Chronic Obstructive Pulmonary Disease (COPD)

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February 28, 2012

NDFS 356

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a progressive condition which leads to decreased ability to expel air. The two types of COPD are chronic bronchitis and emphysema. While these two coexist in many COPD patients, they refer to slightly different problems. Chronic bronchitis is associated with airflow obstruction due to inflammation and excess mucus in the bronchi. Emphysema, on the other hand, is the destruction of alveolar walls and a decrease in the elastic fibers of the lungs. Together, these create large, floppy alveoli that cannot expel air efficiently.

This pulmonary disease is the fifth most common cause of death worldwide (1) and the fourth most common cause of death in the U.S. and Europe (2). It is projected to be the third most common cause of death by 2020 (3,4). One Chinese epidemiological study with over 20,000 participants above the age of 40 shows a prevalence of 8.2% with men more than twice as likely to have the disease as women (5). It is also a major problem in the United States. In 2000, 3.5% of the population was diagnosed with the disease, and this number is likely less than half of the existing cases (6). In recent years about 3 per 1000 adults in the Netherlands were diagnosed annually with COPD (7). Incidence data from other countries are not too different (7).

ETIOLOGY & PATHOPHYSIOLOGY

Etiology

COPD is caused by irritation to the lungs through cigarette smoking or other irritants, and smoking is considered to cause 80-90% of COPD cases (8). Some of the toxic irritants are tar nicotine, carbon monoxide, cyanide, and coal dust. These are found in cigarettes, air pollution, welding fumes, as well as other places (9). These chemicals damage the walls of the bronchi and alveoli. The risk of developing COPD is increased for smokers who have a genetic deficiency of

α_1 antitrypsin (8). This protein coats the lungs from the damage of neutrophil elastase, which is increased with higher amounts of the irritants.

Pathophysiology

Irritation by these substances causes chronic inflammation and oxidation of the lung tissue. This results in metaplasia of the bronchi's epithelial cells which leads to chronic bronchitis. First, chronic swelling and inflammation of the small bronchi leads to airway narrowing. This causes air to be trapped in the lungs during expiration and keeps the lungs hyperinflated at rest. Second, the goblet cells of the bronchi undergo hyperplasia, and the submucosal glands hypertrophy to cause an increase in mucus production. This leads to a chronic productive cough. The metaplasia of epithelial cells, inflammation of the bronchi, and excess mucus all lead to ciliary dysfunction. Because the cilia cannot move the bacteria away from the lungs, recurrent respiratory infections are common (2,10).

The chronic irritation from smoke also causes destruction of the alveolar septum as seen in emphysema. As the walls between the alveoli disintegrate and lose their elasticity, the alveoli cannot expel air efficiently. They become enlarged and limp, decreasing their ability to push air out of the lungs (2,10).

Progression of Disease

The combined effects of the irritation of the lungs as recognized in chronic bronchitis and emphysema cause many functional problems with the lungs. Some of these include: decreased forced expiratory volume (FEV—the amount of air the lungs can exhale in one breath), increased residual volume (RV—the amount of air left in the lungs after expiration), and increased breathing rate (2,10). The increased airway resistance leaves the patient short of breath, fatigued,

and unable to exert much energy in exercise or even daily activities. The patient also often experiences anorexia, difficulty chewing and swallowing, and loss of lean body mass (11).

Later in the progression of the disease, the patient may experience decreased gas exchange due to less surface area of the alveoli. This causes a domino effect of hypoxemia, polycythemia, hypercapnia, respiratory acidosis, respiratory failure, and cor pulmonale (pulmonary hypertension and right ventricular enlargement) (2).

MEDICAL DIAGNOSIS

When patients' physical examinations reveal signs, symptoms, and risk factors of COPD, they should be tested. For instance, patients who are older than 35, are smokers, complain of breathlessness, have a decreased exercise capacity, or mention a chronic productive cough should be tested (2). It is important to rule out asthma when testing for COPD. Asthma patients can be any age, lack a productive cough, and often have a family history of asthma (2).

Pulmonary function tests are also used to rule out asthma and assure the diagnosis. The groups of pulmonary function tests include spirometry, lung volume measurement, and diffusion capacity (12). Spirometry is often one of the first tests done to show lung capacity. It measures the amount of air breathed in and out over a period of time. Emphysema is diagnosed by decreased FEV and forced vital capacity (FVC—the amount of air that can be exhaled after taking the deepest breath possible) and increased RV and functional residual capacity (FRC—the amount of air left in the lungs after normal expiration) (12,13).

Values for each measurement have a diagnostic range for each level of severity of the disease. For example, according to the Global Initiative for Chronic Obstructive Lung Disease (the GOLD standard), mild COPD is FEV greater than 80% of the predicted value, moderate is FEV between 30-79%, and severe is less than 30% (2). Beyond spirometry, the diffusion

capacity of the lungs can be measured by using a harmless tracer gas to determine the amount of perfusion of the gas into circulation. On top of that, flattened diaphragms viewed on a radiograph can also help diagnose the disease (13).

Chronic Bronchitis is diagnosed when patients have a history of symptoms, pulmonary function test results similar to emphysema, and a chronic productive cough lasting three months within a year for two consecutive years (13). Physical examinations and chest radiographs help to confirm the existence of the disease. Blood gas analyses can identify hypoxemia and hypercapnia later in the disease when these signs show.

MEDICAL THERAPIES

Medical therapy for COPD is not intended to cure the disease. Rather, the goals are to manage stable COPD and its exacerbations, which are flare ups caused by further irritation or infection (11). Pharmacological methods of managing both types of COPD include bronchodilators through an inhaler or nebulizer, immediate use of oral corticosteroids, and antibiotics (14). Bronchodilators such as beta agonists and anticholinergic agents relax the muscles around the airways. This makes breathing easier by allowing the bronchi to dilate, thus decreasing airflow obstruction. Corticosteroids decrease airflow obstruction by decreasing inflammation in the bronchi. While these are effective, use for extended periods of time can lead to decreased bone strength, high blood pressure, cataracts, and diabetes. Thus, they are not used for mild cases of COPD. Antibiotics help fight current infections that aggravate the symptoms. Chronic bronchitis patients also benefit from expectorants to break up the mucus and manage dyspnea.

Nonpharmacological treatments include oxygen therapy, lifestyle changes, learning breathing techniques, and lung surgeries (13). Oxygen therapy can be used short-term or long-

term for various conditions. Short-term use is prescribed for patients to use during exacerbations or when exertions leave them breathless. Long-term treatment should not be used unless patients have stopped smoking, have arterial oxygen partial pressures below 7.3 kPa, and are willing to use oxygen therapy for at least 15 hours a day (2). Termination of smoking is a lifestyle change that is essential for long-term treatment. Respiratory hygiene, or staying away from irritants, including vapors, fumes, carbon monoxide, and other chemicals is critical. Improved nutrition to help the body function also helps as well as learning breathing techniques.

Some helpful breathing techniques include bending forward at the waist or breathing in through the nose and out through pursed lips (13). In rare cases, lung reduction surgery or transplantation can be considered. Lung reduction surgery removes the unhealthy lung and frees space for the functioning lung to work. Lung transplantation is rare because of the small number of donors and risks associated with transplants, such as infection and rejection.

TOOLS FOR NUTRITION ASSESSMENT

The anthropometric tools used in nutrition assessment include BMI, skin-fold measurement, and weight (11). It is important to check for weight loss and muscle wasting, especially in patients with emphysema. These patients tend to be thin and underweight. However, it is important to note that weight loss may by masked by fluid retention. One important biochemical marker indicating COPD is increased hematocrit. Hypoxemia late in the disease stimulated erythropoeisis, increasing the hematocrit. Decreased serum transport proteins and increased acute phase proteins are seen due to systemic inflammation. Oxygen saturation is also significant because the lower levels of oxygen cause the hemoglobin to be more saturated. Imbalanced serum electrolytes and negative nitrogen balance are also factors to watch to assess fluid retention and muscle wasting.

Clinical factors to assess are respiratory status, sense of smell and taste, and GI function (11). This includes swallowing ability, constipation, and diarrhea. Swallowing can be more difficult due to dyspnea, constipation may result from low-fiber food choices, and diarrhea may be a secondary result of decreased oxygen to the bowel causing impaired peristalsis. Dietary factors to evaluate include supplements, usual foods, where the patient eats, and the level of social activity during meals. It should also be determined if the patient has a difficult time finding the energy to prepare meals. Motor function should be assessed along with medical, nutritional, and family histories.

MNT

Providing adequate calories and protein are essential for these patients who are at risk for anorexia and malnutrition. Their increased calorie need is affected by the increased work of breathing along with systemic inflammation, carbon dioxide retention, gas diffusing capacity, and cytokines and hormone levels (15). REE should be calculated through indirect calorimetry or doubly labeled water. Protein is needed to help maintain lean muscle mass. Between 1.2-1.7 g/kg/day is recommended (11). This protein level also helps to restore lung function and prevent respiratory infections.

Addressing feeding difficulties is essential to ensuring that patients receive enough calories and protein. Small appetite, fatigue, shortness of breath, and a distended stomach all decrease food intake (11). To manage these problems, nutrition education should be provided. The small appetite and fatigue can be combated by eating small, frequent meals, preparing meals ahead of time, eating easy-to-prepare meals, and resting before meals (16). Other helpful suggestions could be to keep a mini fridge by the bed or to eat soon after waking up.

Shortness of breath and difficulty swallowing or chewing can be reduced by sitting upright when eating, eating soft foods, eating slowly, and breathing deeply while eating (16). The distended stomach can be slightly ameliorated by avoiding gas-forming foods such as certain raw vegetables, beans, and carbonated beverages. Also, drinking between meals helps decrease the volume of the meal. If patients desire, they can use enteral supplementation to increase calorie and protein consumption without having to go through the work of preparing a meal (11). Nutrition education also includes clearing up misconceptions that patients may have. For example, some patients may believe that milk increases mucus production. However, this is not supported by the research (17).

Various other vitamins and minerals are of less concern than energy and protein but still may be helpful. For instance, vitamin C may help those who continue to smoke (11). Magnesium helps muscle contraction and relaxation. Calcium, vitamin D, and vitamin K help maintain bone density for patients on glucocorticoids. Last of all, sodium restrictions may be helpful for patients with cor pulmonale and fluid retention.

LONG TERM PROGNOSIS

The long term prognosis of COPD is poor, and it is worse for those who are malnourished. The level of severity plays a part in the prognosis, although at no point is the disease reversible or curable. COPD reduces life expectancy in varying degrees. One study showed COPD to reduce life expectancy by 1.8 years (2). In another study in the Netherlands, 26% of patients with severe COPD died within 1 year after follow-up (7).

Because prognosis is poor, dietitians play a role in helping improve the quality of life for those experiencing this disease. Good nutrition helps patients manage the symptoms and have a

higher quality of life. Nutrition education on overcoming feeding difficulties and consuming adequate calories and protein is especially important.

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