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Chronic Obstructive Pulmonary Disease

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Abstract: *Chronic obstructive pulmonary disease is a widely prevalent condition usually associated with cigarette consumption. It has a worldwide impact on the allocation of healthcare resources. The diagnosis is made by history and physical examination supported by measures of airflow and lung volume. The levels of impairment, disability and handicap dictate the requirements for medical care. Smoking cessation, immunization, maximal pharmacologic therapy including the use of supplemental oxygen and prompt attention to exacerbations, represent the basic principles of management. Pulmonary rehabilitation has been shown in a number of randomized clinical trials to improve functional exercise capacity and health-related quality of life. Many interesting approaches, such as lung volume reduction, lung transplantation, nutritional support and mechanical ventilation for acute exacerbations, are in various stages of clinical use and evaluation.*

Keywords: COPD, Smoking Cessation, airflow obstruction and information, bronchodilator

I. INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common and treatable disease characterized by progressive airflow limitation and tissue destruction. It is associated with structural lung changes due to chronic inflammation from prolonged exposure to noxious particles or gases most commonly cigarette smoke. Chronic inflammation causes airway narrowing and decreased lung recoil. The disease often presents with symptoms of cough, dyspnea, and sputum production. Symptoms can range from being asymptomatic to respiratory failure. [5]

1.1 What is COPD

COPD comprises a diverse group of clinical syndromes that share the common feature of limitation of expiratory airflow. The American Thoracic Society defines COPD in terms of chronic bronchitis and emphysema. 2 Chronic bronchitis is characterized by the clinical symptoms of excessive cough and sputum production; emphysema refers to chronic dyspnea, resulting from enlarged air spaces and destruction of lung tissue. The GOLD initiative defines COPD as “a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases.3 “Asthma is also characterized by airflow obstruction and inflammation, but in addition it involves hyperresponsiveness of the airways to stimulus; therefore, the reversibility of functional deficits in asthma differentiates it from COPD.4

1.2 Sign and Symptoms

Signs and symptoms of COPD may include :

- Shortness of breath, especially during physical activities
- Wheezing
- Chest tightness
- A chronic cough that may produce mucus (sputum) that may be clear, white, yellow or greenish
- Frequent respiratory infections
- Lack of energy
- Unintended weight loss (in later stages)
- Swelling in ankles, feet or legs
- People with COPD are also likely to experience episodes called exacerbations, during which their symptoms become worse than the usual day-to-day variation and persist for at least several days. (12)

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1.3 Causes

The main cause of COPD in developed countries is tobacco smoking. In the developing world, COPD often occurs in people exposed to fumes from burning fuel for cooking and heating in poorly ventilated homes.

Only some chronic smokers develop clinically apparent COPD, although many smokers with long smoking histories may develop reduced lung function. Some smokers develop less common lung conditions. They may be misdiagnosed as having COPD until a more thorough evaluation is performed.12)

How your lungs are affected

Air travels down your windpipe (trachea) and into your lungs through two large tubes (bronchi).

Inside your lungs, these tubes divide many times — like the branches of a tree — into many smaller tubes (bronchioles) that end in clusters of tiny air sacs (alveoli).

The air sacs have very thin walls full of tiny blood vessels (capillaries). The oxygen in the air you inhale passes into these blood vessels and enters your bloodstream. At the same time, carbon dioxide — a gas that is a waste product of metabolism — is exhaled.

Your lungs rely on the natural elasticity of the bronchial tubes and air sacs to force air out of your body. COPD causes them to lose their elasticity and over-expand, which leaves some air trapped in your lungs when you exhale.(12)

1.4 Causes of Airway Obstruction

- **Emphysema:** This lung disease causes destruction of the fragile walls and elastic fibers of the alveoli. Small airways collapse when you exhale, impairing airflow out of your lungs.
- **Chronic bronchitis:** In this condition, your bronchial tubes become inflamed and narrowed and your lungs produce more mucus, which can further block the narrowed tubes. You develop a chronic cough trying to clear your airways.
- **Cigarette smoke and other irritants**: In the vast majority of people with COPD, the lung damage that leads to COPD is caused by long-term cigarette smoking. But there are likely other factors at play in the development of COPD, such as a genetic susceptibility to the disease, because not all smokers develop COPD. Other irritants can cause COPD, including cigar smoke, secondhand smoke, pipe smoke, air pollution, and workplace exposure to dust, smoke or fumes.
- **Alpha-1-antitrypsin deficiency:** In about 1% of people with COPD, the disease results from a genetic disorder that causes low levels of a protein called alpha-1-antitrypsin (AAt). AAt is made in the liver and secreted into the bloodstream to help protect the lungs. Alpha-1-antitrypsin deficiency can cause liver disease, lung disease or both.
- For adults with COPD related to AAt deficiency, treatment options include those used for people with more-common types of COPD. In addition, some people can be treated by replacing the missing AAt protein, which may prevent further damage to the lungs.(12)

1.5 Treatment of COPD

Systemic Steroid Therapy

A short course (1-3) weeks of oral glucocorticoid may benefit some patients of COPD during an exacerbation. The airway inflammation in COPD is not very responsive to corticosteroid. As such, only High dose inhaled Steroid are beneficial in advanced COPD with frequent exacerbation; should not be used in early/mild cases. There is no proof that they slow disease progression.

Approaches to Treatment

1) Preventing of AG:AB retention :-

Avoidance of antigen, hyposenstitization possible in extrinsic asthma and if antigen can be identified .

2) Neutralization of IgE (reaginic antibody) –

Omalizumab

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3) Suppression of inflammation and bronchial hyper reactivity –
Corticosteroid
4) Prevention of release of mediators –
Mast cell stabilizer
5) Antagonisms of released mediator –
Leukotriene antagonist, antihistamine , PAF , antagonists
6) Blockade of constrictor neurotransmitter –
Anticholinergic
7) Mimicking dilators neurotransmitter –
Sympathomimetic
8) Directly acting bronchodilator – Methylxanthines

1.6 Smoking Cessation

The single most important intervention in modifying the course of COPD in patients who smoke is smoking cessation. The Lung Health Study reported a progressive decline in postbronchodilator FEV1 in men and women who continued to smoke over an 11-year period. (6)At 11 years, 38% of continuing smokers had an FEV1 <60 of the predicted normal value compared with 10% of sustained quitters. (6)Most patients will make several attempts before they succeed in giving up the use of tobacco, but even a 3-minute counseling session has been shown to result in quitting rates of 5% to 10%.(7) A number of drugs are effective in promoting smoking cessation, including nicotine replacement products (eg, nicotine gum, patch, inhaler), the antidepressant bupropion (Zyban), the drug varenicline (Chantix), in addition to counseling.(8,9)Most smokers should be treated with varenicline as a first-line agent. Smoking-cessation rates are highest when medical management is combined with counseling. Relapse is common, and patients need to be coached and realize that multiple attempts at quitting are often required before quitting permanently. Acupuncture and hypnosis are often advertised as smoking cures; however, a meta-analysis of 22 studies comparing acupuncture with sham acupuncture or with other methods of smoking cessation found no differences
in outcome.(11)

1.7 Medicament

Several kinds of medications are used to treat the symptoms and complications of COPD. You may take some medications on a regular basis and others as needed.(14)

1.8 Bronchodilators

Bronchodilators are medications that usually come in inhalers — they relax the muscles around your airways. This can help relieve coughing and shortness of breath and make breathing easier. Depending on the severity of your disease, you may need a short-acting bronchodilator before activities, a long-acting bronchodilator that you use every day or both. Examples of short-acting bronchodilators include:

- Albuterol (ProAir HFA, Ventolin HFA, others)
- Ipratropium (Atrovent HFA)
- Levalbuterol (Xopenex)

Examples of long-acting bronchodilators include:

- Aclidinium (Tudorza Pressair)
- Arformoterol (Brovana)
- Formoterol (Perforomist)
- Indacaterol (Arcapta Neoinhaler)
- Tiotropium (Spiriva)
- Salmeterol (Serevent)
- Umeclidinium (Incruse Ellipta)

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1.9 Inhaled Steroids

Inhaled corticosteroid medications can reduce airway inflammation and help prevent exacerbations. Side effects may include bruising, oral infections and hoarseness. These medications are useful for people with frequent exacerbations of COPD. Examples of inhaled steroids include:

- Fluticasone (Flovent HFA)
- Budesonide (Pulmicort Flexhaler

Combination inhalers -

Some medications combine bronchodilators and inhaled steroids. Examples of these combination inhalers include:

- Fluticasone and vilanterol (Breo Ellipta)
- Fluticasone, umeclidinium and vilanterol (Trelegy Ellipta)
- Formoterol and budesonide (Symbicort)
- Salmeterol and fluticasone (Advair HFA, AirDuo Digihaler, others)

Combination inhalers that include more than one type of bronchodilator also are available. Examples of these include:

- Aclidinium and formoterol (Duaklir Pressair)
- Albuterol and ipratropium (Combivent Respimat)
- Formoterol and glycopyrrolate (Bevespi Aerosphere)
- Glycopyrrolate and indacaterol (Utibron)
- Olodaterol and tiotropium (Stiolto Respimat)
- Umeclidinium and vilanterol (Anoro Ellipta)

1.10 Oral Steroids

For people who experience periods when their COPD becomes more severe, called moderate or severe acute exacerbation, short courses (for example, five days) of oral corticosteroids may prevent further worsening of COPD. However, long-term use of these medications can have serious side effects, such as weight gain, diabetes, osteoporosis, cataracts and an increased risk of infection.

Phosphodiesterase-4 inhibitors -

A medication approved for people with severe COPD and symptoms of chronic bronchitis is roflumilast (Daliresp), a phosphodiesterase-4 inhibitor. This drug decreases airway inflammation and relaxes the airways. Common side effects include diarrhea and weight loss.

Theophylline -

When other treatment has been ineffective or if cost is a factor, theophylline (Elixophyllin, Theo-24, Theochron), a less expensive medication, may help improve breathing and prevent episodes of worsening COPD. Side effects are dose related and may include nausea, headache, fast heartbeat and tremor, so tests are used to monitor blood levels of the medication.(12,14)

Future Direction for Research

Several rather small-scale interventional trials suggest That improved oral care may reduce the incidence of Nosocomial pneumonia. Additional multi-center trials Will determine the generalizability of oral intervention In the prevention of pneumonia in the institutional settings. Such studies must monitor appropriate aspects Of oral health status to draw proper conclusions Regarding the role of oral health in the prevention of Pneumonia. Additional longitudinal epidemiologic studies are Required to validate the reported association between Periodontal disease and COPD. Randomized controlled Intervention studies that test the effect of periodontal Treatment on the progression of COPD are needed.

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II. CONCLUSION

COPD will remain a significant healthcare problem for years to come. Early identification of the disease through primary care screening for the common symptoms in smokers or those exposed to air pollutants or toxins will lead to earlier diagnosis and treatment. Focusing on smoking cessation will have a great impact on the progression of disease. Advancements in treatment will require translation of a more fundamental understanding of the pathophysiologic pathways involved into disease-modifying interventions. At present, management efforts are directed toward improving patients' symptoms and functional limitations through carefully selected treatment modalities.

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