# COPD (Chronic Obstructive Pulmonary Disease)

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## 1 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.

**Chronic bronchitis** is a type of chronic obstructive pulmonary disease (COPD) that can be defined as a chronic productive cough lasting more than 3 months and occurring within a span of 2 years. There is a strong causal association with smoking.

**Pulmonary emphysema** is a form of chronic obstructive pulmonary disease that is defined as a pathological, permanent dilatation of distal airways (respiratory bronchioles, alveolar ducts, and alveolar sacs) due to the destruction of airway walls and alveoli. Emphysema interrupts gas exchange, causing an obstructive ventilation defect.

## 2 Epidemiology

COPD is primarily present in smokers and those greater than age 40. Prevalence increases with age and it is currently the third most common cause of morbidity and mortality worldwide. In 2015, the prevalence of COPD was 174 million and there were approximately 3.2 million deaths due to COPD worldwide. However, the prevalence is likely to be underestimated due to the underdiagnosis of COPD.

## 3 Etiology

There are many known causes of chronic bronchitis, but the most important causative factor is exposure to cigarette smoke, either due to active smoking or passive inhalation. Many inhaled irritants to the respiratory tract, such as smog, industrial pollutants, and toxic chemicals, can cause chronic bronchitis. Repeated exposure to infections can cause COPD. Other causes may include environmental and occupational exposures, and alpha-1 antitrypsin deficiency (AATD).

## 4 Pathophysiology

COPD is an inflammatory condition involving the airways, lung parenchyma, and pulmonary vasculature. In emphysema, an irritant (e.g., smoking) causes an inflammatory response. Neutrophils and macrophages are recruited and release multiple inflammatory mediators. The process is thought to involve oxidative stress and protease-antiprotease imbalances. Emphysema describes one of the structural changes seen in COPD where there is destruction of the alveolar air sacs (gas-exchanging surfaces of the lungs) leading to permanent dilatation decreasing the surface area of the ventilatory units, and also obstructive physiology (destruction of the elastic component of the lung).

In chronic bronchitis, irritants cause an overproduction and hypersecretion of mucus by goblet cells. Epithelial cells lining the airway respond to toxic stimuli by releasing inflammatory mediators (pro-inflammatory cytokines). The bronchial mucous membrane becomes hyperemic and edematous with diminished bronchial mucociliary function. This, in turn, leads to airflow impediment because of luminal obstruction to small airways. The airways become clogged by debris, and this further increases the irritation. The characteristic cough of bronchitis is caused by the copious secretion of mucus in chronic bronchitis.

The inflammatory response and obstruction of the airways cause a decrease in the forced expiratory volume in the first second (FEV1) and tissue destruction leads to airflow limitation and impaired gas exchange. Hyperinflation of the lungs is often seen on imaging studies and occurs due to air trapping from airway collapse during exhalation.

## 5 Clinical presentation

A typical patient with COPD would be in their fourth-fifth decade or over, with extensive smoking history presenting with a chronic, productive cough and worsening shortness of breath. A productive cough with sputum is present in about 50% of patients. The sputum color may vary from clear, yellow, green, or at times blood-tinged. The color of sputum may be dependent on the presence of secondary bacterial infection. In advanced COPD, the symptoms progressively worsen interfering with the activities of daily living. They may also experience unintentional weight loss and would appear cachexic. Patients typically demonstrate "purse lip breathing" and can be visibly tachypneic and use their accessory muscles of respiration. Auscultation usually reveals reduced air entry. The term "pink puffers" is used to describe these patients with emphysema as they are plethoric and have hyperexpanded (barrel-shaped) chest. On the contrary, patients with decompensated chronic bronchitis are referred to as "blue bloaters" as they are cyanotic.

## 6 Evaluation and diagnosis

After a good history and physical examination, the next investigation necessary for a diagnosis of COPD is a complete spirometry. The spirometry typically shows a reduction

in both forced vital capacity (FVC) as well as the forced expiratory volume at first second (FEV1). The FEV1 generally declines more in proportion than the decline in FVC and the FEV1/FVC ratio is less than 0.7. The spirometry can show an obstructive ventilatory defect, but also a restrictive defect. Obstruction is graded from mild to very severe based on the FEV1. The lung volumes could show air trapping and hyperinflation. According to the Global Initiative for Chronic Obstructive Lung Diseases (GOLD), the severity of obstruction in patients with **FEV1/FVC ratio less than 0.7** grading is as follows:

- Mild when FEV1 greater than or equal to 80 of predicted
- Moderate when FEV1 greater than or equal to 50 and less than 80% of predicted
- Severe when FEV1 greater than or equal to 30 and less than 50% of predicted
- Very severe when FEV1 less than 30% of predicted

Radiology plays a significant role in diagnosing emphysema. A chest X-ray would show hyperinflation and a flattened diaphragm. Computerized tomogram (CT) of the chest is more specific and confirmatory for emphysema.

# 7 Treatment

The classes of commonly used medications in COPD include **bronchodilators** (beta2-agonists, antimuscarinics), inhaled corticosteroids (ICS), systemic glucocorticoids[1].

Beta2-agonists work by relaxing the smooth muscle in the airways. Antimuscarinics work by blocking the M3 muscarinic receptors in the smooth muscle and therefore preventing bronchoconstriction. Inhaled corticosteroids are often used in combination with beta2-agonists and antimuscarinics to decrease inflammation. A combination of corticoids and beta2-agonists has been shown to be more beneficial than either of the drugs when used alone. Oral corticoids and antibiotics should be reserved for the management of acute exacerbations.

[1] Corticosteroids and glucocorticoids are the same