L8: Chronic obstructive pulmonary disease

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MED 433

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objectives

1. Definition of the two conditions.
2. Clinical and radiological diagnosis.
3. Differential diagnosis.
4. General outline of management.
5. Create a link to 341 clinical teaching.

Color index: Step up to medicine , slide , Doctor's note , Davidson , Extra Explanation

Chronic obstructive pulmonary disease

- is a preventable and treatable disease characterised 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
- There are **two** classic types of COPD:

Chronic bronchitis	Emphysema
A clinical diagnosis: chronic cough productive of sputum for at least 3 months per year for at least 2 consecutive years.	A pathologic diagnosis: permanent enlargement of air spaces distal to terminal bronchioles due to destruction of alveolar walls.

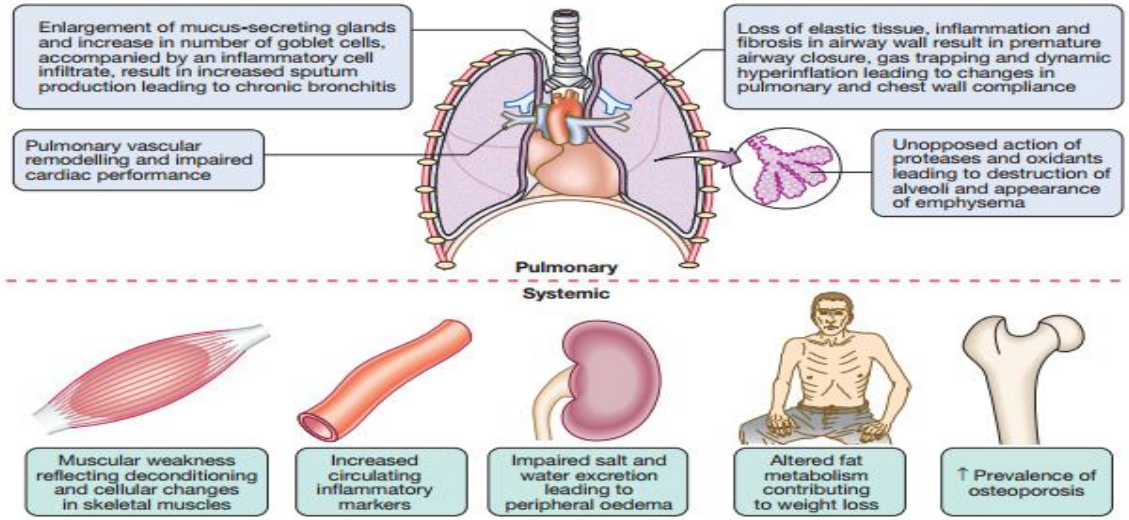
- **The two often coexist.** Pure emphysema or pure chronic bronchitis is rare.

Pathophysiology of chronic bronchitis

Excess mucus production narrows the airways; patients often have a productive cough.

Inflammation and scarring in airways, enlargement in mucous glands

smooth muscle hyperplasia lead to obstruction



The presence of airflow limitation, combined with premature airway closure, leads to gas trapping and hyperinflation, reducing pulmonary and chest wall compliance. Pulmonary hyperinflation also flattens the diaphragmatic muscles and leads to an increasingly horizontal alignment of the intercostal muscles, placing the respiratory muscles at a mechanical disadvantage. The work of breathing is therefore markedly increased, first on exercise, when the time for expiration is further shortened, but then, as the disease advances, at rest.

Pathophysiology of Emphysema

Destruction of alveolar walls is due to relative excess in protease (elastase) activity, or relative deficiency of ant protease (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.

Emphysema may be classified by the pattern of the enlarged airspaces as centriacinar, panacinar or paraseptal. Bullae form in some individuals. This results in impaired gas exchange and respiratory failure.

Risk Factors

- **Tobacco smoke** (indicated in almost 90% of COPD cases)
- α1-Antitrypsin deficiency—risk is even worse in combination with smoking
- Environmental factors (e.g., second-hand smoke)
- Chronic asthma—speculated by some to be an independent risk factor
- Indoor air pollution
- Occupational exposures, such as coal dust, silica
- Lung growth: childhood infections or maternal smoking
- Infections:HIV infection is associated with emphysema

Signs & Symptoms

Symptoms	Signs
• Cough • Sputum production • Dyspnea (on exertion or at rest, depending on severity) • Some patients have very sedentary lifestyles but few complaints They may avoid exertional dyspnea which is the **most common early symptom of COPD** by limiting their activity.	• **Prolonged forced expiratory time.** Timed full exhalation of vital capacity ≥6 seconds (evidence for obstruction with 75% sensitivity). • During auscultation, end-expiratory wheezes on forced expiration, decreased breath sounds, and/or inspiratory crackles • Tachypnea, tachycardia • Cyanosis • Use of accessory respiratory muscles • Hyperresonance on percussion • 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 $PaCO_2$ 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.

How To Diagnose

1. Pulmonary function testing (spirometry)

a. This is the definitive diagnostic test.

b. Obstruction is evident based on the following:

Decreased FEV1 and decreased FEV1/FVC ratio

If FEV1 is reduced to 70% of predicted value, mild disease is suggested.

If FEV1 is reduced to 50% or less of predicted value, severe disease is present.

Values in between indicate moderate disease

How To Diagnose

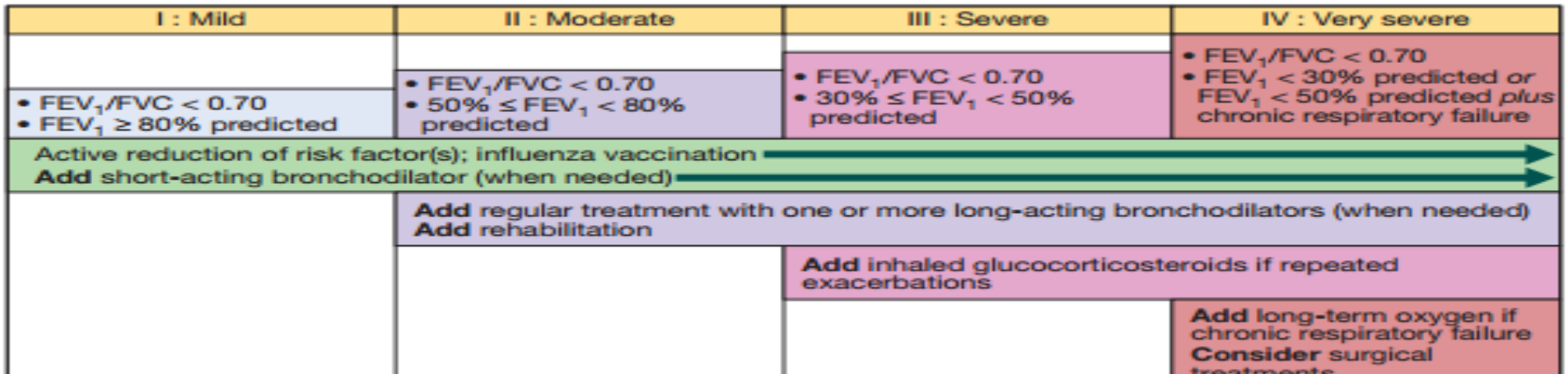
2. Chest radiograph (CXR)	4. Measure α1 antitrypsin levels	4. ABG
a. Low sensitivity for diagnosing COPD; only severe, advanced emphysema will show the typical changes, which include: Hyperinflation, flattened diaphragm, enlarged retrosternal space Diminished vascular markings	in patients with a personal or family history of premature emphysema (≤50 years old).	▪ chronic pCO2 retention ▪ decreased pO2
b. Useful in an acute exacerbation to rule out complications such as pneumonia or pneumothorax		

Management

Smoking cessation	Inhaled anticholinergic drugs (ipratropium bromide):	Inhaled β2-agonists (e.g., albuterol):	Combination of β-agonist albuterol with ipratropium bromide	Theophylline (oral)—role is controversial
• **The most important intervention** • Disease progression slowed by cessation • **prolongs the survival rate** • Sustained smoking cessation in mild to moderate COPD is accompanied by a reduced decline in FEV1 compared to persistent smokers.	• Bronchodilatos • Slower onset of action than the β-agonists, **but last longer** • is central to the management of breathlessness.	• bronchodilators • Provide symptomatic relief. Use long-acting agents (e.g., salmeterol) for patients requiring frequent use. • is central to the management of breathlessness.	• More efficacious than either agent alone in bronchodilation • Also helps with adherence to therapy (both medications in one inhaler)	• May improve mucociliary clearance and central respiratory drive • **Narrow therapeutic index, so serum levels must be monitored** • Only modestly effective and has more side effects than other bronchodilators. Occasionally used for patients with refractory COPD.

Management

Oxygen therapy	Vaccination	Pulmonary rehabilitation
▪ **Shown to improve survival and quality of life in patients with COPD and chronic hypoxemia** ▪ Some patients need continuous oxygen, whereas others only require it during exertion or sleep. ▪ Get an ABG to determine need for oxygen	▪ **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.	▪ Education, exercise, physiotherapy: A major goal is to improve exercise tolerance. ▪ Pulmonary rehabilitation improves functional status and quality of life



Acute COPD exacerbation

A persistent increase in dyspnea (not relieved with bronchodilators) Increased sputum production and cough are common.

Acute COPD exacerbation can lead to acute respiratory failure requiring hospitalization, and possibly mechanical ventilation; potentially fatal.

Management:

1. Bronchodilators (β2-agonist) alone or in combination with anticholinergics are first-line therapy
2. Systemic corticosteroids are used for patients requiring hospitalization (IV methylprednisolone is a common choice). Taper with oral prednisone on clinical improvement. Do not use inhaled corticosteroids in acute exacerbations.
3. Antibiotics (azithromycin or levofloxacin)
4. **Supplemental oxygen** is used to keep O2 saturation above 90%.
5. Noninvasive positive-pressure ventilation (NPPV) (BIPAP or CPAP): It may decrease the likelihood of respiratory failure requiring invasive mechanical ventilation.
6. Intubation and mechanical ventilation may be required if the above do not stabilize the patient. **Intubation is avoided unless there is severe, acute respiratory acidosis and CO2 retention.**

Complications

1. **Acute exacerbations**—most common causes are infection, noncompliance with therapy, and cardiac disease

2. **Secondary polycythemia** (Hct >55% in men or >47% in women)—compensatory response to chronic hypoxemia

3. **Pulmonary HTN and cor pulmonale**—may occur in patients with severe, longstanding COPD who have chronic hypoxemia

Bronchiectasis

- There is **permanent**, **abnormal dilation and destruction of bronchial walls**. **Cilia are damaged**; onset usually in childhood.
- Infection in a patient with airway obstruction or impaired defense or drainage mechanism precipitates the disease.
- Cause is identified in fewer than half of all patients.
- Less common today because modern antibiotics are used for respiratory infections.

Bronchiectasis

Causes	clinical features
1. **Cystic fibrosis** (CF) is **most common cause** of bronchiectasis (accounts for half of all cases) 2. Ciliary dysfunction syndromes Primary ciliary dyskinesia (immotile cilia syndrome) Kartagener's syndrome (sinusitis and transposition of the viscera) 3. Primary hypogammaglobulinaemia 4. Pneumonia (complicating whooping cough or measles) 5. Primary TB 6. Inhaled foreign body 7. Suppurative pneumonia 8. Pulmonary TB 9. Allergic bronchopulmonary aspergillosis complicating asthma 10. Bronchial tumours 11. Infection, humoral immunodeficiency (abnormal lung defense), airway obstruction	1. **Chronic cough** with **large amounts of mucopurulent**, **foul-smelling sputum** 2. Dyspnea 3. **Hemoptysis**—due to rupture of blood vessels near bronchial wall surfaces; usually mild and self-limited, but sometimes can be brisk and present as an emergency 4. **Recurrent or persistent pneumonia** **5. Pleuritic pain** **6. Halitosis:**frequently accompanies purulent sputum **7. General debility**: difficulty maintaining weight, anorexia, exertional breathlessness **5. Coarse crakles can be heard**

Bronchiectasis

Diagnosis	Treatment
▪ **High-resolution CT scan is the diagnostic study of choice.** ▪ PFTs reveal an obstructive pattern. ▪ CXR is abnormal in most cases, but findings are nonspecific. ▪ Bronchoscopy applies in certain cases.	**1. Antibiotics*** for acute exacerbations superimposed infections are signaled by change in quality/quantity of sputum, fever, chest pain, etc. Gentamycin or tobramycin Twice daily **2. Bronchial hygiene is very important.** I. Hydration II. Chest physiotherapy (postural drainage, chest percussion) to help remove the mucus III. Inhaled bronchodilators *in particular *Pseudomonas* species, antibiotic therapy becomes more challenging and should be guided by the microbiological sensitivities. For*Pseudomonas*, oral ciprofloxacin (500–750 mg twice daily) or ceftazidime by intravenous injection or infusion (1–2 g 3 times daily)

MCQs

1. Which type of pleural effusion usually comes with Empyema ?
2. Exudate, 100% lymphocytes
3. Bloody effusion
4. Milky appearing

2. A 60-year-old male has had a chronic cough for over five years with clear sputum production. He smoked one pack of cigarettes per day for 20 years and continues to do so. X-ray of the chest shows hyperinflation without infiltrates. Arterial blood gases show a pH of 7.38, PCO2 of 40 mm Hg, PO2 of 65 mm Hg. Spirometry shows a FEV1/FVC of 65%. The most important treatment modality for this patient would be :

3. Oral corticosteroids
4. Home oxygen
5. Broad-spectrum antibiotics
6. Smoking cessation program

3. A 50-year-old male with emphysema and a chest x-ray that has shown apical blebs develops the sudden onset of shortness of breath and left-sided pleuritic chest pain. Pneumothorax is suspected. Physical examination findings that would confirm the diagnosis are

A. Localized wheezes at the left base
B. Hyperresonance of the left chest, decreased breath sounds
C. Increased tactile fremitus on the left side
D. Decreased breath sounds left side

MCQs

4. Which of the following is a symptom of COPD ?

A. Severe headache
B. Clogged sinuses
C. Excess sputum
D. All of the above

5. COPD cuts the airflow in the lungs because

A. Bronchial tubes become inflamed and thickened
B. Walls between air sacs are destroyed
C. Either of them
D. Neither of them

6. 70 year old patient with COPD requires 2L/min of nasal O2 to treat his hypoxia,which is sometimes associated with angina. The patient develops pleuritic chest pain ,fever and purulent sputum . While using his Oxygen at an increased flow of 5 L/min , he becomes stuporous and develops respiratory acidosis with CO2 retention and worsening hypoxia. What would be the most appropriate next step in the management of this pt ?

A. stop oxygen
B. begin sodium bicarbonate
C. treat with antibiotics
D. intubate and begin mechanical ventilation

MCQs

7. 55 year old woman with long standing COPD and episodes of chronic bronchitis complain of increasing sputum production which now occurs in daily basis. Sputum is thick and daily sputum production has dramatically increased over several months . There are flecks of blood in the sputum. The pt has lost 8 Ib . Fever and chills are absent and sputum culture have not revealed specific pathogen . Which of the following the most likely cause of pt symptoms ?

A. pulmonary TB
B. Exacerbation of chronic bronchitis
C. Bronchiectasis
D. Carcinoma of the lung

Answers: 1- A 2- D 3-A 4-C 5-C 6-D 7-C

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Medicine is a science of uncertainty and an art of probability