Chronic obstructive pulmonary disease (COPD) and bronchiectasis

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Acinus: respiratory bronchiole, alveolar ducts and alveoli
- the site of gas exchange (functioning unit)

- **Obstructive lung diseases:** - associated with difficulty in exhaling all air from lungs (getting air out of the lungs)
  - due to partial or complete obstruction in airway
  - increase in lung compliance (ability to expand)
  - decrease in lung elasticity
  - include: 1- COPD  2- bronchiectasis  3- asthma

- **Restrictive lung diseases:** - patients can not fully fill the lungs with air (getting air in the lungs)
due to reduced lung capacity
(restricted expanding)
- lung compliance is decreased
- elasticity is increased

**Pulmonary function tests in obstructive lung diseases:**
1- Forced expiratory volume in 1 sec (FEV 1sec) is decreased
   - Normal FEV 1sec = 4L
   - less than 2 L in obstructive diseases.
2- Forced vital capacity (FVC) is decreased
   - Normal is 5 L
   - less than 4 L in obstructive diseases
3- FEV 1sec : FVC ratio is decreased
   - Normal is 4:5 = 80%
   - In obstructive diseases (1:3 = 33%)

**COPD:**
- include: 1- emphysema
  2- chronic bronchitis
- in USA, COPD affects more than 10% of adult population and is the fourth leading cause of death.
- COPD associated with *irreversible* airflow obstruction (but asthma, is characterized largely by *reversible* airflow obstruction).
1- **Emphysema:**
- is abnormal permanent enlargement of the airspaces distal to the terminal bronchioles (acinus) due to destruction of the walls and loss of elastic tissue

**Types of Emphysema:**
- is classified according to its anatomic distribution within the lobule into:
  1. centriacinar
  2. panacinar
  3. distal acinar
  4. irregular

1- **Centriacinar (Centrilobular) Emphysema:**
- is the most common type
- involves the central or proximal parts of the acini (respiratory bronchiole)
- more common in the upper lobes (in the apical segments)
- associated with cigarette smoking

2- **Panacinar (Panlobular) Emphysema:**
- less common than centriacinar
- In this type the acini are uniformly enlarged
- occur more commonly in the lower lobes
- associated with α1-antitrypsin deficiency.

3- **Distal Acinar (Paraseptal) Emphysema:**
- involves the distal part of acini
- beneath the pleura, near interlobular septa
- more common in the upper lobes
- underlies many cases of spontaneous pneumothorax

4- **Irregular Emphysema:**
- airspace enlargement with fibrosis
- usually clinically asymptomatic

**Pathogenesis:** two mechanisms involved:

1. **protease- antiprotease mechanism:**
   - emphysema arises as a consequence of imbalances between pulmonary proteases and antiproteases
   - the imbalance results in tissue destruction and loss of alveolar walls
   - proteases secreted by neutrophils (elastase)
   - antiproteases: present in serum, tissue fluids, and macrophages (α1-Antitrypsin)
     - tobacco smoke (and other factors: air pollution, genetics (α1-Antitrypsin deficiency) causes:
       1. recruitment of inflammatory cells (neutrophils, macrophages)
       2. release of elastase
       3. free radical release that inactivating antitrypsin
   - imbalance between proteases and antiproteases
   - leading to tissue damage with enlargement of airspaces

- those with congenital antitrypsin deficiency are at risk to develop emphysema at younger age if they smoke

2. **Oxidant – antioxidant mechanism:**
   - in lungs present antioxidants (dismutase)
   - they prevent oxidative tissue damage
   - tobacco induces free radicals release that deplete antioxidant in lung and causes tissue damage
**Morphology:** Centriacinar emphysema: appears as holes in the lung tissue

Panacinar emphysema: appears as holes in the lung tissue

**Microscopically:** There is marked enlargement of airspaces, with thinning and destruction of alveolar septa.

**Clinical Course:**
- Dyspnea
- cough
- wheezes
- Weight loss
- Pulmonary function tests reveal: - reduced FEV1 - reduced FVC - reduced FEV1 to FVC ratio
- Radiology (CT-scan) can show changes in lung
  (Hyperlucent lung fields)
2- Chronic Bronchitis:
- is defined as a persistent productive cough for at least 3 consecutive months in at least 2 consecutive years
- is common among cigarette smokers
- **Pathogenesis:**
  - caused by cigarette smoking
  - also associated with air pollution, infection, genetic factors
  - These irritants induce: -hypertrophy of mucous glands
    - increase in goblet cells
  - mucus hypersecretion develops
  - bronchial or bronchiolar mucus plug, inflammation (chronic bronchitis)
  - involvement of bronchioles results in peribronchiolar fibrosis and airway obstruction (chronic bronchiolitis: dyspnea)

**Morphology:**
- hypertrophy of mucus glands
- increase in goblet cells
- inflammation and fibrosis
- squamous metaplasia or dysplasia of bronchial epithelium

**Figure:**
- marked thickening of the mucous gland layer
- squamous metaplasia of lung epithelium
Clinical course:
- productive cough
- dyspnea (bronchiolitis)

Complications of COPD:
1. secondary pulmonary hypertension: hypoxia-induced pulmonary vascular spasm
2. respiratory failure
3. right-sided heart failure (core pulmonale)
4. recurrent infections

Bronchiectasis:
- permanent dilation of bronchi and bronchioles caused by destruction of the muscle and elastic tissue, resulting from or associated with chronic necrotizing infections

Pathogenesis:
- It is secondary to: 1. persisting infection (Necrotizing, or suppurative, pneumonia tuberculosis)
2. airway obstruction (tumors, foreign bodies, mucus impaction)
- Either of these two processes may come first:
  * 1. obstruction leads to
    2. impairment of clearance of secretions
    3. secondary infection, leading to
4- damage, weakening and dilation

*1- persistent necrotizing infections lead to
2- inflammation with obstruction of secretions leading to
3- damage, weakening and dilatation

Morphology:
- common in lower lobes
- either localized (tumor, foreign body) or diffuse (infection)
- dilated airspaces on gross examination
- microscopically: - inflammatory process
  - ulceration (loss of lining epithelium)
  - Fibrosis of the walls
  - lung abscess (necrosis)

Clinical manifestations:
- severe, persistent cough with purulent sputum (may contain blood)
- cyanosis (hypoxemia, hypercapnia)
- complications: 1- pulmonary hypertension
  2- (rarely) cor pulmonale
  3- Metastatic brain abscesses
  4- amyloidosis (very rare)
- diagnosis depends on history and radiologic demonstration of bronchial dilatation

Thank you