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 (Hyperluscent 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

- loss of pulmonary capillary

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