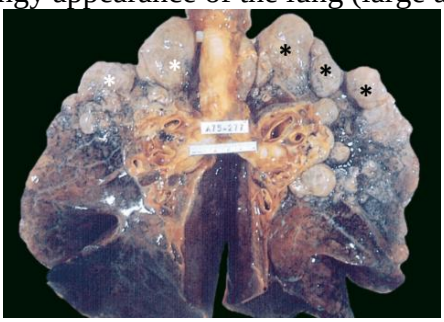
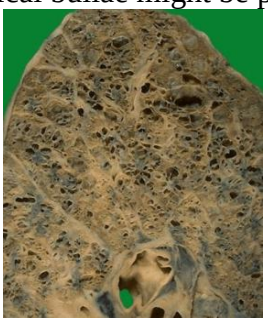
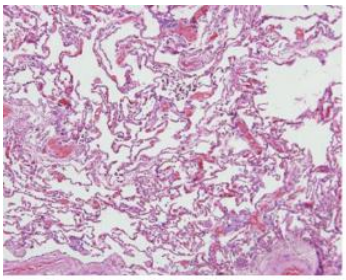
Unit II – Problem 4 – Pathology: Obstructive Lung Diseases

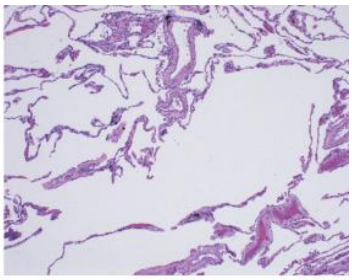
- Emphysema

- **Definition**: destruction of alveolar air sacs. Obstruction is physiologic instead of being anatomic, When walls of alveoli are destructed → elastic recoil of the lung is lost → air is trapped and cannot be expired because obstruction will be created as walls of bronchioles are dragged with air that the patient is trying to expire.
- **Morphology and histopathology:**
  - ✓ Gross: spongy appearance of the lung (large apical bullae might be present).

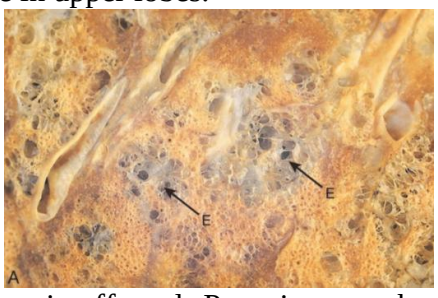



  - ✓ Histology: huge dilated air sacs separated by thin septa with decrease in capillary bed.

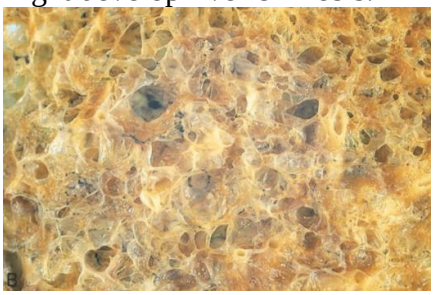

Normal lung


Emphysematous lung

- **Cause**: imbalance between proteases and antiproteases
  - ✓ Overproduction of proteases: occurs in smokers mainly (centriacinar emphysema).
  - ✓ Decreased antiproteases: patients with α_1-antitrypsin deficiency (panacinar).
- **Types:**
  - ✓ Centriacinar (most common: 95%): in central parts of acinus. Acinus is considered as the functional unit of the lung. Centriacinar emphysema is smokers is more severe in upper lobes.

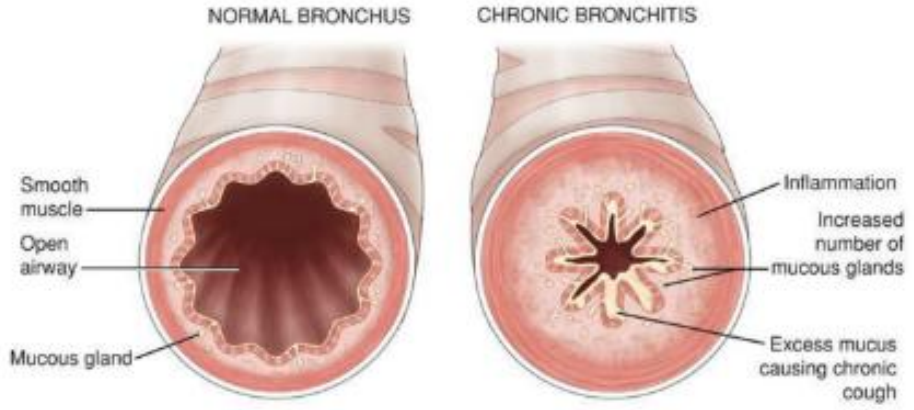


  - ✓ Panacinar: whole acinus is affected. Panacinar emphysema in α_1-antitrypsin deficiency is more severe in lower lobes. Notice that patients with α_1-antitrypsin deficiency might develop liver cirrhosis.



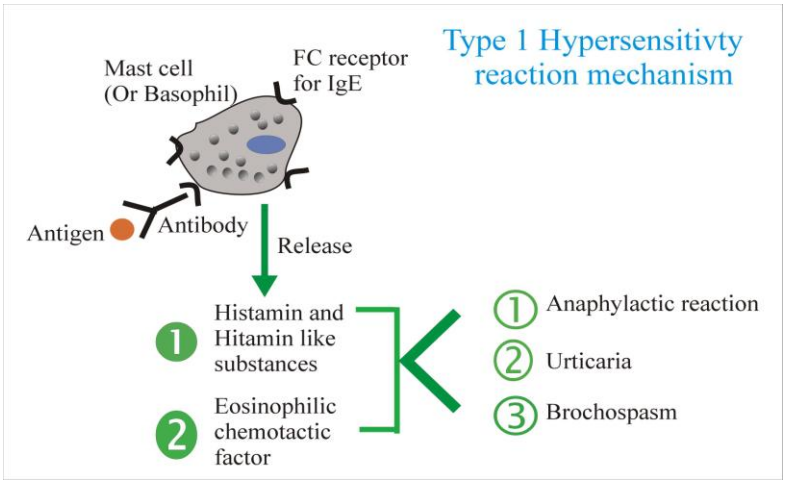
- **Clinical presentation:**
  - ✓ Dyspnea.
  - ✓ Cough with minimal sputum.
  - ✓ Prolonged expiration with pursed lips (pink-puffers).
  - ✓ Weight loss.
  - ✓ Increased antero-posterior diameter of the chest (barrel-chest).
- **Late complications:**
  - ✓ Hypoxemia: because capillaries will also be destroyed along with air sacs → no more gas exchange.
  - ✓ Cor pulmonale.

- **Chronic bronchitis:**
  - **Definition**: chronic productive cough lasting ≥ 3 months over a minimum of 2 years (a lot of mucus is produced in these patients).
  - **Cause**: smoking → lung responds to pollutants by increasing the amount of mucus produced (through hyperplasia and hypertrophy of mucinous glands in submucosa). This mucous will be coughed up and some will go back to plug the airways.
  - **Morphology and histopathology:**
    - ✓ Gross: hyperemia, swelling and edema of mucous membranes ± mucinous or mucopurulent secretions.
    - ✓ Histology: chronic inflammation of airways and enlargement of mucus-secreting glands of the trachea and bronchi.
    - ✓ Reid index: measuring thickness of mucinous glands relative to the entire thickness of the wall. In chronic bronchitis, it becomes > 50%.



  - **Clinical features:**
    - ✓ Productive cough.
    - ✓ ↑$PaCO_2$ and PaO_2 (cyanosis).
    - ✓ ↑ Risk of infection (blockage of tube causes ↑ risk of infection distal to site of blockage).
    - ✓ ↑ risk of cor pulmonale.

- **Asthma:**
  - **Definition**: reversible airway bronchoconstriction.
  - **Cause**: allergic stimulant resulting in type-I hypersensitivity reaction.
  - Presents in childhood and often associated with other allergic conditions such as allergic rhinitis and eczema.
  - **Pathogenesis**: genetically susceptible individuals are exposed to allergens which will induce Th-2 phenotype in helper T-cells

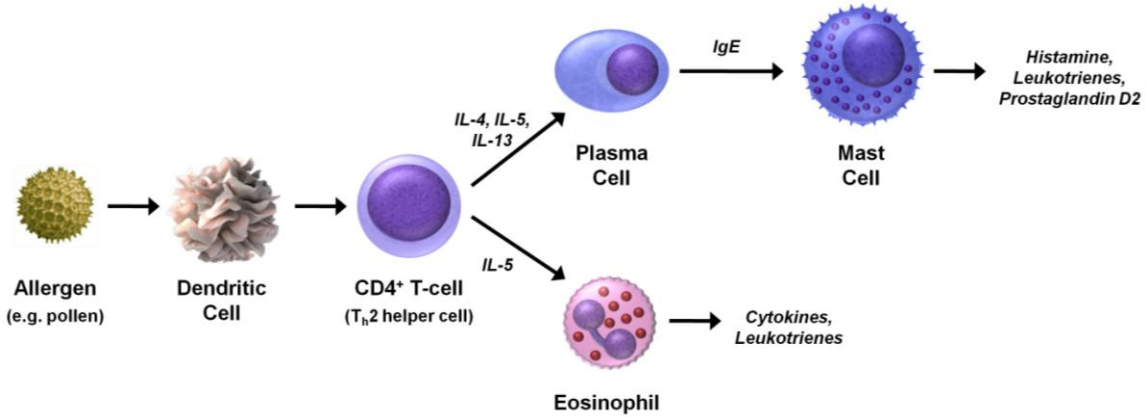


(CD4+). These cells in turn will secrete:

- ✓ IL-4: for production of IgE.
- ✓ IL-5: calls in eosinophils.
- ✓ IL-10: inhibits production Th-1 subtype of helper T-cells.

When patients are re-exposed to allergen, it will cross-link surface IgE present on surface of mast cells. Therefore, mast cells will get activated:

- ✓ Release of pre-formed histamine granules which will cause vasodilation and increased vascular permeability.
- ✓ Production of leukotrienes (C4, D4 and E4): causing vasoconstriction, increased vascular permeability and bronchoconstriction.



- **Clinical presentation (episodic):**
  - ✓ Dyspnea and wheezing.
  - ✓ Productive cough.
- **Non-allergic causes of asthma:**
  - ✓ Exercise.
  - ✓ Viral infection.
  - ✓ Aspirin intolerant asthma.
  - ✓ Occupational exposure.

- **Bronchiectasis:**

- **Definition**: permanent dilation of bronchioles and bronchi.
- Loss of airway tone results in air trapping (air will be accelerated, rolling around in the large airway without being expired).
- **Cause**: necrotizing inflammation with damage to airway walls which occurs in
  - ✓ Cystic fibrosis.
  - ✓ Kartagener syndrome.
  - ✓ Tumor of foreign body.
  - ✓ Necrotizing infection.
  - ✓ Allergic bronchopulmonary aspergillosis (occurring more in patients with asthma and those with cystic fibrosis).
- **Clinical features:**
  - ✓ Cough and dyspnea.
  - ✓ Foul-smelling sputum.
- **Complications:**
  - ✓ Hypoxemia.
  - ✓ Cor pulmonale.
  - ✓ Secondary amyloidosis.