Patho – RS

Atelectasis

- Atelectasis (Collapse)
  - **Inadequate expansion** of air spaces → loss of lung volume → shunting of blood to veins → V/Q imbalance → hypoxia
  - Should be treated quickly to prevent hypoxemia and superimposed infection
  - 1) Resorption atelectasis (Obstruction → air can't reach distal airways)
    - Most common causes
      1. Obstruction of a bronchus
      2. Postoperatively due to intrabronchial mucous or mucopurulent **plugs**
      3. Foreign body aspiration
      4. Bronchial asthma, bronchiectasis, chronic bronchitis, intrabronchial tumor
  - 2) Compression atelectasis (accumulation of fluid blood, blood, or air in pleural cavity)
    - Frequent causes:
      1. Pleural effusion due to congestive heart failure
      2. Pneumothorax
      3. (at the base) **Failure to breath deeply** in bedridden patients, in patients with ascites and during and after surgery
  - 3) Contraction atelectasis (Cicatrization atelectasis)
    - Caused by local or diffuse **fibrosis** (affecting lung or pleura) → decrease lung expansion
    - **Only irreversible form** of atelectasis

Thromboembolism 1

- Pulmonary thromboembolism
  - Blood clots in lung are **almost always embolic**
  - 95% of emboli are from thrombi within **large deep veins of legs** (involving popliteal vein and larger veins above)
    - Risk factors for deep vein thrombosis DVT
      - Prolonged bed rest (immobilized legs)
      - Surgery (<orthopedic surgery on knee or hip)
      - Sever trauma (e.g. burns or multiple fractures
      - Congestive heart failure
      - High estrogen in women (Period around childbirth and oral contraception)
      - Disseminated cancer
      - Primary disorders of hypercoagulability (factor V Leiden)
  - Prognosis of pulmonary thromboembolism depend on 1. **size of embolus** → size of artery occluded 2. **cardiopulmonary status**
  - Consequences of pulmonary artery occlusion:
    - Increase in pulmonary artery pressure from blockage of flow (can also be due to vasospasm.)
    - Ischemia of downstream pulmonary parenchyma.
  - If a **Large embolus embeds in main pulmonary artery** of its major branches or lodge along the bifurcation (saddle embolus) → Immediate hypoxia or acute right-sided heart failure (cor pulmonale) → No time for morphological changes in lung → **sudden death**

Thromboembolism 2

- Smaller emboli occlude smaller arteries
  - With **good circulation** → lung parenchyma maintained but ischemic damage to epithelial cells → **alveolar hemorrhage**
  - **Bad cardiovascular status** (e.g. congestive heart failure) → **Infarction** (more peripheral emboli → higher risk of infarction so ~75% of infarcts in lower lobes, more than 50% multiple)
- Morphology of infarcts
  - **Wedge-shaped**, base at pleural surface, apex toward hilum of lung
  - In the early stages: Hemorrhagic, raised, **red-blue** areas of **coagulative necrosis**... pleural surface covered by fibrinous exudate.. occluded vessel is at apex
  - Within 48 hours: Lysis of RBC → infarct becomes **red-brown** as hemosiderin is produced
  - After a while: Fibrous replacement occurs at margins→ gray-white peripheral zone → → infarct turns to **scar**
- Clinical consequences of pulmonary thromboembolism
  - **60-80% are clinically silent** because emboli is **small** → bronchial circulation keeps parenchyma alive → emboli mass **removed by fibrinolytic activity**
  - 5% of cases: Sudden **death**, acute **cor pulmonale** or **cardiovascular shock**, this happens when more than 60% of pulmonary vessels are obstructed (by **one large embolus or multiple simultaneous small emboli**)
  - 10-15% of cases: small to medium pulmonary branches occluded → pulmonary **infarction** (if circulatory insufficiency is present) → present with **dyspnea**
  - < 3% of cases: **recurrent showers of emboli** → pulmonary **hypertension** → **chronic** right-sided heart failure → → vascular **sclerosis** → **worsening dyspnea**

Emphysema 1

- Emphysema
  - Permanent enlargement of air spaces distal to the terminal bronchioles AND Destruction of their walls, without significant fibrosis
  - Grouped with Chronic bronchitis as Chronic Obstructive Pulmonary disease (COPD)
  - 4 major types:
    - 1) Centriacinar (centrilobular) emphysema
      - Respiratory bronchioles are affected (Central/proximal part) Alveoli not affected
      - More in upper lobes (especially apical)
      - Most common in smokers (in association with chronic bronchitis)
      - Macroscopic: lungs are deeper pink than in panacinar and less voluminous, upper two thirds of lungs more severely affected
    - 2) Panacinar (panlobular) emphysema
      - Acini uniformly enlarged (ALL)
      - More in lower lobes
      - Associated with alpha1-anti-trypsin deficiency
        - A1-anti trypsin present in: serum, tissue fluids. macrophages
          - Major inhibitor of proteases (elastases)
          - Secreted by neutrophils
          - Encoded by gene in Pi locus on chromosome 14
      - Macroscopic: produces pale, voluminous lungs (can obscure the heart at autopsy)

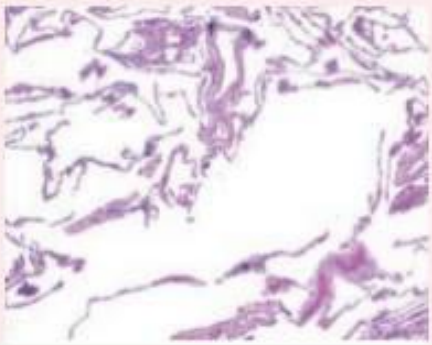
Emphysema 2

- 3) Distal acinar (paraceptal) emphysema
  - Distal part of acinus involved (proximal normal)
  - More obvious next to pleura, along septa, and at the margins of lobules
  - Occurs next to areas of fibrosis, scarring and atelectasis
  - More severe in upper half
  - Characteristic finding: multiple, neighboring enlarged air spaces (diameters range from <0.5 to >2 cm)
  - Sometimes forms cystic structures that may give rise to bullae
  - Unknown cause
  - Most common in young adults presenting with spontaneous pneumothorax
- 4) Irregular emphysema
  - Acinus irregularly involved
  - Almost always associated with scarring
  - Clinically asymptomatic
- Factors that influence development of emphysema
  1. Inflammatory cells and mediators
  2. Protease-anti-protease imbalance
  3. Oxidative stress
  4. Airway infection

Emphysema 3

▼ Histological examination

- Destruction of alveolar wall (without fibrosis) → enlarged air spaces
- Alveolar loss
- Number of alveolar capillaries diminished
- Possible deformation of terminal and respiratory bronchioles because of loss of septa
- In advanced disease: Bronchial inflammation and submucosal fibrosis



▼ Clinical features

Dyspnea, Weight loss, Barrel chest, Pink buffers

When Obese and with chronic bronchitis → blue bloaters

COPD → Secondary hypertension

Respiratory failure or right-sided heart failure → death

▼ Conditions related to emphysema

1. Compensatory emphysema
2. Obstructive overinflation
3. Bullous emphysema
4. Mediastinal (interstitial) emphysema

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