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Comorbid Conditions of Chronic Obstructive Pulmonary Disease

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Abstract

Chronic obstructive pulmonary disease (COPD) is a common lung disease causing restricted airflow and breathing problems. It is sometimes called emphysema or chronic bronchitis.In people with COPD, the lungs can get damaged or clogged with phlegm. Symptoms include cough, sometimes with phlegm, difficulty breathing, wheezing and tiredness.Smoking and air pollution are the most common causes of COPD. People with COPD are at higher risk of other health problems.COPD is not curable but symptoms can improve if one avoids smoking and exposure to air pollution and gets vaccines to prevent infections. It can also be treated with medicines, oxygen and pulmonary rehabilitation.

Keywords: chronic obstructive lung disease, social discrimination, stigma; self- management empowerment

Introduction

Chronic obstructive pulmonary disease (COPD) is a poorly reversible disease of the lungs that is one of the major causes of morbidity and mortality worldwide. In the United States, it is the fourth leading cause of death after heart disease, cancer, and cerebrovascular disease. By 2020, it is projected to become the third leading cause of death worldwide. Contrary to the trends for other major chronic diseases in the United States, the prevalence of and mortality from COPD have continued to rise,the death rates doubled between 1970 and 2002, and for the first time in 2000, mortality figures for women surpassed those for men. In the United States, 12 million patients are currently diagnosed with COPD, but there is believed to be at least an equal number of individuals with impaired lung function suggestive of COPD who are undiagnosed. Given that the majority of COPD cases are caused by smoking, it is primarily a preventable disease.

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. 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." 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.

Risk Factors

Almost by definition, tobacco use is by far the most important risk factor for COPD, best summarized as cumulative dose or pack-years. However, as noted above, not all heavy smokers develop COPD; in fact, most do not, and there has been considerable interest in other risks. COPD is familial to a greater extent than can be accounted for by the relatively few cases of α_1-antitrypsin (α_1-AT) deficiency. It is not known whether this familial tendency reflects genetic or environmental influences, or both. Dusty occupational environments are well established risks, though probably not major factors in North America. Childhood respiratory illnesses may render some people susceptible to tobacco-induced lung damage. All of these influences are minor compared to that of smoking, and none satisfactorily explains the differences between smokers who develop COPD and those who do not.

Therapy

Measures of quality of life include assessment of symptoms, exercise performance, and health care utilization. None is easy to measure in reproducible fashion, and all have subjective aspects that make things like standardization between different centers difficult. Further, quality of life measured in the short term may or may not apply in the longer term, and long-term studies are expensive and difficult. Some COPD therapies have been justified on the basis of short-term changes in lung function. Improvements in FEV_1 have been related to improvements in quality of life in the short term, so that FEV_1 can function as a surrogate for quality of life. On the other hand, the use of short-term studies as the rationale for long-term therapy carries a number of assumptions that are seldom justified. As indicated above, smoking cessation is the best way to change the course of the disease. Nicotine substitution improves cessation success rates, but as illustrated by the Lung Health Study, most "good" cessation programs are expensive and produce long-term quit rates on the order of 25%.

Bronchodilators. As is perhaps best illustrated by data from the DLD-sponsored IPPB trial, most patients with COPD have a measurable increase in FEV_1 with the inhalation of beta-agonists, and in some the change is substantial Responses to anticholinergic agents are at least comparable, and these agents have been shown to improve quality of life over the short term. The method of delivery of inhaled bronchodilators has not been shown to influence their effect in a clinically significant way. Though there were suggestions that regular, inhaled bronchodilator therapy might alter the long-term course of COPD, this issue was studied in the Lung Health Study and no long-term effect was found.

Corticosteroids. Numerous studies show that some patients with stable COPD have improvements in lung function when given anti-inflammatory corticosteroids. Responses are substantial in a minority of patients and

are most common when steroids are given systemically in large doses. The long-term therapeutic implications of these findings have not been explored adequately. It is not clear how reproducible steroid responses are in a given patient with COPD nor whether steroids change the course of COPD in steroid responders or unselected patients. The advent of high-dose inhaled steroids has made steroid therapy safe and practical, and at present there are at least three major clinical trials of these agents in COPD, one of them sponsored by DLD. The results of these trials will be of great practical and theoretical interest.

Antibiotics for exacerbations. Acute exacerbations of symptoms of COPD are often accompanied by increased sputum volume and purulence that suggest infection of the airways. Treatment of exacerbations with broad spectrum antibiotics is common, and the balance of the evidence indicates that such treatment improves the quality of life by speeding symptomatic recovery. However, the effect is by no means dramatic and it is difficult to use these data to argue a purely bacterial origin of exacerbations. Most of the acceptable studies of this issue were completed more than 10 years ago and used relatively unsophisticated agents. It is not known whether the organisms involved in exacerbations have changed or whether newer antibiotics offer advantages.

It is worth noting that neither the causes nor the consequences of COPD exacerbations are known. The effects of antibiotics and of immunostimulatory agents suggest that exacerbations are in part infectious, a hypothesis supported by the benefits of flu vaccine. However, steroid responses in exacerbations may imply other mechanisms. As to consequences, the studies of Fletcher and colleagues, mentioned previously, showed that exacerbations did not alter the long-term course of COPD in a relatively normal population. They did not study individuals with severe airways obstruction, among whom it is axiomatic that some will develop respiratory failure and die during exacerbations.

Nonpharmacologic therapy. Pulmonary rehabilitation for patients with COPD has a long and controversial history. Broadly speaking, the term refers to patient education and exercise training, and its supporters believe that it improves exercise tolerance and quality of life. There is little doubt that these benefits can occur, and that they can outlast the program.

Comorbidities

Clinicians need to be aware of comorbidities in patients with COPD, which can adversely affect health status and complicate management. COPD is associated not only with other respiratory diseases (eg, pneumonia) but also with diseases affecting organ systems, such as the musculoskeletal system (eg, osteoporosis) and the cardiovascular system (eg, angina).

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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