Review Article

A review on chronic obstructive pulmonary disease

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Abstract

Chronic obstructive pulmonary disease (COPD) commonly occurs in the older age. It is a progressive lung disease which is due to a group of disorders such as refractory asthma, emphysema, and chronic bronchitis. Most people with COPD have both emphysema and chronic bronchitis, and they experience difficulty in breathing. The disease is mainly characterized by increased breathlessness. Acute exacerbations of COPD are mainly caused by bacteria such as nontypeable *Haemophilus influenzae*, *Moraxella catarrhalis*, *Streptococcus pneumoniae*, and *Chlamydia pneumoniae*. The severity of the exacerbation is based on three cardinal symptoms. The three cardinal symptoms are increased sputum volume, increased sputum purulence, and increased dyspnea compared with baseline. Patients experiencing two symptoms are considered to have moderate and all three symptoms are considered to have severe disease status. Antibacterial therapy is the definite treatment for the COPD.

Keywords: Bacteria, breathlessness, bronchitis, chronic obstructive pulmonary disease, emphysema

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INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is also known as Chronic Obstructive Lung Disease and Chronic Obstructive Airway Disease. COPD is a type of lung disease which progresses over a period of time restricting the flow of airway. The main symptoms are shortness of breath and cough with sputum production.[1] Most people with chronic bronchitis are known to have COPD.[2] Tobacco smoking is the most common cause of COPD. Air pollution and genetics have a small role in COPD.[3] Prolonged exposure of irritants causes an inflammatory response in the lungs resulting in narrowing of the small airways and breakdown of lung tissue. This condition is known as emphysema. This shows a poor airflow in the lungs which can be measured by lung function tests. Thus LFT (Lung Function Test) helps in the diagnosis of COPD. In contrast to asthma, the airflow reduction does not improve significantly with the administration of medication. COPD can be prevented by decreasing the smoking rate and improving the quality of air. The treatment for COPD is to quit smoking, bronchodilators, vaccinations, rehabilitation, and steroids. Some people may benefit from long-term oxygen therapy or lung transplantation.[4] Worldwide, COPD affects 329 million people or nearly 5% of the population.[5]

ETIOLOGY

The causes of COPD are smoking, air pollution, occupational exposure, and genetics. Symptoms are evident after several decades of exposure to these irritants. A person's genetic makeup also affects the risk of COPD.[6]

Smoking

The primary risk factor for COPD worldwide is tobacco smoking.[7] About 20% of smokers are known to be

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suffering from COPD,[8] and 50% of lifelong smokers have COPD.[8-10] The development of COPD increases with the total smoke exposure. It is also known that women are more susceptible to the harmful effects of smoke than men. In nonsmokers, second-hand smoke is the cause of about 20% of cases. The use of marijuana, cigar, and waterpipe smoke is also at a risk of developing COPD. Women who smoke during pregnancy may increase the risk of COPD in their child.[10]

Air pollution

Poorly ventilated cooking fires which uses coal or biomass fuels such as wood and animal dung lead to indoor air pollution and are one of the most common causes of COPD in developing countries.[11] People who live in large cities have a higher rate of COPD compared to people who live in rural areas due to the amount of exposure to chemical irritants and air pollution.[12] Areas with poor outdoor air quality, including that from exhaust gas, generally have higher rates of COPD.[13]

Genetics

Genetics also plays a role in the development of COPD. The inherited risk factor is alpha 1-antitrypsin deficiency (AAT). The risk is specifically high in people deficient in alpha 1-antitrypsin and who also smokes.[14] It is responsible for about 1%–5% of cases, and the condition is present in about 3–4 in 10,000 people.[15]

Occupational exposure

Long-term and intense exposure to chemicals, fumes, and workplace hazards increase the risk of COPD in both smokers and nonsmokers.[16] 10%–20% of cases are due to workplace exposures.[17] In the United States, they are believed to be related to more than 30% of cases among those who have never smoked and probably represent a greater risk in countries without sufficient regulations. A number of industries and sources have been implicated, including high levels of dust in coal mining, gold mining, and the cotton textile industry, occupations involving cadmium and isocyanates, and fumes from welding. Silica dust exposure can also lead to COPD, with the risk unrelated to that for silicosis.[18]

SIGNS AND SYMPTOMS

The symptoms of COPD are breathlessness, cough, and sputum production.[19] These symptoms are present for a prolonged period of time and typically worsen over time.

Cough

The first symptom to occur is a chronic cough. If the cough exists for more than 3 months a year for more than 2 years with sputum production, it is defined as chronic bronchitis. Chronic bronchitis can occur even before COPD completely develops. In some cases, the cough may not be present or only occurs occasionally and may not be productive. Rib fracture can happen due to vigorous coughing. Those with COPD often have a history of common cold that lasts for a long period of time.[19]

Shortness of breath

The symptom that bothers people the most is shortness of breath. Typically, the shortness of breath is worse on exertion, of a prolonged duration, and worsens over time. People with more advanced COPD breathe through open mouth or pursed lips to improve shortness of breath.[20,21]

Exacerbation

Increased shortness of breath with increased sputum production is known as acute exacerbations. A change in the color of the sputum from clear to green or yellow is seen. The signs to elicit in these cases are dyspnea, Tachycardia, increased sweating, stiffness of the neck, cyanosis and altered behaviour. Crackles may also be heard over the lungs on examination with a stethoscope.[22]

Other symptoms

In COPD, it may take longer to breathe out than to breathe in. Chest tightness may occur but is not common and may be caused by another problem.[23] On examination of the chest with a stethoscope of those with obstructed airflow, decreased sounds are heard.[23] A barrel chest is a characteristic sign of COPD but is relatively uncommon.[23] Tripod positioning may occur as the disease worsens. Advanced COPD leads to high pressure on the lung arteries, which strains the right ventricle of the heart. This situation is referred to as cor pulmonale and leads to symptoms of leg swelling and bulging neck veins. COPD is more common than any other lung disease as a cause of cor pulmonale.[24]

RISK FACTORS

Smoking

Cigarette smoking is clearly the single most important risk factor in the development of COPD. Current smoking is also associated with an increased risk of death. Pipe and cigar smoking also significantly increase morbidity and mortality from COPD although the risk is less than for cigarettes. Although smoking is the most important risk factor, it is not a prerequisite. COPD can occur in nonsmokers with long-standing asthma or with $\alpha 1$ antitrypsin deficiency. Moreover, about 20% of COPD cases in men are not attributable to smoking. Current cigarette smoking is the most important risk factor for the development of COPD.[25]

Air pollution

Urban air pollution may affect lung function development and consequently be a risk factor for COPD. Cross-sectional studies have shown that higher concentrations of atmospheric air pollution are associated with increased cough, sputum production, and breathlessness and reduced ventilator function. Exposure to particulate and nitrogen dioxide air pollution has been associated with impaired ventilatory function in adults and reduced lung growth in children.[26]

Occupation

Intense prolonged exposure to dusts and chemicals can cause COPD independently of cigarette smoking although smoking seems to enhance the effects of such occupational exposure to increase the risk of developing COPD. About 20% of diagnosed cases of COPD are thought to be attributable to occupation.[27]

α1 antitrypsin deficiency

The best-documented genetic risk factor for COPD is α1 antitrypsin deficiency. However, this is rare and is present in only 1%–2% of patients with COPD. α1 antitrypsin is a glycoprotein responsible for most of the antiprotease activity in serum. The α1 antitrypsin status of patients with severe COPD who are <40 years should be determined since over half of such patients have this deficiency.[28]

PATHOPHYSIOLOGY

COPD is a group of complex progressive disorders comprised of airway inflammation, mucociliary dysfunction followed by airway structural changes.[29]

Airway inflammation

COPD is characterized by chronic inflammation of the airways, lung tissue, and pulmonary blood vessels as a result of exposure to inhaled irritants such as tobacco smoke. The inhaled irritants cause inflammatory cells such as neutrophils, CD8+ T-lymphocytes, B-cells, and macrophages to accumulate. When activated, these cells initiate an inflammatory cascade that triggers the release of inflammatory mediators such as tumor necrosis factor alpha, interferon gamma, matrix-metalloproteinase (MMP)-6, MMP-9, C-reactive protein (CRP), interleukins (IL)-1, IL-6, IL-8, and fibrinogen. With the release of these inflammatory mediators, ther will be tissue damage that results in systemic effects. The chronic inflammation is present from the outset of the disease and leads to various structural changes in the lung which further perpetuate airflow limitation.[30]

Mucociliary dysfunction

Smoking and inflammation enlarge the mucous glands that line airway walls in the lungs, causing goblet cell metaplasia and leading to healthy cells being replaced by more mucus-secreting cells.[31] In addition, inflammation associated with COPD causes damage to the mucociliary transport system which is responsible for clearing mucus from the airways. Both these factors contribute to excess mucus in the airways which eventually accumulates, blocking them and worsening airflow.

DIAGNOSIS

If symptoms such as shortness of breath, a chronic cough, sputum production, or frequent colds are seen, then a diagnostic test for COPD should be done, especially in people over 40 year of age. Spirometer is then used to confirm the diagnosis.[32]

Spirometer

Spirometer measures the amount of airflow obstruction present and is generally carried out after the use of a bronchodilator, a medication to open up the airways.[33] The parameters that indicate the airflow in spirometry are, Forced vital capacity (FVC) which is the total amount (volume) of air expelled from the lungs after a full breath into the lungs (to total lung capacity), Forced Expiratory Volume in one second (FEV1) which is the amount (volume) of air expelled in the first second after a full breath into the lungs and then breathing out hard and fast, as above, trying to push all the air out of the lungs, Forced Expiratory Ratio [(FEV): FEV1/FVC], which helps work out whether there is an obstructive or restrictive breathing pattern and finally Peak Expiratory Flow (PEF) which is the greatest speed that can be reached on full exhalation and fast as possible from the biggest and deep inhalation.[34] The National Institute of Clinical Excellence criteria additionally required FEV_1 of <80% of predicted. Evidence for using spirometer among those without symptoms in an effort to diagnose the condition earlier is of uncertain effect and is therefore currently not recommended. A peak expiratory flow (the maximum speed of expiration), commonly used in asthma, is not sufficient for the diagnosis of COPD.

Other tests

A chest X-ray and complete blood count may be useful.[35] Characteristic signs on X-ray are over expanded lungs, a flattened diaphragm, increased retrosternal airspace, and bullae while it can help exclude other lung diseases such as pneumonia, pulmonary edema, or a pneumothorax.[36]

DIFFERENTIAL DIAGNOSIS

Differentiation of COPD from other causes of shortness of breath such as congestive heart failure, pulmonary embolism, and pneumonia is necessary. Many people with COPD mistakenly think they have asthma.[37] The distinction between asthma and COPD however cannot be made through spirometry. Tuberculosis may also present with a chronic cough and should be considered in locations where it is common. Less common conditions that may present similarly include bronchopulmonary dysplasia and obliterative bronchiolitis. Chronic bronchitis may occur with normal airflow, and in this situation, it is not classified as COPD.[38]

MANAGEMENT

The aim is to reduce risk factors, manage COPD, prevent and treat acute exacerbations, and manage its associated illnesses. Stopping smoking decreases the risk of death by 18%. Other recommendations include influenza vaccination once a year, pneumococcal vaccination once every 5 years, and reduction in exposure to environmental air pollution. In those with advanced disease, palliative care may reduce symptoms, with morphine improving the feelings of shortness of breath. Noninvasive ventilation may be used to support breathing.

Exercise

Pulmonary rehabilitation is an exercise program, disease management, and counseling to benefit the individual. Pulmonary rehabilitation helps to improve the lung function and reduces the mortality rate of Chronic Pulmonary Obstructive Disease.[39]

Bronchodilators

Inhaled bronchodilators are the primary medications used.[40] There are two major types: β2 agonists and anticholinergics; both exist in long-acting and short-acting forms. There are several short-acting β2 agonists available including salbutamol (Ventolin) and terbutaline. They provide some relief of symptoms for 4 to 6 h. Long-acting β2 agonists such as salmeterol and formoterol are often used as maintenance therapy. The adverse effects of usage of bronchodilators over a period of year are shakiness and heart palpitations.[41] There are two main anticholinergics used in COPD: ipratropium and tiotropium. Ipratropium is a short-acting agent while tiotropium is long acting. Tiotropium is associated with a decrease in exacerbations and improved quality of life, and tiotropium provides those benefits better than ipratropium. Anticholinergics can cause dry mouth and urinary tract symptoms.[42]

Corticosteroids

Corticosteroids are usually used in inhaled form but may also be used as tablets to treat and prevent acute exacerbations. While inhaled corticosteroids have not shown benefit for people with mild COPD, they decrease acute exacerbations in those with either moderate or severe disease.[43]

Surgery

For those with very severe disease, surgery is sometimes helpful and may include lung transplantation or lung volume reduction surgery. Lung volume reduction surgery involves removing the parts of the lung most damaged by emphysema allowing the remaining, relatively good lung to expand and work better. Lung transplantation is sometimes performed for severe COPD, particularly in younger individuals.[44]

PREVENTION

By reducing exposure to smoke and improving air quality, most of the cases of COPD are preventable.[45] Annual influenza vaccinations in those with COPD reduce exacerbations, hospitalizations, and death. Pneumococcal vaccination may also be beneficial.[46]

Smoking cessation

Keeping people from starting smoking is a key aspect of preventing COPD. The policies of governments, public health agencies, and anti-smoking organizations can reduce smoking rates by discouraging people from starting and encouraging people to stop smoking. Often, several attempts are required before long-term abstinence is achieved. Attempts over 5 years lead to success in nearly 40% of people. Some smokers can achieve long-term smoking cessation through willpower alone. Smoking, however, is highly addictive, and many smokers need further support. The chance of quitting is improved by use of medications such as nicotine replacement therapy, bupropion, or varenicline.[47]

Occupational health

A number of measures have been taken to reduce the likelihood that workers in at-risk industries – such as coal mining, construction, and stonemasonry – will develop COPD. Examples of these measures include the creation of public policy, education of workers and management about the risks, promoting smoking cessation, checking workers for early signs of COPD, use of respirators, and dust control. Effective dust control can be achieved by improving ventilation, using water sprays and using mining techniques that minimize dust generation.[48,49]

Air pollution

Both indoor and outdoor air quality can be improved, which may prevent COPD or slow the worsening of existing disease. This may be achieved by public policy efforts, cultural changes, and personal involvement. One key effort is to reduce exposure to smoke from cooking and heating fuels through improved ventilation of homes and better stoves and chimneys. Proper stoves may improve indoor air quality by 85%. Using alternative energy sources such as solar cooking and electrical heating is effective, as is using fuels such as kerosene or coal rather than biomass.[49-53]

CONCLUSION

Therefore, COPD is a significant clinical challenge for patients and clinicians. Clinicians' expert knowledge regarding diagnosis and management can enhance patients' longevity and quality of life. Results of emerging studies will likely lead to enhancements in current management and new paradigms in managing patients with COPD.

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