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