Team Medicine

Chronic Obstructive Pulmonary Disease & Bronchiectasis

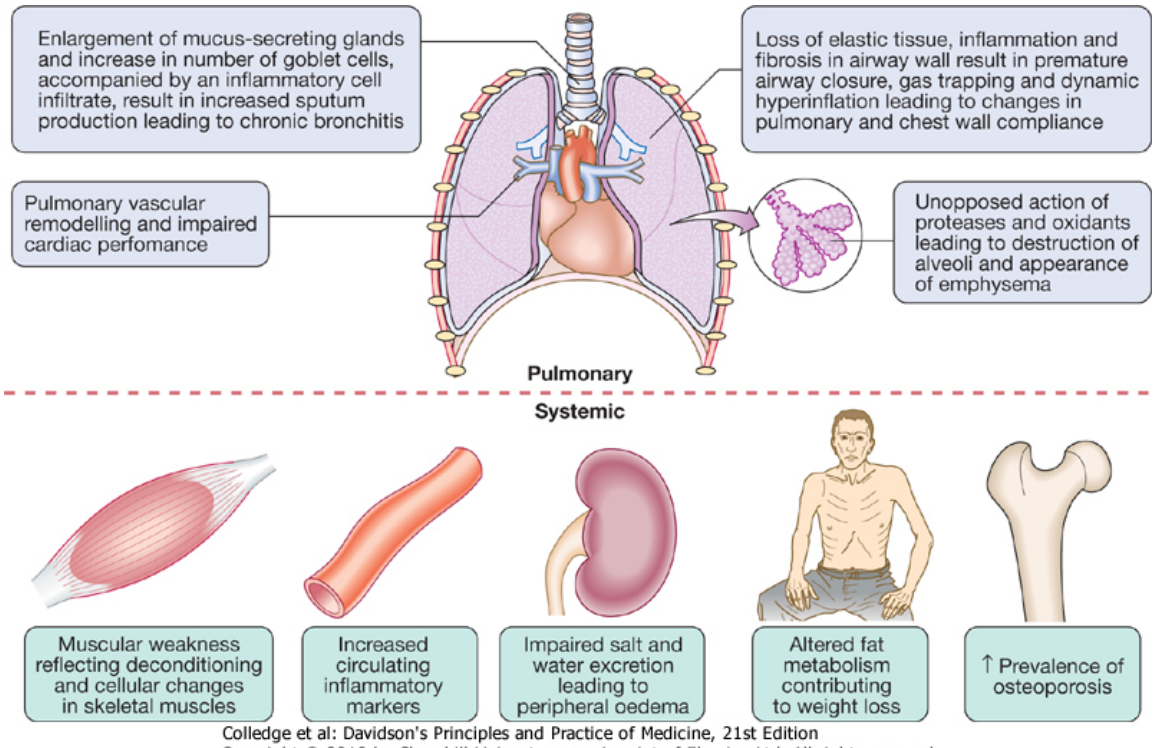
Writer: Saad Khashogji
Reviser: Shatha Altahan
Leader: Alanood Asiri

Chronic obstructive pulmonary disease (COPD)

- **DEFINITION:** "Is 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".

This disease has both intra and extra pulmonary manifestations, the intra-pulmonary can be classified into:

A. **Chronic bronchitis** is a **clinical diagnosis**: chronic cough productive of sputum for at least 3 months per year for at least 2 consecutive years.
B. **Emphysema** is a **pathologic diagnosis**: permanent enlargement of air spaces distal to terminal bronchioles due to destruction of alveolar walls.
C. **The two often coexist.** Pure emphysema or pure chronic bronchitis is rare.
The extra-pulmonary manifestations include impaired nutrition, weight loss and skeletal muscle dysfunction.



Pulmonary and systemic features of COPD

- **RISK FACTORS:**

A. **Tobacco smoke (indicated in almost 90% of COPD cases)**
B. α1-Antitrypsin deficiency—risk is even worse in combination with smoking
C. Environmental factors (e.g., second-hand smoke)
D. Chronic asthma—speculated by some to be an independent risk factor

- **PATHOGENESIS:**

A. Chronic bronchitis
• Excess mucus production narrows the airways; patients often have a productive cough.

• Inflammation and scarring in airways, enlargement in mucous glands, and smooth muscle hyperplasia lead to obstruction.

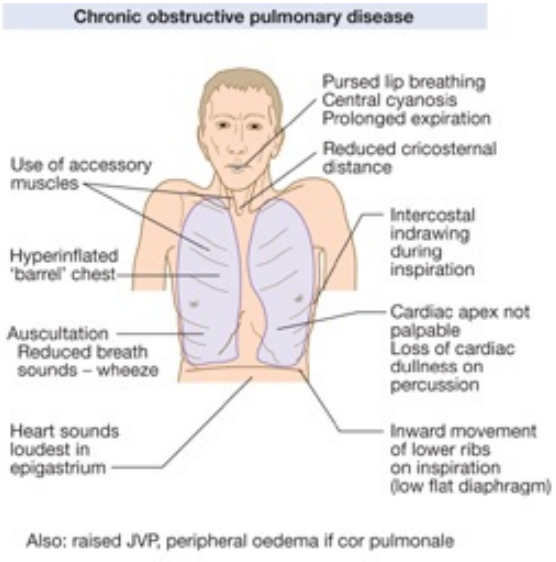
B. Emphysema
• Destruction of alveolar walls is due to relative excess in protease (elastase) activity, or relative deficiency of antiprotease (α1-antitrypsin) activity in the lung. Elastase is released from PMNs and macrophages and digests human lung. This is inhibited by α1-antitrypsin.
• Tobacco smoke increases the number of activated PMNs and macrophages, inhibits α1-antitrypsin, and increases oxidative stress on the lung by free radical production.

- **CLINICAL FEATURES:**

COPD should be suspected in any patient over the age of 40 years who presents with symptoms of **chronic bronchitis (cough, sputum production) and/or breathlessness.**
Cough and associated sputum production are usually the first symptoms, often referred to as a 'smoker's cough'.
Breathlessness usually brings about the first presentation to medical attention. The level should be quantified for future reference by documenting the exercise the patient can manage before stopping; the modified Medical Research Council (MRC) dyspnea scale may also be useful.

Modified MRC dyspnea scale	
Grade	**Degree of breathlessness related to activities**
0	No breathlessness except with strenuous exercise
1	Breathlessness when hurrying on the level or walking up a slight hill
2	Walks slower than contemporaries on level ground because of breathlessness or has to stop for breath when walking at own pace
3	Stops for breath after walking about 100 m or after a few minutes on level ground
4	Too breathless to leave the house, or breathless when dressing or undressing

Signs:
A. During auscultation, end-expiratory wheezes on forced expiration, decreased breath sounds, and/or inspiratory crackles
B. Tachypnea, tachycardia
C. Cyanosis
D. Use of accessory respiratory muscles
E. Hyperresonance on percussion
F. Signs of cor pulmonale



Two classical phenotypes have been described: **'pink puffers' and 'blue bloaters'**. The former are typically thin and breathless, and maintain a normal PaCO2 until the late stage of disease. The latter develop (or tolerate) hypercapnia earlier and may develop oedema and secondary polycythaemia. In practice, these phenotypes often overlap.

- **INVESTIGATIONS:**

1- **Chest radiograph (CXR):** is essential to identify alternative diagnoses such as cardiac failure, other complications of smoking such as lung cancer, and the presence of bullae (an abnormal air-filled cavity in the lung).
2- **A full blood count:** is useful to exclude anaemia or document polycythaemia, and in **younger patients with predominantly basal emphysema, α1-antiproteinase should be assayed.**
3- **Pulmonary function testing (spirometry):** the diagnosis is established when the **post-bronchodilator FEV1 is less than 80% of the predicted value and accompanied by FEV1/FVC < 70%.**
4- **DLCO:** A substantial loss of lung surface area available for effective oxygen exchange causes diminished carbon monoxide diffusion in the lung (DLCO) in patients with emphysema. **This finding may help distinguish COPD from asthma, because patients with asthma typically have normal DLCO values.**

- **DIFFERENTIAL DIAGNOSIS WITH ASTHMA:**
- Age of onset (in asthma is usually less than 40 while COPD usually starts at older age).
- History of atopy (in asthma only, because atopy is a form of allergy).
- Eosinophilia and IgE(more in asthma, and that's why inhalated cortisol is more effective which is directed mainly on eosinophilia and not good in COPD).
  o Note: COPD is mediated by neutrophils mainly
- Bullae (only in COPD).
- Chronic respiratory failure (only in COPD).
- Diffusing capacity (affected in COPD mainly & almost normal in asthma).
- Trial of inhaled corticosteroids (not effective in COPD except for frequent exacerbations).

- **MANAGEMENT:**

1- **Smoking cessation**
The most important intervention, Disease progression is accelerated by continued smoking and can be greatly slowed by its cessation.

2- **Bronchodilator therapy**

A. Short-acting bronchodilators, such as the β2-agonists *salbutamol* and *terbutaline,* or the anticholinergic, ipratropium bromide, may be used for patients with mild disease.

B. Longer-acting bronchodilators, such as the β2-agonists *salmeterol* and *formoterol*, or the anticholinergic *tiotropium bromide*, are more appropriate for patients with moderate to severe disease.

Significant improvements in breathlessness may be reported despite minimal changes in FEV1.

C. Oral bronchodilator therapy may be contemplated in patients who cannot use inhaled devices efficiently. Theophylline preparations improve breathlessness and quality of life, but their use has been limited by side-effects, unpredictable metabolism and drug interactions.

D. Corticosteroids
Inhaled corticosteroids (ICS) reduce the frequency and severity of exacerbations; they are currently recommended in patients with severe disease (FEV1 < 50%) who report two or more exacerbations requiring antibiotics or oral steroids per year.
Oral corticosteroids are useful during exacerbations but maintenance therapy contributes to osteoporosis and impaired skeletal muscle function and should be avoided.

3- Pulmonary rehabilitation
Education, exercise, physiotherapy. Used for patients Grade 3-5 S.O.B, A major goal is to improve exercise tolerance. Pulmonary rehabilitation improves functional status and quality of life.

4- Mucolytics
5- Oxygen therapy
- 'Long-term home oxygen therapy improves survival in selected patients with COPD complicated by severe hypoxaemia (arterial PaO2 less than 8.0 kPa (55 mmHg)).'

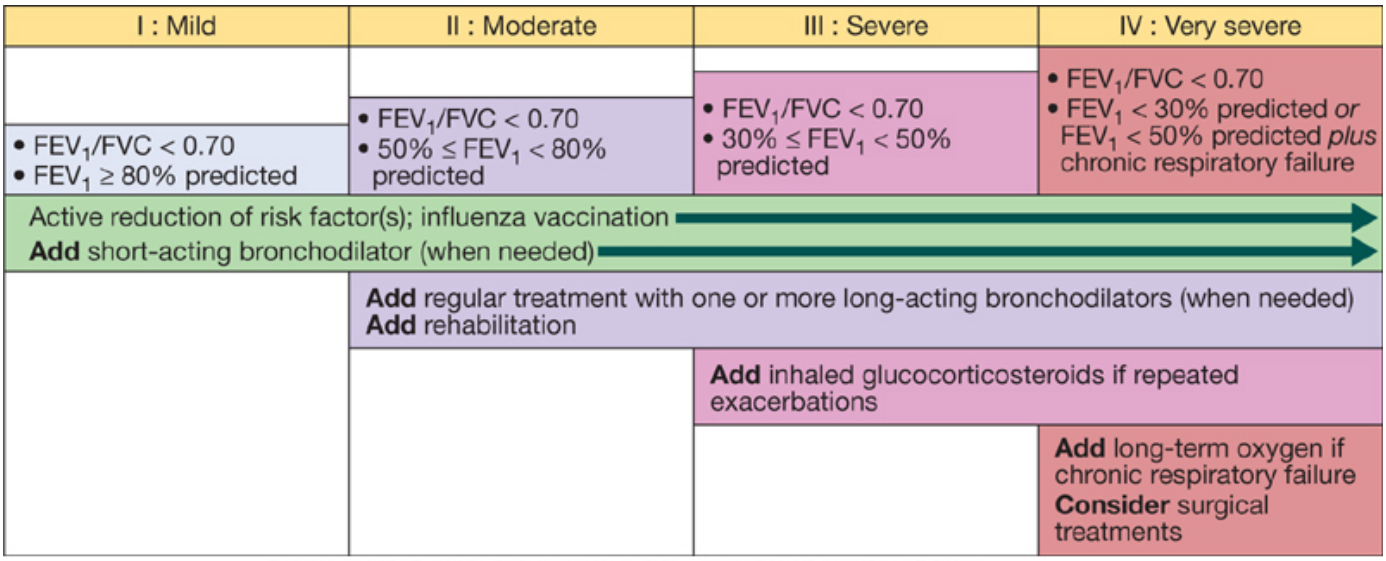
Prescription of long-term oxygen therapy (LTOT) in COPD
Arterial blood gases measured in clinically stable patients on optimal medical therapy on at least two occasions 3 weeks apart:

- PaO_2 < 7.3 kPa (55 mmHg) irrespective of $PaCO_2$ and FEV_1 < 1.5 L
- PaO_2 7.3-8 kPa (55-60 mmHg) plus pulmonary hypertension, peripheral oedema or nocturnal hypoxaemia
- patient stopped smoking.

Use at least 15 hours/day at 2-4 L/min to achieve a PaO_2 > 8 kPa (60 mmHg) without unacceptable rise in $PaCO_2$.

6- Vaccination
- Influenza vaccination annually for all patients.
- Vaccination against Streptococcus pneumoniae every 5 to 6 years should be offered to patients with COPD over 65 years old, or under 65 who have severe disease.

7- Surgery—may be beneficial in selected patients; carefully weigh potential benefits with risks. Options include:
Bullectomy and lung volume reduction surgery.



Guidelines for treatment of COPD

- **Acute exacerbations of COPD:**

Acute exacerbations of COPD are characterized by an increase in symptoms and deterioration in lung function and health status. They become more frequent as the disease progresses and are **usually triggered by bacteria, viruses or a change in air quality**.

1- Oxygen therapy

In patients with an exacerbation of severe COPD, high concentrations of oxygen may cause respiratory depression and worsening acidosis. Controlled oxygen at 24% or 28% should be used with the aim of maintaining a PaO2 > 8 kPa (60 mmHg) (or an SaO2 > 90%) without worsening acidosis.

2- Bronchodilators

Nebulised short-acting β2-agonists combined with an anticholinergic agent (e.g. salbutamol with ipratropium) should be administered.

3- Corticosteroids

Oral prednisolone reduces symptoms and improves lung function.

4- Antibiotic therapy

They are currently recommended for patients reporting an increase in sputum purulence, sputum volume or breathlessness. Studies have shown that patients who receive broad-spectrum antibiotics do slightly better than a placebo group.

5- **Noninvasive positive-pressure ventilation (NPPV) (BIPAP or CPAP):**

Studies have shown a benefit in acute exacerbations. It may decrease the likelihood of respiratory failure requiring invasive mechanical ventilation.

- **COMPLICATIONS:**
- Respiratory failure
- Cor pulmonale
- Bacterial colonisation
- Hemoptysis
- Pneumothorax
- Extrapulmonary manifestation

Bronchiectasis

- **Definition:** Bronchiectasis means abnormal dilatation of the bronchi. Chronic suppurative airway infection with sputum production, progressive scarring and lung damage are present, whatever the cause.

- **Aetiology and pathogenesis:**

Bronchiectasis may result from a congenital defect affecting airway ion transport or ciliary function, such as cystic fibrosis, or be acquired secondary to damage to the airways by a destructive infection, inhaled toxin or foreign body. The result is chronic inflammation and infection in airways.
Localised bronchiectasis may occur due to the accumulation of pus beyond an obstructing bronchial lesion, such as enlarged tuberculous hilar lymph nodes, a bronchial tumour or an inhaled foreign body (e.g. an aspirated peanut).

Causes of bronchiectasis:

Congenital

- Cystic fibrosis
- Ciliary dysfunction syndromes
  - Primary ciliary dyskinesia (immotile cilia syndrome)
  - Kartagener's syndrome { sinusitis, male infertility bronchiectasis and transposition of the viscera (dextrocardia)}
- Primary hypogammaglobulinaemia

Acquired: children

- Pneumonia (complicating whooping cough or measles)
- **Primary TB (most common cause world wide)**
- Inhaled foreign body

Acquired: adults

- Suppurative pneumonia
- Pulmonary TB
- Allergic bronchopulmonary aspergillosis complicating asthma Bronchial tumours

Clinical features:

Physical signs include coarse crackles, diminished breath sounds, and bronchial breathing.
The symptoms of bronchiectasis are summarised in the box :

Symptoms of bronchiectasis

Cough

- Chronic productive cough due to accumulation of pus in dilated bronchi; usually worse in mornings and often brought on by changes of posture. Sputum often copious and persistently purulent in advanced disease. Halitosis is a common accompanying feature

Pneumonia and pleurisy

- Due to inflammatory changes in lung and pleura surrounding dilated bronchi when spread of infection occurs: fever, malaise and increased cough and sputum volume, which may be associated with pleurisy. Recurrent pleurisy in the same site often occurs in bronchiectasis

Haemoptysis

- Can be slight or massive and is often recurrent. Usually associated with purulent sputum or an increase in sputum purulence. Can, however, be the only symptom in so-called 'dry bronchiectasis'

Poor general health

- When disease is extensive and sputum persistently purulent, there may be associated weight loss, anorexia, lassitude, low-grade fever, and failure to thrive in children. In these patients, digital clubbing is common

- ## Investigations:

1. High-resolution CT scan is the diagnostic study of choice.
2. CXR is abnormal in most cases, but findings are nonspecific.
3. Bacteriological and mycological examination of sputum.

In addition to common respiratory pathogens, sputum culture may reveal Pseudomonas aeruginosa, fungi such as Aspergillus and various mycobacteria. Frequent cultures are necessary to ensure appropriate treatment of resistant organisms.

4. A screening test can be performed in patients suspected of having a ciliary dysfunction syndrome.

- **Management:**

In patients with airflow obstruction, inhaled bronchodilators and corticosteroids should be used to enhance airway patency.

1- Physiotherapy: postural drainage, chest percussion to help remove the mucus.

2- Antibiotics for acute exacerbations—superimposed infections are signaled by change in quality/quantity of sputum, fever, chest pain, etc.

3- Surgical treatment: Excision of bronchiectatic areas is only indicated in a small proportion of cases. These are usually young patients in whom the bronchiectasis is unilateral and confined to a single lobe or segment on CT.