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