Chronic Obstructive Pulmonary Disease

Chronic Obstructive Pulmonary Disease (COPD) is a common, preventable, and treatable disease characterized by persistent respiratory symptoms and airflow limitation due to airway and/or alveolar abnormalities. These changes are typically caused by significant exposure to noxious particles or gases and are not fully reversible. COPD is often progressive and associated with chronic inflammation of the lungs.

> **Key Insight:** Patients with COPD often describe the sensation of being "hungry for air."

Major Disease Entities

COPD includes the following primary conditions:

- **Chronic Bronchitis:** Defined clinically as a productive cough lasting at least 3 months in each of two consecutive years, in the absence of other causes.
- **Emphysema:** Defined pathologically as the permanent enlargement of airspaces distal to the terminal bronchioles, accompanied by destruction of alveolar walls.
- **Asthma-COPD Overlap (ACO):** A subset of patients show features of both asthma and COPD and may benefit from tailored therapy.

Epidemiology

- **Prevalence (U.S.):** ~13.9% in adults aged ?25 years.
- **Underdiagnosis:** Up to 60% of cases may remain undiagnosed.
- **Occupational Risk:** 15–19% of cases are attributed to workplace exposures.

Etiology and Risk Factors

Primary Risk Factor:

- **Cigarette smoking:** Responsible for 80–90% of cases.
    - Leads to:
        - Reduced ciliary function
        - Increased mucus production
        - Alveolar destruction
        - Chronic inflammation

Other Contributing Factors:

- **Environmental Exposure:**
    - Second-hand smoke
    - Industrial dusts (grains, silica, coal)
    - Chemical fumes/vapors

- **Genetic Predisposition:**
  - *Alpha-1 antitrypsin (AAT) deficiency* : Autosomal codominant condition affecting 1–2% of COPD cases.
- **History of respiratory infections in childhood**
- **Air pollution**
- **Low socioeconomic status**

Pathophysiology

Emphysema:

- **Destruction of alveolar walls** ? decreased surface area for gas exchange
- **Loss of elastic recoil** ? airway collapse during exhalation
- **Air trapping and hyperinflation** ? "barrel chest" appearance
- **Types:**
  - **Centrilobular (centriacinar):** Primarily affects respiratory bronchioles (common in smokers).
  - **Panlobular (panacinar):** Uniform involvement of alveoli, associated with AAT deficiency.

Chronic Bronchitis:

- **Hypertrophy and hyperplasia of mucus-secreting glands**
- **Goblet cell proliferation** ? excess mucus
- **Loss of cilia** ? impaired mucus clearance
- **Airway narrowing due to inflammation**
- **Chronic hypoxia** ? polycythemia, cyanosis

Common Pathophysiological Changes:

- Increased airway resistance
- Impaired gas exchange (? PaO?, ? PaCO?)
- V/Q mismatch
- Chronic respiratory acidosis with metabolic compensation in advanced disease

Clinical Features

Shared Symptoms:

- Progressive **dyspnea** (initially on exertion, eventually at rest)
- **Chronic cough** (productive in bronchitis, dry in emphysema)
- **Wheezing** and chest tightness
- **Fatigue** and reduced exercise tolerance

Emphysema Dominant (Pink Puffer):

- Dyspnea predominant
- Minimal cough

- Thin body habitus
- Pursed-lip breathing
- Barrel chest

Chronic Bronchitis Dominant (Blue Bloater):

- Productive cough
- Frequent infections
- Cyanosis
- Obesity
- Peripheral edema (cor pulmonale)

Diagnosis

1. Pulmonary Function Tests (PFTs)

- **Key Diagnostic Criterion:** Post-bronchodilator FEV?/FVC ratio < 0.70 confirms persistent airflow limitation.
- ? FEV?, normal or ? FVC
- ? Residual Volume (RV), ? Total Lung Capacity (TLC)

2. Arterial Blood Gases (ABGs)

- ? PaO?, ? PaCO? (especially in chronic bronchitis)
- Compensated respiratory acidosis in advanced stages

3. Imaging

- **Chest X-ray:**
  - Flattened diaphragm
  - Hyperinflated lungs
  - Increased retrosternal air space
- **High-resolution CT (HRCT):** Useful for emphysema detection and AAT deficiency assessment

4. Other Tests

- **Alpha-1 antitrypsin levels** in early-onset or family history cases
- **Six-minute walk test** or **BODE index** for prognosis

Management

Prevention:

- **Smoking cessation** is the most effective intervention.
- Avoid exposure to occupational and environmental irritants.
- Annual **influenza vaccine** and **pneumococcal vaccine** .

Pharmacological Treatment:

Bronchodilators:

- **Short-acting beta-agonists (SABAs):** e.g., albuterol
- **Long-acting beta-agonists (LABAs):** e.g., salmeterol
- **Long-acting muscarinic antagonists (LAMAs):** e.g., tiotropium

Inhaled Corticosteroids (ICS):

- Indicated in patients with frequent exacerbations, especially with eosinophilia

Combination Therapy:

- LABA + LAMA ± ICS depending on exacerbation history and symptom burden

Others:

- **Phosphodiesterase-4 inhibitors (e.g., roflumilast)** in severe chronic bronchitis
- **Theophylline** (rarely used due to narrow therapeutic index)

? Oxygen Therapy:

- For PaO? ? 55 mmHg or O? saturation ? 88%
- Goal: Maintain SpO? > 90%

Non-Pharmacological:

- **Pulmonary rehabilitation**
- **Nutritional support**
- **Breathing techniques:** Pursed-lip breathing, diaphragmatic breathing
- **Surgical options:** Bullectomy, lung volume reduction surgery, lung transplantation (select cases)

Complications

- Pulmonary hypertension and right heart failure (cor pulmonale)
- Acute exacerbations (triggered by infection or environmental exposure)
- Respiratory failure
- Depression and anxiety

Patient Education

- Encourage smoking cessation programs
- Correct inhaler technique
- Recognize early signs of exacerbation
- Adhere to medications and pulmonary rehab
- Nutritional advice (avoid underweight or obesity)

High-Yield Pearls

- COPD is **not fully reversible** , unlike asthma.
- **FEV? decline is slowed, not reversed** , with smoking cessation.
- **Hypercapnia is more common in chronic bronchitis** .
- **O? therapy must be titrated carefully** to avoid suppressing hypoxic drive in CO? retainers.