Several definitions of chronic obstructive pulmonary disease (COPD) exist, but a recent definition has been embraced by the Global Initiative for Chronic Obstructive Lung Disease, or the so-called GOLD guidelines. Chronic obstructive pulmonary disease is a common preventable and treatable disease characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases.
Exacerbations and a variety of comorbidities contribute to the overall severity in individual patients. A clinical diagnosis of COPD should be considered in a patient who has dyspnea, chronic cough, or sputum production as well as a history of exposure to risk factors for this condition. Spirometry is required to make the diagnosis. The presence of a postbronchodilator forced expiratory volume (FEV) in the first second of expiration/forced vital capacity (FVC), or FEV/FVC, ratio less than 0.70 confirms the presence of persistent airflow limitation and thus COPD.

**Postbronchodilator FEV/FVC <0.70 is diagnostic of COPD.**

There are 2 general components to COPD. Chronic bronchitis with obstructive bronchiolitis and lung parenchyma destruction or emphysema are found in the majority of patients with this disease. However, certain patients express 1 pathology more than the other, and it is important to understand how each pathology influences the disease expression for that individual patient. Therefore, patients can be split into certain phenotypes. A phenotype is the physical appearance or biochemical characteristic resulting from an interaction between its genotype and the environment. Given that genotypes are poorly understood in COPD, phenotypes have become synonymous with clinical subgrouping. Several phenotypes have been suggested beyond those of emphysema and chronic bronchitis. These include bronchial hyperresponsiveness, bronchodilator reversibility, hyperinflation, cachexia, frequent exacerbations, and systemic inflammation. A COPD phenotype can have a single or combination description and seems to play a substantial role in how the disease unfolds over time. For example, frequent exacerbations are associated with a poorer prognosis and an excessive decline in airflow as measured by the FEV, which is found in approximately 38% of COPD patients.

**Prevalence**

More than 15 million people in the United States have COPD and more than 210 million worldwide. It has been projected that by 2030 COPD will become the third most common cause of death in the world. True prevalence statistics depend upon the criteria used. According to the more recently accepted definition of COPD, there appears to be between 12 and 24 million adults in the United States with COPD. Older patients, and particularly women, have higher prevalence rates. Prevalence rates vary substantially across different states. There seems to be an ongoing reduction in the prevalence of COPD in the United States, but this does not seem to be the case worldwide, especially in developing countries.

**Morbidity and mortality**

Morbidity from COPD is influenced by a variety of other chronic conditions, considered comorbidities, because they have certain disease-related connections to COPD and have an effect on the patient’s health, often interfering with COPD management. Such comorbidities include cardiovascular diseases, musculoskeletal problems, and diabetes mellitus, which will be discussed below.

Mortality data accuracy is affected by the presence and influences of comorbidities and both the underrecognition and underdiagnosis of COPD itself. Although COPD is often listed as a primary cause of death, it is more likely to be listed as a contributory cause or omitted altogether from the death certificate. In the United States, the estimated direct costs of COPD each year are $29.5 billion and the indirect costs are $20.4 billion, with COPD exacerbations responsible for the largest portion of these costs. Hospitalizations soar as COPD severity increases. Home care costs are underestimated because they ignore the economic value of care of patients with COPD by family members.
Causation
Certain risk factors have been associated with the development of COPD. There is overwhelming evidence that confirms that smoking tobacco remains the major risk factor for this disease. Cigarette smoking pack-years are associated with increased risk for COPD, including passive or secondhand cigarette smoke exposure. Waterpipe smoking seems to be associated with COPD, an observation of importance because, in the United States, there is an epidemic of hookah smoking, which is related to waterpipe smoking. Nonsmokers, however, also can develop COPD.

Certain occupational exposures may increase the risk for COPD. Exposure to high levels of organic particles, as bacterial or fungal toxins, found in farming and certain industrial jobs, such as mining, woodworking, and construction, have been associated with an increased risk for COPD. Air pollution, both inside the home and outdoors, represents an important risk factor for COPD. Residential exposure to traffic-related air pollutants and wood smoke are of particular concern.

A better understanding of risk factors helps prevent the development and progression of COPD. Finally, there are genetic factors that must be considered, such as alpha-1 antitrypsin deficiency; other genetic relationships are poorly understood at this time.

Differentiating COPD from asthma
There is a difference between COPD and asthma. In children and young adults, the most likely chronic airway disease is asthma, whereas in adults, generally after age 40 years, COPD becomes more common and distinguishing it from asthma with permanent airflow limitation can be difficult. Both are airflow obstructive diseases, but they differ in etiology, clinical presentation, and course. COPD includes both chronic bronchitis and emphysema and has a strong connection to cigarette smoking. Many patients improve with bronchodilator therapy, but the airflow obstruction associated with COPD is not completely reversible to normal. The airflow obstruction in asthma is often completely reversible and the patient becomes asymptomatic. Asthma can also occur in nonsmokers. The clinical features of both COPD and asthma are very similar and it is difficult to be completely sure which disease a patient has; in some cases, he or she has both. This is one of the so-called overlap syndromes. It often occurs in older patients who perhaps had asthma at an earlier stage in their life, and then smoked cigarettes for an extended period of time and so developed the clinical picture of COPD. These patients often have a more difficult-to-manage disease, with frequent exacerbations, poor quality of life, a rapid decline in lung function, and high mortality.

Comorbidities and systemic effects
Chronic obstructive pulmonary disease is now thought to be a disease that affects other organs besides the lungs, the so-called systemic effects and other comorbidities of this chronic inflammatory condition. Chronic obstructive pulmonary disease is now thought to be a substantial risk factor for the development of atherosclerosis and subsequent cardiovascular complications.

Cardiovascular disease is the most significant nonrespiratory contributor to both morbidity and mortality in COPD. Coronary artery disease, congestive heart failure, and cardiac arrhythmias all have increased prevalence in patients with COPD, and the degree of airflow obstruction is known to be an independent predictor of these cardiovascular complications in COPD patients. For every 10% decrease in FEV₁, cardiovascular mortality increases by approximately 28% and nonfatal coronary events increase by approximately 20% in mild-to-moderate COPD. Skeletal muscle dysfunction with and without muscle mass loss.
cachexia, and autonomic nervous system dysfunction have all been shown to occur as systemic effects in patients with COPD. Lung cancer, diabetes, osteoporosis, anxiety and depression, and obstructive sleep apnea are comorbidities strongly associated with COPD. Systemic inflammation is a well-established phenomenon in COPD patients. A variety of inflammatory mediators are elevated and may be the common link between COPD and its comorbidities. Serum markers of inflammation, such as C-reactive protein, have been linked to increased risk for cardiac disease and have been shown to be increased in patients with stable COPD as well as during exacerbations. This relationship seems to be greatest in COPD patients who continue to smoke cigarettes. Treatment strategies for patients with COPD should include management of these nonpulmonary sequelae that contribute to the overall disease burden of COPD.

References


