# Copd exacerbation



Case Study: COPD Exacerbation Jeffrey Sherman Due Date: January 16, 2012 Wayland Baptist University Adult Health II COPD Exacerbation The purpose of this paper is to discuss an exacerbation of Chronic Obstructive Pulmonary Disease (COPD) and its effect on my patient, Mr. HS, a 78 year old male. In this paper we will look at the various facets in the disease process including its incidence, pathophysiology, presenting complaints, analysis of his clinical presentation, and discuss treatment. We will analyze the effect the disease process has on Mr.

HS and will examine his clinical manifestations and laboratory work, as well as provide an outcome analysis. Understanding these various facets will enable one to understand various long term effects of this chronic disease process. Incidence COPD is defined as the persistent obstruction of the airways and occurs with emphysema, chronic bronchitis, or both. Cigarette smoking most commonly causes the disease process. Typically patients will develop a cough and become short of breath. In the United States, an estimated 15. 5 million people now suffer from this disease.

Statistics show that 13. 5 million people suffer chronic bronchitis and 2 million from emphysema (Workman, 2010). COPD is now considered the fourth leading cause of death in the United States and is projected to be the third leading cause of death for both males and females by the year 2020 (COPD Intl, 2004). COPD is second only to heart disease as a cause of disability that forces people to stop working (Wise, 2007). This disease process affects men more commonly then women and 95% of all COPD related deaths occur in people over the age of 55 (Wise, 2007).

Statistically, Men are 7 times more likely to be diagnosed with emphysema although the prevalence of in women is on a steady increase (COPD Intl, 2004). In a recent study, the median length of each hospital stay in patients with COPD was 4 days while mean hospital costs equaled \$5, 357 (Shore, et. al). Unfortunately, COPD has become a major cause of morbidity, mortality and disability in the United States (Manino, et al. , 2002) With increasing severity of COPD, the risk of respiratory tract infection also increases.

The impairment of the immune system is most likely responsible for both the colonization of respiratory tract with bacteria and for an increased risk for infection with new strains of bacteria causing acute exacerbations. Also lung infections like pneumonia, lung abscesses, and empyema are more often seen in patients with COPD than in healthy subjects (Lange, 2009). According to Griffen, et al, secondary viral lung infection in patients with COPD accounted for a 9% increase in mortality in patients over 65 years old.

Worldwide statistics are staggering. According to the World Health Organization, in 2005, COPD accounted for 5% of all deaths globally and that number is expected to rise. The World Health Organization projects an increase of total deaths worldwide form COPD by than 30% in the next ten years (WHO, 2011). Pathophysiology Chronic obstructive pulmonary disease (COPD) is a mixture of 3 separate disease processes that together form the complete clinical and pathophysiological picture.

It is characterized by progressive inflammation in the small airways and lung parenchyma, is mediated by the increased expression of multiple inflammatory genes (Barnes, 2009). The processes that are responsible for this disease are chronic bronchitis, emphysema, and, to a lesser extent, asthma. The specific pathophysiology of COPD is not well understood (Hunter & King, 2001). However, the progression of COPD is characterized by accumulation of inflammatory mucous exudates in the lumens of the small airways in the lungs, which are known as bronchioles.

Chronic inflammation causes a thickening of their walls resulting in infiltration by adaptive and innate inflammatory immune cells. According to Barnes, activity of the gene histone deacetylase 2 (HDAC2) is reduced in peripheral lung and in alveolar macrophages, resulting in amplification of the inflammatory process. This causes the airways to become hyperactive, creating constriction and narrowing of the airways excessively. Infiltration of the airways with substances such as polynuclear, mononuclear phagocytes, and CD4 T cells increases with each stage of disease progression.

The entire chronic inflammatory process, occurring in the lungs is associated with tissue repair and remodeling that ultimately results in COPD. COPD is seen commonly in patients who smoke. It appears that smoking may overcome the body's natural mechanisms for limiting this immune response. The process may also continue in susceptible individuals even after smoking cessation. In this case, even if the original noxious insults are removed, the continuation of COPD may develop and is characterized by progressive accumulation of cells of the immune system, fibrosis, and mucus hypersecretion.

The molecular basis for the lung inflammation seen in COPD is still an area of great research and debate, with the potential roles of cytokines, complex

autoimmune processes, and immune modulation from chronic infection all under investigation (Shapiro, 2003). The defining feature of COPD is irreversible airflow limitation during forced expiration. This may be a result of a loss of elastic recoil due to lung tissue destruction or an increase in the resistance of the conducting airways.

The standard measure of COPD is the measure of forced expiratory volume in 1 second (FEV1) and its ratio to forced vital capacity (FVC), FEV1/FVC. COPD exacerbations are linked to diminished health status and can be life threatening, particularly in patients with advanced disease (O'Donnell & Parker, 2006). Analysis of Clinical Presentation Mr. HS is a 78-year-old retired male, who presented to the emergency room at Northeast Methodist Hospital initially on February 11, 2011, with complaints of shortness of breath and coughing.

He was diagnosed as having a COPD Exacerbation and was placed on antibiotic therapy and was released home. He was also advised at that time to complete the entire course of antibiotics and return to his primary treating physician if his condition did not improve. He reported that he was compliant with is treatment regimen and after the 10 day course of antibiotic therapy, his condition, however, did not improve. He subsequently returned to the emergency room on February 21, 2011, with ongoing chest pain, shortness of breath, and a productive cough.

He was evaluated at that time the Northeast Methodist's emergency room and was admitted. His antibiotic therapy was changed to IV Clindamycin and Azithromycin and he was referred to pulmonary specialist, Alison Brown, MD for consultation on February 24, 2011. Following consultation, blood studies were ordered which showed an ongoing elevated white cell count. Blood cultures showed no growth. Influenza antigens were negative and sputum gram-stain showed many white blood cells with normal upper respiratory tract flora.

Chest x-ray showed previous coronary artery bypass surgery, a pacemaker and diminished lung volumes and infiltrates in each costophrenic sulcus. Dr. Brown felt that the organism was a community acquired infection, most likely streptococcus or hemophilus influenza. She agreed that azithromycin and clindamycin were appropriate and could be changed to oral. He was placed on Cleocin (clindamycin) 300 mg PO, q6h and Zithromax (azithromycin) 500 mg PO, daily. She also ordered breathing treatments be initiated. Mr. HS continues to be symptomatic with ongoing chest pain, shortness of breath, and cough.

His past medical history is significant for coronary artery disease, status post coronary artery bypass surgery, history of atrial fibrillation, status post pacemaker placement, history of atypical chest pain, chronic obstructive pulmonary disease, and bronchial atelectasis. He is diabetic, Type 1. Mr. HS is married and has two grown children ages 34 and 38, both are female. He states that in his entire life, he has smoked only six cigarettes. Analysis of Clinical Findings Mr. HS was admitted to Main Methodist Hospital MICU. At the time of his examination, he was alert and oriented X3.

He was placed on 2 liters nasal cannula with O2 saturation at 96%. Lung sounds showed crackles in the right and left lower lobes and he was audibly wheezing. His breathing was labored and shallow. Peripheral pulses were +2 and strong. His temperature was 97. 6 deg. F. , pulse rate 81 bpm, respiration 20 breaths per minute, and blood pressure 131/72 mmHg. He weighs 198. 5 lbs (90. 2 kg) and is 5' 10" tall. Bowel sounds were normal and he reported having one bowel movement per day. His blood glucose is 121. There were no signs of clubbing in the fingers.

He was placed on fall precautions, and was unable to perform activities of daily living. He does not utilize any assistive devices. His gait was slow and guarded and he sleeps 5 hours per night, interrupted. Analysis of Lab Values and Diagnostic Tests Labs were performed on Mr. HS to help derive additional information and gain a better understanding of what could be the cause of his ongoing condition. He has a history of COPD and it appears that as a result of an exacerbation of his COPD, he has developed a communityacquired infection in his lung. Mr. HS lab values were as follows: CBC: Hbg/Hct - 15. mg/dl / 44/9 %; WBC - 16. 5 X 103/ul ; Sodium – 135 mEg/L; Chloride – 101 mEq/L; Potassium – 4. 6 mEq/L; PaCO2 – 45 mmHg; pH – 7. 34; Platelets - 179 X103/L; PT - 29. 5 sec; INR - 3. 0. His gram stain showed many white blood cells. Sputum culture was negative. CT of the chest showed bibasilar dependent atelectasis and infiltrate. Reviewing his lab and special studies, Mr. HS has an elevated white blood cell count which is indicative of the infectious process. The sputum gram stain showed many white blood cells but was not differential and could not specifically identify the organism.

His sputum culture was negative and once again the organism could not be identified. The CT scan was abnormal confirming the presence of infiltrate in https://assignbuster.com/copd-exacerbation/

the both lower lobes of the lungs. This would be consistent with a lung infection, progressing bibasilar atelectasis, and could indicate the possibility for aspiration. When reviewing the lab work, additionally, we see borderline abnormalities in acid/base balance. His pH is on the lower end of normal and his PaCO2 is at the lower end of normal indicating the potential for respiratory acidosis. At this point, it would seem that Mr.

HS as developed an exacerbation of his COPD and had developed a community-acquired infection, most likely streptococcus or hemophilus influenza that is contributing and complicating his symptoms. He is at risk for respiratory acidosis. Treatments At the time he was admitted and treated at the hospital, he was already taking the following medications: Fluticasone/Salmeterol Advair, 2 Puffs, PO BID for Respiratory Inflammation; Montelkast Sodium/Singular, 10 mg, PO HS for Chronic Asthma; Azelastine HCL/Astelin, 1ml, Each Nostril, daily for Allergic Rhinitis; Albuterol Sulfate/Proventil, 2. mg, PO q4h for COPD; Ipratropium Bromide/Atrovent 0. 5 mg, PO q4h also for COPD. Following evaluation initially at the Emergency Room, Mr. HS was placed on Clindamycin HCL/Cleocin, 1500 mg IV BID, and Azithromycin/Zithromax, 500 mg IV q24 and was admitted to the MICU Unit. There, the course antibiotic medications were continued and a pulmonary consultation was requested. Following further evaluation by Dr. Brown, a pulmonologist, his antibiotic regimen was changed to oral (Cleocin, 300 mg PO g6H and Zitromax 500 mg PO at Lunch) and breathing treatments with a nebulizer were ordered.

From a nursing standpoint, the treatment plan should be directed at ensuring compliance with his present prescribed regimen in order to control https://assignbuster.com/copd-exacerbation/

the infection and prevent further deterioration in his pulmonary condition. Long term planning should be aimed at helping him to achieve optimal wellbeing through knowledge, improved pulmonary function, and prevention of infection recurrence. Once the nursing treatment goals have been set, it will necessary to evaluate that each of the goals have been met. Initially as part of his nursing care plan, it will be important for Mr.

HS to demonstrate proper deep breathing and coughing techniques. He will demonstrate proper use of incentive spirometer will be compliant with his medication regimen. Within 48 hours, we will expect that Mr. HS will have improved airway clearance as evidenced by improved lung sounds. We would expect that his lung fields are clear to auscultation and that is pH and PaCO2 are stable. Additional teaching would be to encourage cessation of smoking or smoke exposure. Outcome Analysis By the second day of his hospitalization and through compliance with his treatment program, Mr.

HS had reported improvement. He noted that he was able to clear his lungs the night before and was able to sleep 6 hours through the night undisturbed. Auscultation the following morning showed his lung fields to be clear and he was quite happy and encouraged to continue his prescribed treatment program. Following completion of his medication regimen, repeat CBC will show normal levels of White Blood Cells and his repeat chest x-ray and CT study will be free from any evidence of infectious process at the time of his discharge.

Once he has been discharged, it will be important that he follow the longterm care plan to help prevent recurrence of infections process. Additionally,

the plan will help to ensure that his pulmonary condition does not continue to deteriorate. Having the client and nursing staff formulate and accept realistic short and long-term goals will help him realize that he does have control over his life and condition. This will help to allow him to make appropriate choices to improve the quality of his life. By following the nursing care plan, Mr.

HS will be capable of controlling his dyspnea and will remain free from infection. He will understand his diagnosis of COPD as well as his prescribed treatment regimen. He will be compliant with his medications and will be able to properly demonstrate the breathing exercises and other pulmonary rehabilitation procedures that he was taught. He will institute these measures to ensure that his longs remain clear and that he remains free from infection. Mr. HS will understand and verbalize symptoms associated with worsening of his condition and will agree to report to his primary care physician if his condition deteriorates.

According to the U. S. Department of Health and Human Services' National Guideline Clearing House, this patient was treated appropriately with oxygen therapy, bronchodilator therapy, glucocorticosteroids, antibiotics, and lifestyle modification. Conclusion Chronic Obstructive Pulmonary Disease is debilitating disease that has become increasingly epidemic affecting an estimated 15. 5 million people in the United States. Death rates continue to climb and by 2020, COPD is projected to account for 5% of all deaths worldwide.

Due to the chronic inflammatory processes involved with this disease, patients are at increased risk for infection. Certainly, as we have seen, Mr. HS is no exception. As a result of his pathological lung process, he developed a secondary lung infection requiring him to be hospitalized and placed on antibiotic medication and respiratory therapy. The infection complicated his underlying COPD condition creating a dangerous and potentially deadly disease process. Secondary lung infection in older patients with COPD is very serious causing an increase in mortality in the elderly population.

Quickly presenting to the emergency room, following up with his physician, and being compliant with his treatment regimen was instrumental in his ability to recover relatively quickly from his COPD exacerbation. Fortunately, he was placed on an appropriate medication regimen and his condition improved. The nursing plan implemented was designed to help control his infection and help him to understand his primary disease process, COPD. The focus on both his short-term and long-term goals will help him to cope realistically with his ondition, and to implement appropriate therapies that will allow him achieve optimal health, preventing further deterioration of his condition, and avoid recurrence of a secondary infections process. References Barnes, P. J. (2009). Role of HDAC2 in the Pathophysiology of COPD. National Heart and Lung Institute: Annual Review of Physiology. 71: 451-64. Carpenito-Moyet, L. J. (2009), Chronic Obstructive Pulmonary Disease. Nursing Care Plans & Documentation, 5th Edition (pp 125-137). Philadelphia, PA: Lippincott Williams & Wilkins. COPD International. (2004). COPD Statistical Information. Retrieved from http://www.opd-international. com/library/statistics. htm Griffen, M. R. , Coffey, C. S. , Neuzil, K. M. , Mitchel

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