A patient diagnosed with emphysema must be educated on the lifestyle choices they must make to accommodate the disease (Hinkle, 2018), and nurses must be prepared to both educate and treat the patient.

Anatomy and Physiology of Healthy Lung tissue

The lower respiratory system is composed of a series of "tubes" which eventually terminate in small pockets called alveoli (Hinkle, 2018). The conduction of the lungs, beginning at the trachea, is as follows: trachea, bronchus, bronchi, bronchiole, alveolar duct, and finally alveoli (Hinkle, 2018). Gas exchange and oxygenation of the blood occurs at the membranes of the alveoli in the lungs. Alveoli are small pockets of lung tissue that come in contact with deoxygenated blood, and allow oxygen from inspired air to diffuse through the alveolar

membrane into the blood (SUNNY, 2008). This contact also allows carbon dioxide to diffuse from the blood to the air in the alveoli, which is then exhaled. In a healthy lung, the alveoli form small, grape-like bundles at the end of each bronchiole. The bundle formation of the alveoli provides an increased surface area with which the capillary bed of the pulmonary vascular system may come in contact (SUNNY, 2008). The ratio of the amount of alveolar membrane that comes in contact with the pulmonary capillary bed is called the *ventilation/perfusion ratio* (V/Q ratio).

Pathophysiology and Etiology

Emphysema is one of the diseases encompassed within chronic obstructive pulmonary disease (COPD) (Capriotti, 2016). Any form of COPD involves a chronic inflammatory response in the lungs that slowly damages certain aspects of normal respiratory function (Capriotti, 2016). Emphysema describes an abnormal distension of the pulmonary tissue distal to the terminal bronchioles (Hinkle, 2018). This distension may take place in the alveoli or in the bronchiole branches themselves. Alveoli become enlarged when they are distended beyond their capacity, and the membranes between alveolar pockets rupture, combining alveolar pockets into one large sac (Hinkle, 2018). As the alveolar walls break down, the pulmonary capillary bed is reduced; this leads to increased dead space, decreased gas exchange, and increased resistance of pulmonary blood flow (which may lead to pulmonary hypertension and right sided heart failure (Capriottie, 2016).

There are four types of Emphysema; all types progress slowly over many years, and all types may occur in the same patient (Sverzellati, 2014). The four types are *panlobular*, *centrilobular, paraseptal* and *irregular* emphysema (Sverzellati, 2014). In panlobular emphysema, the respiratory bronchiole, alveolar duct and alveolus are destroyed, causing

essentially all airspace within the lobule to be enlarged. Panlobular emphysema impacts the whole lung evenly but usually begins in lung bases (Sverzellati, 2014). Centrilobular emphysema primarily effect the proximal bronchiole and alveoli of the upper lobes, leaving distal structures and lower lobes intact (until the disease progresses outward and downward)(Sverzellati, 2014). Paraseptal emphysema is named so because it targets the most distal lung structures, beginning around the septae of the lungs and pleurae. This type of emphysema progresses medially (Sverzellati, 2014). Irregular emphysema has no particular pattern or relationship to the lung structure (Sverzellati, 2014). Each of these types of emphysema have subtypes based on the way in which the alveoli are distended, which are as follows: bullous, giant bullous, focal dust, congenital lobar, and pulmonary interstitial emphysema (Sverzellati, 2014). However, for the sake of brevity, this paper will not delve into the subtypes of emphysema, but will focus on the larger picture of emphysema as a whole.

Smoking is the number one cause of most emphysema (Schuster, 2015). Inhaled cigarette smoke contains toxins that breakdown alveolar walls, kills pulmonary macrophages, and smothers ciliary cells which leads to the increase of mucous buildup and emphysematous changes in lung structure (Schuster, 2015). Environmental exposure to inhaled toxins such as corrosive chemicals, second hand smoke, and airborne toxins can also increase risk for development of emphysema (Hinkle, 2018). Certain individuals (about 1% of patients with emphysema) are also genetically predisposed to emphysema through a genetic mutation called *alpha-antitripsyn* (AAT) *deficiency* (Saito, 2004). AAT is a protein which stops the action elastase. Elastase is an enzyme produced by white blood cells in response damage or irritations (such as smoke or other inhaled toxins) (Saito, 2004). As a result, inflammation of the lungs is limited and damage to lung structures is controlled. When AAT is lacking, small amounts of

toxin cause sustained inflammation and significant damage can occur to the bronchioles and alveoli, even with slight irritation (Saito, 2004). Individuals with AAT deficiency usually develop emphysema or chronic bronchitis (another form of COPD) at an early age (Saito, 2004).

Clinical manifestation and diagnosis

Regardless of the type of emphysema, most patients present with similar symptoms, although the severity of signs and symptoms may vary depending on the progression of the patient's emphysema. The common symptoms include shortness of breath, dyspnea (especially upon exertion), decreased arterial oxygen saturation, cough, cyanosis, headache (especially upon waking), and weight loss (Hinkle, 2018). The common signs are barrel chest (AP diameter equal to or greater than transverse diameter), crackles, wheeze, diminished breath sounds, prolonged expiration, and pursed lip breathing (Hinkle, 2018). Common complications of emphysema are pulmonary hypertension and cor pulmonale (Hinkle, 2018). Pulmonary hypertension is a result of the decreased pulmonary capillary bed, as well as hypoxemia which causes vasoconstriction and increases vascular resistance (Hinkle, 2018). Cor pulmonale is right sided heart failure as a result of pulmonary hypertension. These complications may cause jugular vein distention and edema (Hinkle, 2018).

The primary diagnostic tests for Emphysema are chest x-ray (CXR) and pulmonary function test (PFT) (Hinkle, 2018). A PFT will reveal high total lung capacity, compromised expiratory flow and low ventilation/perfusion ratio (FEV1/FVC ratio of lower than 70%) (Capriotti, 2016). Emphysema will show hollowed out pocket of lung tissue on CXR, and may also show increased heart size due to right sided heart failure (Hinkle, 2018). If the CXR is inconclusive, a detailed CT scan of the lungs may be done to attain a better picture of the alveoli (Hinkle, 2018). A complete blood count (CBC) may reveal a high erythrocyte count and elevated

erythropoietin as the body attempts to compensate for the hypoxemia (Hinkle, 2018). An arterial blood gas measurement (ABG) will show increased CO2 levels (above 42 mm Hg), decreased oxygen (below 60 mm Hg), and possibly a lowered pH (below 7.35) if the patient has gone into respiratory acidosis (Hinkle, 2018).

Treatment

Once a patient has emphysema, the damage already done to the lungs is irreversible (Hinkle, 2018). However, symptoms can be treated and the progression of the disease can be slowed. The goal of treatment is to improve quality of life, slow or halt the progression of alveolar distension as much as possible, and maintain airway clearance and adequate gas exchange (Hinkle, 2018). These goals are accomplished through risk reduction, pharmacological therapy, pulmonary rehabilitation, and in some cases surgery (Hinkle, 2018). Risk reduction includes lifestyle changes that would prevent emphysematous changed in the lungs, such as avoidance of toxic fumes. Smoking cessation is the most cost effective and successful intervention in preventing emphysema (Schuster, 2015).

Pharmacological therapy is applied in a step-wise approach. Most patients are started on a short acting bronchodilator (such as albuterol) for mild emphysema, and progressing up to a combination of inhaled corticosteroids and bronchodilators (such as symbicort) for severe emphysema (Hinkle, 2018). Pulmonary rehabilitation involves at least 6 weeks of patient education and pulmonary exercises (Hinkle, 2018). The individual with emphysema is taught how to modify lifestyle around the limitations that come with emphysema, how to oxygenate themselves more effectively through diaphragmatic breathing and oxygen therapy, and how to recognize the signs and symptoms of acute exacerbations (Hinkle, 2018). A bullectomy (removal

of enlarged airspace) or lobectomy (removal of lobe of lung) may be performed for patients who do not respond to any other form of therapy (Hinkle, 2018).

Conclusion

Emphysema, although permanent, can usually be prevented. It is the nurse's job to educate every patient on the importance of smoking cessation and the risk of emphysema. COPD does not need to be the third leading cause of death in the United States; the nurses of the nation can change this. The nurses of America can also ease the burden of having a life-long disease by being fully informed of how to best care for and treat a patient with emphysema. The heart of nursing is holistic care; how better to holistically care for the client than to do everything possible to prevent their suffering, and diminish it once present. This is accomplished through educating both the nurse and the patient.

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