

Chronic Obstructive Lung Disease

Purpose: This course is designed to give an overview of chronic obstructive pulmonary disease (COPD). Focus will be placed on the pathophysiology, causes, signs and symptoms, diagnosis, staging and medical management. The course will also look at surgical options, pulmonary rehabilitation, preventative measures, prognosis and exacerbations. A focus on the medications used to treat the disease in different stages will be presented.

Objectives

- Differentiate between chronic bronchitis and emphysema
- Describe the relationship between smoking and chronic obstructive pulmonary disease (COPD)
- Review the pathophysiological changes that occur in COPD
- Define the four different stages of COPD
- Discuss treatment options for each stage of COPD
- List three major classes of drugs used to treat COPD
- Discuss three surgical options available for the treatment of COPD
- Discuss three nursing diagnoses that assist in caring for the patient with COPD
- List three nursing responsibilities when caring for a patient with COPD

John P is a 58 year-old white male who presents to his primary care doctor's office in late January with a chest cold he "just can't shake". "What is wrong with me doc, this is the third year I got a cold I just can't get rid of."

The cough and chest congestion first presented in late October at which time he was treated with amoxicillin and bronchodilators. He felt better after this course of treatment, but the cough never went away. It got worse in early December at which time he was treated with azithromycin and an oral steroid taper.

He is currently afebrile, has a productive cough of thick yellow sputum, which clears as the day progresses. He reports not sleeping well, being short of breath on exertion and is fatigued during the day. The ears, nose and throat exam are unremarkable. There is no jugular venous distention. The lungs have scattered rhonchi and wheezes throughout.

His past medical history is positive for hypertension and hyperlipidemia. He has never had a surgery. His current medications include: lisinopril and simvastatin. He drinks socially, has smoked one to two packs of cigarettes a day since the age of 17, and denies the use of illicit drugs.

Chest x-ray reveals no active disease and lab work – including a complete blood count and metabolic panel - is unremarkable.

Chronic obstructive pulmonary disease (COPD) is a broad term describing diseases of the lungs associated with limited airflow. It is the fourth leading cause of death – and likely the third leading cause of death within the next 10 to 15 years. Chronic bronchitis affects 8.9 million community dwelling people and 3.8 million are affected by emphysema¹. In the United States the prevalence of serious COPD is 10.1 percent². The costs – mostly attributed to hospitalization - associated with COPD are high. In 2002, the United States spent 18 billion dollars directly and 14.1 billion dollars indirectly to fight COPD³.

Pathophysiology

Most cases of COPD are a combination of emphysema and chronic bronchitis and involve changes in the large airways, the small bronchioles and the lung parenchyma (4).

Leukocytes and macrophages destroy the lungs faster than the body can heal them. In addition, oxidative stress caused by free radicals in cigarette smoke and other irritants to the lungs, leads to destruction of lung cells.

In emphysema, the walls between many of the alveoli are destroyed, leading to a few large air sacs instead of many tiny ones. The alveoli are stretched out and are unable to effectively transfer oxygen and carbon dioxide. This decreased ability to release carbon dioxide from the blood contributes to shortness of breath.

Emphysema has three patterns. The first type destroys respiratory bronchioles and the central portions of acinus. It is most severe in the upper lobes and caused by cigarette smoking. The second type is common with alpha-1 antitrypsin deficiency and typically affects the lower lungs. The third type is not common and leads to the bullae formation⁴.

Chronic bronchitis is characterized by excessive mucous production and chronic inflammation of the breathing passages. The most common histological feature is enlarged mucous glands. Hallmark symptoms are cough with mucous production and difficulty breathing. The presence of chronic productive cough for at least 3 consecutive months in 2 consecutive years is necessary for the diagnosis of chronic bronchitis. Early in the disease the mucous can be coughed out but as the disease progresses the cough mechanism weakens and mucous can not be coughed out as well. This leads to mucous pooling, which is a breeding ground for bacteria. In addition, excessive mucous in the breathing passages results in the body being unable to get in enough oxygen or eliminate enough carbon dioxide⁴.

COPD starts years before it is diagnosed, damages the airways and decreases the amount of air that one can get in and/or out of the lungs. These limitations are not completely reversible, progressive and associated with inflammation of the lungs.

Causes

Causes of COPD are genetics, environmental exposure (especially smoking) and age. Smoking causes greater than 75-90% of the cases of COPD⁵ but only 15% of smokers develop COPD⁴. Scientists are currently unable to describe why certain individuals with exposure to smoking, environmental or occupational toxins develop COPD and others do not.

Pollution and occupational exposures (dust and chemicals) are environmental factors associated with COPD. Being male, older age, respiratory problems, as a child and low socioeconomic status are known risk factors associated with COPD. It takes 20-30 years of exposure before signs and symptoms of COPD are noted.

Alpha-1-antitrypsin deficiency is a genetic defect that results in a decrease in an enzyme named elastin, which is an essential enzyme that stops the body from eating away healthy lung tissue. When this genetic deficit is present, the lung gets destroyed at fast rates and emphysema starts at an early age. Emphysema typically starts at the age of about 53 for non-smokers with this deficiency and 40 for smokers⁴.

Symptoms

Cough, increased sputum production, shortness of breath and wheezing are common symptoms of COPD. Symptoms vary depending on whether chronic bronchitis or emphysema predominates. A history of asthma as a child, frequent pneumonia and frequent sinus infection are common in those with COPD.

Symptoms of emphysema include shortness of breath, fatigue, pursed lip breathing, and weight loss. Pure emphysema is characterized by dry cough without sputum production. Individuals with emphysema are typically thin, have a round chest and a nice pink color to their skin.

Symptoms of bronchitis include cough with mucous production especially in morning, frequent respiratory infections and occasional shortness of breath. Mucous volume is initially small and is clear to slightly off colored. As the disease progresses sputum becomes thicker and more off-colored. Late problems associated with bronchitis include: decreased oxygen levels, edema and respiratory failure. Those with bronchitis are typically robust and have a more dusky appearance to the skin.

Diagnosis

The diagnosis of COPD is based on history and diagnostic testing. Physical exam is not very helpful in the diagnosis of COPD. Limited airflow, as evidence by decreased breath sounds, is not present until late in the disease and has a low sensitivity and specificity. As the disease progresses other features present, including: tachypnea, respiratory distress with simple activities, cyanosis, elevated jugular venous pulse (JVP), peripheral edema, thoracic hyperinflation, decreased breath sounds, wheezing, hyperresonance on percussion and prolonged expiration.

Chest X-rays rule out other diseases - such as pneumonia or heart failure – but are not diagnostic of COPD, unless obvious bullous disease is present. Hyperinflation may be seen on x-ray.

Computed axial tomography (CAT) scans, though usually not done to diagnosis COPD, help confirm the diagnosis and rule out other lung diseases. While it is highly specific to COPD, it is often not done because its use does not alter the disease course or treatment decisions.

Lung function tests – spirometry - are the gold standard in the diagnosis of COPD. Unfortunately, it is underused. Only 32 percent of those with COPD have spirometry testing and only half of those patients have bronchodilator testing to confirm the diagnosis⁶.

Spirometry measures lung capacity and helps classify COPD into one of four stages (Table 1). It determines forced vital capacity (FVC) and forced volume capacity in one second (FEV₁). While sitting down the patient blows into a tube as hard and as long as possible. People without COPD, blow out most of their air in one second, but COPD patients do not. The FEV₁ defines the severity of COPD. The lower the percentage of predicted the more severe the disease is. The ratio of the amount air of blown out in one second (FEV₁) to the total air blown out (FVC) is reported in terms of a percentage and COPD patients have a level below 0.70.

Arterial blood gases are useful in advanced COPD and should be used in those with FEV₁ of less than 50% of predicted or have respiratory or right heart failure.

Other diseases - congestive heart failure, heart disease, hypertension and arrhythmia –

are associated with COPD and worsen the prognosis. These diseases need to be sought in those with COPD, as optimal treatment of these diseases improves quality and quantity of life.

Differential Diagnosis

When typical signs and symptoms of COPD present other diseases processes need to be considered. Asthma can be difficult to differentiate, but it is typically characterized by significant improvements on pulmonary function tests after the patient is given a bronchodilator. Congestive heart failure typically presents with rales or crackles, edema and weight gain. Chest x-rays and certain laboratory tests (brain natriuretic peptide) help rule out heart failure. Bronchiectasis is characterized by large amount of purulent sputum, coarse crackles, and clubbing. Again a chest x-ray is helpful⁴.

Case Study

Given the signs and symptoms John is likely afflicted with COPD, bronchitis predominate. The physician orders a chest x-ray to rule out an occult pneumonia or another cause of cough and pulmonary function tests. The chest x-ray is unremarkable and blood work shows a complete blood count, metabolic panel and brain natriuretic peptide all within normal limits. His pulmonary function tests reveal his FEV₁ to be 60% of what is predicted. He is diagnosed with COPD in the moderate stage.

Treatment

No treatment reverses the process of COPD, but treatments exist to control the signs and symptoms. Treatment goals for COPD are to maintain open airways, decrease airway constriction, decrease signs and symptoms, prevent disease progression, prevent exacerbations, improve overall health, decrease mortality, preserve function and prevent hospitalizations.

Stopping smoking will hasten the progression of the disease. Non-smokers lose 15 ml/year of FEV₁ while smokers lose 60 ml/year⁵.

Given the chronic nature of the disease, stepping down the medications is not typical in COPD once symptoms are controlled. As the disease progresses, medications need to be added to control symptoms⁷.

Pulmonary Rehabilitation

Pulmonary rehabilitation, which includes exercise, nutritional support, social support and education, is an essential step in improving patient function. It helps the patient understand the disease process, treatment options and develop coping strategies. Its goals are to improve baseline level of physical activity, reduce symptoms, decrease risk of hospitalization and improve quality of life. Pulmonary rehabilitation added to standard therapy improves function and symptoms by helping patients increase activity through exercise training. The use of long acting anticholinergic drugs improves the effectiveness of exercise in pulmonary rehabilitation⁸.

Exercise

Exercise training is an essential component to pulmonary rehabilitation, but it should be continued after formal pulmonary rehabilitation ceases. The goals of exercise training are to improve functional ability and reduce dyspnea. When helping the COPD patient

develop an exercise plan it is important to consider a program that is safe, balanced and effective.

After obtaining physician approval for starting an exercise program, incorporate an appropriate warm-up and cool-down into each exercise session. Five to ten minutes of light activity helps assure that the muscles, bones, heart and lungs are gradually warmed-up and cooled-down. An example of a warm-up/cool-down may include 5-10 minutes of light walking followed by some stretching.

The exercise plan should incorporate aerobic exercise, resistance training and flexibility training.

Aerobic exercise prescription can be broken down into frequency, intensity, time and type of exercise. There is no cookie cutter recipe for exercise prescription. But those with more severe disease or a high level of deconditioning will start out at a much lower level. Gradually working into an exercise program is important to prevent soreness and injury.

Aerobic exercise should be carried out three to seven times a week. Exercising three times a week with one day rest between exercise sessions is a good starting goal. After a few weeks increase the frequency to 4 days a week. Adding one day a week every month assures the body adjusts to the demands exercise puts on it without resulting in undue soreness, injury or psychological burnout.

A simple method to measure intensity is the talk test. Exercise to a point where breathing is increased but a conversation can be had without gasping for air.

When initiating an exercise program the duration should be kept minimal with the goal of gradually increasing the duration. Start out exercising 10-15 minutes a day is a logical place to start with the addition of 5-10 minutes a week. The eventual goal should be exercising for 30-60 minutes a day. Breaking up the session into intervals is another option. Incorporating 3-ten minute exercise sessions each day will provide the same benefit as exercising for one-30 minute session.

The last component to an aerobic exercise program is the type of exercise. Good aerobic exercises include walking, biking, exercise classes and swimming. Many considerations must be kept in mind when deciding which mode of exercise to choose. Personal preference is an important consideration - exercises that are enjoyable will be complied with much better.

The goal of strength training, for health purposes, is to improve quality of life. While weight training increases the amount and quality of muscle, the main purpose of the training program for a COPD patient is to improve the ability to function in day-to-day life and reduce disease.

Each major muscle group in the body should be stretched. Stretch to a point of minimal discomfort and hold for 10-30 seconds. The duration of the stretch is more important than the intensity of the stretch when it comes to improving flexibility. Do not stretch to the extremes; this will increase the risk of injury.

In order to minimize injury, stretch a warm muscle. Warming up the muscles by performing ten minutes of aerobic exercise (such as light walking) prior to stretching.

Medicines

Medications in COPD improve quality of life, sleep quality, exercise tolerance and survival while reducing exacerbations, dyspnea and hospitalizations, but no medications modifies the disease⁹.

Bronchodilators

Bronchodilators are critical to symptom management. Inhaled bronchodilators, medicines that open up the airways, are usually the first medicines used in the treatment of COPD. They are typically inhaled but some formulations are oral. Inhaled bronchodilators are used on an as needed basis for symptoms or on a regular basis to prevent or reduce chronic symptoms⁷. They help improve exercise capacity without changing the natural course of the disease or lung function.

Two common short acting bronchodilators are ipratropium bromide and albuterol. They are typically given either with a puffer with a dry powder, metered-dosed or by nebulizer. Beta₂ agonists can be given as an oral formulation.

Short-acting beta₂ agonists (albuterol) have an onset of action within 5 minutes with a peak effect in about thirty minutes and a duration of about 4 hours. Short-acting beta₂ agonists improve breathlessness in COPD, but they do not improve exercise tolerance⁹. They are recommended in Stage I disease on an as needed basis or regularly to improve chronic symptoms.

Short-acting ipratropium bromide (Atrovent) bronchodilates in about 15 minutes. The medication lasts about 6 hours but the effectiveness starts to wean down after about 2-3 hours⁵. It is commonly used and is typically dosed 4 times a day.

The long-acting bronchodilators (Salmeterol [Serevent] and formoterol fumarate [Foradil] or tiotropium [Spiriva]) keep the breathing tubes opened for longer periods of times but have a slower onset of action and should not be used for quick relieve. Long-acting bronchodilators are more convenient as they only need to be given once or twice a day as opposed to four to six times a day for the short-acting form.

Long-acting beta₂ agonists (Salmeterol [Servent] and formoterol fumarate [Foradil]) demonstrated a small improvement in FEV₁ but did not demonstrate any improvement in breathlessness¹⁰. Inhaled anticholinergic medications (tiotropium) reduce the rate of COPD exacerbations, reduced hospitalizations and reduce mortality risk¹¹.

The most common side effects for anticholinergic medications are dry mouth. Other side effects are constipation, increased heart rate, blurred vision, glaucoma and urinary retention. Beta₂ agonists have the potential to cause cardiovascular side effects. The most serious being tachyarrhythmias and hypokalemia. Other side effects include: tremor, nervousness, dizziness and headache.

Combined ipratropium and albuterol may help those who require more than one bronchodilator. Each bronchodilator works through a different mechanism and the combination of two or three typically results in improved clinical benefit. The most common combination is albuterol and ipratropium. Those who receive both albuterol and ipratropium had less wheezing, shortness of breath and improvements in FEV₁ when compared to those who got albuterol alone¹².

Theophylline, given as a pill, is a bronchodilator and can increase the strength of the diaphragm muscle. Minimal benefits were demonstrated with this medication except for a small improvement in FEV₁⁹. It is not a first line agent in COPD because it has many

side effects, many drug-to-drug interactions and requires a very specific blood level to avoid toxicity. The blood levels need to be monitored. Side effects of theophylline include nausea and increased heart rate, arrhythmias and seizures.

Inhaled Corticosteroids

Inhaled corticosteroids (ICS), which are used as first line therapy in asthma, can also be used in COPD. The benefits of ICS are not as universal in COPD but those with severe disease do benefit. Those with a FEV₁ of less than 50 percent of predicted and frequent flares benefit from treatment with inhaled steroids, especially in combination with long-acting bronchodilators⁵. Inhaled steroids do increase the risk of pneumonia and do not decrease mortality⁷.

Inhaled steroids are helpful to reduce the number of flairs, reduce symptoms and improve quality of life. Improvements in FEV₁ were shown for six months in those on ICS compared with those on placebo. This improvement did not continue after six months. The benefit was more pronounced in former smokers compared with current smokers and greater in women than men¹³.

Combination therapy with corticosteroids plus an inhaled long-acting beta₂ agonist control COPD symptoms better than either treatment alone without greater risk of an adverse effect. Beta-receptors may be up regulated by ICS resulting in the positive effect¹⁴.

Oral Steroids

The use of oral steroids may be helpful in the short-term for an acute exacerbation of COPD, but are typically not recommended for long-term use, unless clear benefit has been documented. Twenty to thirty percent of COPD patients improve while on long-term steroids, but an improvement in FEV₁ of at least 20% must be documented in order to warrant their consideration for long-term use due to side effects⁴. There are many long-term side effects with the use of oral steroids including thinning bones, cataracts and increased blood sugar levels.

Oxygen

Oxygen therapy, which is the only therapy shown to prolong life, is recommended for patients with low levels of blood oxygen. Therapy is indicated for patients with a PaO₂ of less than 55 mm Hg, a PaO₂ of less than 59 mm Hg with evidence of polycythemia, cor pulmonale, congestive heart failure or pulmonary hypertension. It is generally recommended for those with Stage IV COPD. The goal is to maintain oxygen saturation greater than 90% during rest, sleep and exertion. Many patients do not require long-term therapy so each patient should be reevaluated 1-3 months after starting therapy⁷.

In addition to improved survival, oxygen therapy decreases polycythemia, reduces progression to pulmonary hypertension, improves exercise tolerance and decreases dyspnea^{4,7}.

Oxygen is generally a safe therapy, but patients and families need to be reminded not to smoke around the oxygen. Due to the physical restraints of the oxygen, it can be associated with reduction of physical activity and social isolation.

Other treatments

Antibiotics, mucolytics and antitussives are other medications considered for the treatment of COPD. Antibiotics are helpful during acute infectious exacerbations of COPD, but the use of continuous antibiotics is not helpful. Mucolytics may be somewhat beneficial in some with thick sputum; but most do not see benefit. The widespread use of these medications is not recommended⁷. Antitussives are not recommended as cough is a protective mechanism and suppressing it may have negative impact on health.

Treatment Approach

Treatment is based on the stage of illness, which is based on pulmonary function tests and symptoms. In patients with mild disease (stage I), the use of short-acting bronchodilators as needed is appropriate. With progression to moderate disease (Stage II), one or more long-acting bronchodilators should be added. Progression to severe disease (stage III) warrants the addition of inhaled steroids, and patients with multiple exacerbations may benefit from mucolytics. Patients with end-stage disease (stage IV) and hypoxia should be placed on long-term oxygen therapy. Some patients have a response with theophylline use, but for most patients the risks outweigh the benefits.

Table 1: COPD Stage, Symptoms, Criteria and Treatment (adapted from 7)

Stage	Symptoms	Criteria	Treatment
Mild Stage I	Chronic cough Sputum production (in some)	$FEV_1/FEV < 0.70$ $FEV_1 \Rightarrow$ 80% of predicted	Short-acting bronchodilators when needed Reduce risk-factors Annual influenza and pneumococcal immunization
Moderate – Stage II	Dyspnea on exertion Cough Sputum production	$FEV_1/FEV < 0.70$ $FEV_1 \Rightarrow$ 50 to < 80% of predicted	Above plus add one or more long-acting bronchodilator (tiotropium [Spiriva] or salmeterol [Serevent]), consider pulmonary rehabilitation
Severe Stage III	Increased dyspnea Decreased	$FEV_1/FEV < 0.70$ $FEV_1 \Rightarrow$ 30 to < 50% of	Above plus add inhaled steroids (if exacerbations

	exercise ability Fatigue Exacerbations	predicted	are recurrent)
Very Severe Stage IV	Poor quality of life Signs of cor pulmonale (increased JVP, ankle edema)	FEV ₁ /FEV ₁ < .70 FEV ₁ < 30% of predicted or < 50% of predicted with chronic respiratory failure (SaO ₂ < 88 percent	Long-term oxygen therapy if chronic respiratory failure Surgery (if indicated)

Surgery

Surgery may be considered for the symptomatic patients with advanced COPD. Bullectomy, lung volume reduction surgery (LVRS) and lung transplantation are the current surgical options. There is high risk of death or post-operative complications with lung surgery so surgical candidates are considered carefully.

Bullectomy - removal of giant bullae that range from 1-4 cm in diameter - has been used for years. These bullae press nearby lung tissue, which decreases blood flow and ventilation to the healthy portion of the lung. When the bullae are removed the lung expands, improving function.

Candidates for bullectomy include those with an FEV₁ of less than 50% of predicted. The surgery improves signs and symptoms in some patients especially those with bullae that are causing the majority of their lung problems by compressing nearby lung tissue.

Lung volume reduction surgery improves FEV₁ and the function. This procedure removes the diseased part of the lung and improves breathing. Candidates for the surgery are those that have severe emphysema, CAT scan evidence of emphysema and marked hyperinflation. The procedure has a mortality rate of 0-18 percent. This procedure results in improved FEV₁, walking distance and quality of life⁵.

Lung transplantation are used in patients with severe COPD. Patients should have an expected life expectancy of less than two years due to COPD. The operation improves FEV₁ and exercise capacity. There is conflicting data on whether the surgery improves survival rates⁵. Transplanting both lungs is better for survival than transplanting one lung especially in those less than 60 years old. Bilateral lung transplant improves survival for two more years when compared to unilateral transplantation¹⁵. The widespread use of bilateral lung transplants is debated because the decision to transplant both lungs has the will to take away a potential transplant from another patient.

Case Study

Treatment for John in the moderate disease stage includes routine treatment with a long-acting bronchodilator. He is given another course of oral steroids to get through

this current flair. He is also given a prescription to attend pulmonary rehabilitation. Most importantly he is counseled about the importance of quitting smoking, as this is the most important feature to slow down the progression of his disease. He is told to come back after the steroid taper to get his pneumonia vaccination and to get the influenza vaccination every year.

John continues to smoke and three years later he has his pulmonary function tests redone and they reveal his FEV₁ is now 35% of what is predicted. He continues to have constant flairs of his COPD and is treated at least two times a year with antibiotics and steroids.

His doctor adds on inhaled steroids to his long-acting beta₂ agonists and again encourages him to stop smoking and tells him that he is close to having end-stage COPD. The doctor orders arterial blood gases, which reveals a PaO₂ of 63 mmHg.

Prevention

Those with COPD are at high risk for developing and dying from pneumonia and influenza. COPD patients should be immunized against these two conditions.

Other prevention methods include avoiding patients who are acutely ill with any type of respiratory infections and frequent hand washing.

Treat infections early to prevalent complication of the disease. This is accomplished by starting antiviral agents at the first sign of the flu. Antiviral medication needs to be started within 48 hours of the onset of symptoms – so reporting symptoms and getting to the doctor is essential. In addition, treat at the first signs of a flare of an acute exacerbation of COPD.

Exacerbation of COPD

An exacerbation of COPD occurs when there is an acute change in the baseline level of cough, dyspnea and/or sputum production. Other symptoms associated with exacerbations include: wheezing, chest tightness, fever, increased heart and breathing rates, malaise, sleep difficulties and confusion. It is not always associated with a fever.

Exacerbations are caused by infection, a respiratory irritant such as an allergen or pollution, and at times the cause cannot be determined.

Treatment is oral glucocorticosteroids and inhaled bronchodilators. If infection is present than antibiotics are recommended. Severe exacerbations may result in hospitalization and even intubation.

Frequent exacerbations, typically a marker of more severe disease, are associated with reduced levels of physical activity and increased incidence of depression, anxiety and isolation.

Prognosis

Factors that predict a worse prognosis include continued smoking, older age, lower FEV₁, rapid decline in FEV₁, poor response to bronchodilators, moderate to severe airflow obstruction, presence of retained CO₂, cor pulmonale, frequent exacerbations of COPD, and poor functional capacity.

Case Study – Discussion

Given his years of smoking, he was likely afflicted with COPD years before it was

diagnosed. By the time symptoms became significant enough for the health care system to consider the diagnosis he had moderate disease. If he would have ceased smoking, the progression of the disease may have been slower. Over the course of four years he went from being diagnosed, to be placed on short-acting bronchodilators, to the addition of long-acting bronchodilators to the combination of inhaled steroids and long-acting bronchodilators.

John was eventually placed on long-term oxygen therapy, as his PaO₂ was only 53 mmHg, and was wheelchair bound, due to advanced COPD. He developed a flair of acute infectious COPD, which required hospitalization and intubation. He was unable to be weaned from the respirator and he eventually died from pneumonia and heart failure.

Nursing Role

COPD is a progressive disease that moves through four stages the nurses needs to take a good history to determine which stage the patient is in to direct nursing care. Critical pieces of information for the nurse to obtain include:

- History of exposure – including smoking, environmental and occupational
- Current signs and symptoms
- Recent pulmonary infections

Nursing Care/Management and Patient Education (Based on NANDA diagnosis): Nursing Interventions for corresponding nursing diagnosis in COPD

Ineffective Health Maintenance

- Due to the complex nature of this disease patients are often not managing their disease effectively. The nurse's role is to help assure all aspects of care are up to standards.
- Assist patients to identify factors that are contributing to their disease through individual counseling.
- Help patients understand the disease process and assist them in managing their disease.
- Improve patient's communication skills with their health care provider. Encourage patients to discuss all questions in table 2.
- Assure spirometry is being carried out on those with COPD. This step is often not carried out and is critical to staging the disease as well as treating it properly.
- Encourage patients to attend pulmonary rehabilitation.
- Assure recommended vaccinations.
- Assist in smoking cessation and avoidance of environmental factors known to exacerbate COPD.

Nutrition Imbalanced: Less than Body Requirements

- Identify patients with nutritional problems. Late stage disease often is complicated with weight loss and failure to thrive. Early interventions are critical to prevent complications of malnutrition.
- Monitor weight and calorie intake.
- Increase fluids between meals and to assure adequate hydration and limit fluid intake at meals to maximize food intake.
- Consider small frequent feedings (every 2-3 hours) instead of the standard 3 meals a day.

- Consider between meal supplements if weight loss is an issue.

Activity Intolerance

- Assess patient's ability to tolerate activity as fatigue and dyspnea often limit activity. Teaching patients how to conserve energy and encouraging regular exercise are critical steps in preserving quality of life.
- Encourage progressive activity and consider referral to a formal exercise program including pulmonary rehabilitation.
- Teach safe exercise principles: warm-up before and cool-down after exercise, stopping exercise if one develops any pain, excessive shortness of breath or extreme fatigue.
- Teach patient energy conserving techniques: cooking on a high stool instead of standing, using a shower chair and resting while doing activities of daily living.

Ineffective breathing pattern

- Monitor for shortness or breath, dyspnea on exertion, respiratory rate, use of intercostals and accessory muscles, nasal flaring, breath sounds and cyanosis.
- Encourage use of oxygen if appropriate.
- Avoid high concentration of oxygen as hypoxia stimulates the drive to breathe in the chronic CO₂ retainer. Unsafe increases in the PaO₂ could result in apnea.
- Collaborate with provider on monitoring arterial blood gases.
- Encourage deep breathing and the use of the incentive spirometer as this reduces the risk of alveolar collapse.
- Encourage patients to drink plenty of fluids to help thin secretions.
- Maintain adequate humidification (80%).
- Teach patients pursed lip breathing.
- Assist patient with coughing to make cough more effective.

Individual Coping, Ineffective (also could use Anxiety; Powerlessness)

- Monitor patient's mental state as COPD patients are at high risk for depression, social isolation, anxiety and ineffective coping.
- Assist patient to identify and explore stressful situations and assist in finding alternative solutions.
- Teach relaxation techniques such as deep breathing, exercise, yoga or imagery.
- Refer patients to mental health care providers or support groups if appropriate.
- Help foster supportive relationships.

Table 2: Questions COPD Patients Should Ask their Health Care Provider

<p>How severe is my disease? Mild, moderate, severe or very severe?</p> <p>How fast will this disease progress and what can I expect?</p> <p>Do I need lung function tests? When do I need them repeated?</p> <p>If you are still having problems with your breathing or with excessive coughing: Is there any other medicines or interventions that will reduce symptoms and make me more comfortable?</p> <p>What type of follow-up do I require? How often should I visit the doctor?</p> <p>What types of medicines are being used to treat my lung disease? Do I need oxygen? Are there any non-drug treatments to treat my disease? Could I benefit from pulmonary rehabilitation?</p> <p>Should I see a lung doctor?</p> <p>Should I get a pneumonia vaccine/flu vaccine?</p>
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