PATHOLOGY OF RESPIRATORY SYSTEM

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Upper respiratory tract
- Nose
- Pharynx (Throat)
- Larynx
- Trachea

Lower respiratory tract
- Lung
  - Bronchi
  - Bronchiole
  - Alveoli
CONTENT

- Upper respiratory tract
  - Sinus
    - Sinusitis
  - Larynx
    - Vocal nodule
    - Laryngeal malignancy: laryngeal carcinoma
Lower respiratory tract
- Pulmonary infections
- Atelectasis
- Pulmonary edema
- Acute respiratory distress syndrome
- Obstructive pulmonary diseases
- Disease of vascular origin: pulmonary embolism
- Tumors
- Diseases of pleura

UPPER RESPIRATORY TRACT
SINUSITIS

- Infection of sinuses, associated with acute or chronic rhinitis
- Bacteria or fungus
- s/s: pain at sinus area, post nasal dripping, chronic cough
- Rx: antibiotic

VOCAL NODULES OF LARYNX

- Smoking or singer
- Small nodule at true vocal cord
- Hoarseness
- Benign lesion
VOCAL NODULES OF LARYNX

CARCINOMA OF LARYNX

- Associated with smoking and alcohol
- Squamous cell carcinoma
- Hoarseness or neck mass if metastasis to cervical lymph node
- Treatment by surgery or combined with radiation
CARCINOMA OF LARYNX

LOWER RESPIRATORY TRACT
INFECTIOUS LUNG DISEASE

- Pneumonia
  - Community acquired pneumonia
  - Hospital acquired pneumonia (nosocomial pneumonia)
  - Atypical pneumonia
  - Aspiration pneumonia
- Lung abscess
- Pulmonary tuberculosis

COMMUNITY ACQUIRED PNEUMONIA (CAP)

- Predisposing factors
  - old age
  - chronic disease (COPD, CHF, DM)
  - immune deficiency
  - decreased or absent splenic function
eg. sickle cell disease, post splenectomy
  - risk for infection to encapsulated bacteria eg. pneumococcus
CLINICAL COURSE

- Abrupt onset of high fever with chills
- Mucopurulent productive cough
- Dyspnea
- Involve pleura: pleuritic pain
- PE: crepitation, decrease breath sound

CAUSE OF CAP

- Streptococcus pneumoniae (most common)
- Haemophilus influenzae: Acute exacerbation of COPD
- Staphylococcus aureus: IVDU
- Klebsiella pneumoniae: Chronic alcoholism
- Pseudomonas aeruginosa
GROSS EXAMINATION

- Gross patterns of anatomical distribution
  - Bronchopneumonia: patchy consolidation along bronchi and bronchioles
  - Lobar pneumonia: consolidation involves entire lobe of lung
Bronchopneumonia

Lobar pneumonia (gray hepatization)

- Suppurative neutrophils rich exudate in bronchi, bronchioles and alveoli
Bronchopneumonia  Lobar pneumonia

HOSPITAL ACQUIRED PNEUMONIA

- Nosocomial pneumonia
- Common patients with any conditions
  - severe underlying disease
  - mechanical ventilator
  - immunosuppression
  - prolonged antibiotic therapy
  - invasive access devices eg. intravascular catheters
- Serious and life-threatening
- Common organisms
  - Gram negative bacteria
    - Pseudomonas aeruginosa
    - Acinetobacter baumannii
    - Enterobacteriaceae
  - Gram positive bacteria
    - Staphylococcus aureus

HOSPITAL ACQUIRED PNEUMONIA

- Clinical course (similar CAP)
  - Abrupt onset of high fever with chills
  - Mucopurulent productive cough
  - Dyspnea
  - PE: crepitation, decrease breath sound of affected lungs
ATYPICAL PNEUMONIA

- Mycoplasma or Viral pneumonia
- Common in children and young adults
- Most common organisms
  - Mycoplasma pneumonia
  - Virus: influenza virus type A or B, respiratory syncytial virus, adenovirus, rhinovirus, HSV, varicella virus

ATYPICAL PNEUMONIA

- Clinical course
  - fever
  - headache, muscle aches
  - cough +/−
- PE: non specific
ATYPICAL PNEUMONIA

- Gross
  - patchy or may involve whole lobes
  - affected area: red blue, congested
- Microscopic
  - alveolar septa are wide, edema, mononuclear cell infiltration (lymphocytes, plasma cells, histiocytes)
ASPIRATION PNEUMONIA

- Common in patients with some conditions
  - Unconscious patients or during repeated vomiting (abnormal gag reflex or swallowing reflex that predispose to aspiration)

ASPIRATION PNEUMONIA

- Aspirated food and gastric content (chemical, gastric acid, oral flora bacteria) induces to pneumonia
- Clinical course
  - high fever, cough, dyspnea
- Often more than one organism (mixed aerobes and anaerobes)
ASPIRATION PNEUMONIA

- Gross
  - bronchopneumonia
  - lobar pneumonia
- Microscopic
  - neutrophils rich exudate, often necrosis in alveoli
- Complication
  - lung abscess (common)

NECROTIZING PNEUMONIA AND LUNG ABSCESS

- Local suppurative process in the lung characterized by necrosis of lung tissue
- Organisms (aerobes+anaerobes)
  - aerobes: streptococci, staphylococci, klebsiella, pseudomonas, E. coli
  - anaerobes: peptostreptococcus, bacteroides, fusobacterium spp.
NECROTIZING PNEUMONIA AND LUNG ABSCESS

- Clinical course
  - high fever
  - cough with foul smelling purulent sputum
  - chest pain
  - weight loss
LUNG ABSCESS

PULMONARY TUBERCULOSIS

- Increase risk for TB
  - diabetes mellitus
  - Hodgkin lymphoma
  - chronic lung disease (eg. Silicosis)
  - chronic renal failure
  - malnutrition
  - alcoholism
  - immunocompromised host
PULMONARY TUBERCULOSIS

- Caused by M. tuberculosis
- Most infections are acquired by person-to-person transmission of airborne droplets of organisms from active case to a susceptible host
- TB infection leads to development of delayed hypersensitivity to M. tuberculosis antigen, which can be detected by tuberculin skin test (TT)
- Classified as 2 types
  - Primary pulmonary tuberculosis
  - Secondary pulmonary tuberculosis

PRIMARY PULMONARY TUBERCULOSIS

- Usually a self-limited infection seen in children and adult
- Most patients are asymptomatic
- May be present with fever, malaise, weight loss, cough, and occasional hemoptysis
- Clinical course
  - Most self-limited and scar with calcification in chest radiography
  - Some patients with progression become have extensive lung damage with cavity
  - May be lymphatic or hematogenous spread to other organs eg, liver, spleen, pancreas → miliary TB
PRIMARY PULMONARY TUBERCULOSIS

- Primary infection at lungs composed of “Ghon complex”
  - Lung infection at lower segment of upper lobe or upper segment of lower lobe → Ghon focus
  - Infection of hilar node

Ghon complex
SECONDARY PULMONARY TUBERCULOSIS

- Reactivation or reinfection of primary TB with asymptomatic
- Common infection at high O2 (apex of lung)
- Severe lung damage and produces cavity

SECONDARY PULMONARY TUBERCULOSIS

- Symptom
  - low grade fever, chronic cough, night sweats, anorexia, weight loss
  - may be hemoptysis, pleural effusion
- Clinical course
  - recovery if normal immune or treatment
  - lymphatic or hematogenous spread to other organs eg, liver, spleen, pancreas → miliary TB
SECONDARY PULMONARY TUBERCULOSIS

cheese like lesion with cavity at apex
( reddish bacilli from AFB stain)
ATELECTASIS

- Incomplete expansion of lungs or collapse of previously inflated lungs, producing areas of relatively airless pulmonary parenchyma
- Acquired atelectasis divided 3 types
  - Resorption (obstruction) atelectasis
  - Compression atelectasis
  - Contraction atelectasis
ATELECTASIS

- Clinical features
  - Reversible disorders, except contraction atelectasis
  - Significant atelectasis reduces oxygenation and predisposes to infection
  - Small area or slowly develop: minor symptom or asymptomatic
  - Large area of rapid lung collapse: sudden onset chest pain, dyspnea, cyanosis, hypotension, tachycardia, shock
PULMONARY CONGESTION AND EDEMA

- Pulmonary congestion
  - Increases intravascular blood
- Pulmonary edema
  - Fluid in alveoli
- Hemodynamic cause
  - Increased hydrostatic pressure
  - Decreased oncotic pressure
  - Lymphatic obstruction
- Edema due to microvascular injury
  - Infection
  - Inhale gas or liquid aspiration
  - Drugs and chemicals
  - Shock, trauma, radiation

Pulmonary Edema in congestive heart failure
Pulmonary congestion and edema

Heart failure cells
ACUTE RESPIRATORY DISTRESS SYNDROME

- Acute lung injury (non-cardiogenic pulmonary edema)
- Abrupt onset of hypoxemia and diffuse infiltrates in the absence of cardiac failure
- Diffuse alveolar damage (DAD): inflammation-associated increase in vascular permeability, epithelial and endothelial cell death

The chest radiographs shows a diffuse alveolar infiltration of both lungs
Gross appearance:

The affected lungs are heavy, firm, red, and boggy.

Microscopic pictures:

Interstitial and intra-alveolar edema, hyaline membranes lining the alveolar walls.
- Direct lung injuries:
  - diffuse pulmonary infections (virus)
  - oxygen toxicity
  - inhalation of toxins and other irritants
  - aspiration of gastric contents

- Systemic conditions:
  - septic shock and shock associated with trauma
  - hemorrhagic pancreatitis
  - burns
  - complicated abdominal surgery
  - narcotic overdose
  - hemodialysis
  - cardiac surgery
Pulmonary artery obstruction caused by emboli

The usual cause – thrombosis in the deep vein of the leg

Clinical cause depend on size of the emboli and circulatory condition of the patient
Large emboli at the bifurcation as a saddle embolus

A wedge shaped hemorrhagic infarct with the apex pointing toward the hilus of the lung

- Hypoxemia
- Chest pain, sudden dyspnea, cough, and shock
- Bed ridden
### OBSTRUCTIVE PULMONARY DISEASE

<table>
<thead>
<tr>
<th>Disease</th>
<th>Anatomic Site</th>
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<tbody>
<tr>
<td>Emphysema</td>
<td>Acinus</td>
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<tr>
<td>Chronic bronchitis</td>
<td>Bronchus</td>
</tr>
<tr>
<td>Asthma</td>
<td>Bronchus</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td>Bronchus</td>
</tr>
</tbody>
</table>

- Increase in resistance to airflow owing to partial or complete obstruction at any level from trachea, bronchi, terminal bronchioles and respiratory bronchioles.
- Expiratory airflow obstruction result from anatomic airway narrowing in asthma, or from loss of elastic recoil of lung in emphysema.
EMPHYSEMA

- Irreversible enlargement of airspaces distal to terminal bronchioles (respiratory bronchioles, alveolar ducts and alveoli)
- Accompanied by destruction of their walls and without obvious fibrosis
Pathogenesis of emphysema
- Protease-antiprotease theory
- Oxidant-antioxidant imbalance
PATHOGENESIS OF EMPHYSEMA

- Oxidant-antioxidant imbalance
  - normal lung contains a healthy antioxidants (superoxide, dismutase, glutathione) that keep oxidative damage to minimum
  - tobacco smoke contains abundant reactive $\text{O}_2$ species (free radicals), which deplete antioxidant mechanisms, thereby inciting tissue damage
  - activated neutrophils in alveoli pool of free radicals result alveolar wall damage

Centriacinar emphysema  Panacinar emphysema
BULLOUS EMPHYSEMA

- Large subpleural blebs or bullae and near apex (>1 cm in diameter)
- Can occur in any form of emphysema
- Sometime in relation to old tuberculous scarring
- Rupture of bullae may give rise to pneumothorax
- Hyperinflation lung
- Long narrow heart shadow
- Flat diaphragm
EMPHYSEMA

- Clinical course
  - at least 1/3 of functioning lung parenchyma damage result clinical presentation
  - insidious onset of dyspnea, cough, wheezing, weight loss
  - barrel chest, prolonged expiration, sits forward in a hunched-over position, breathes through pursed lips
  - cor pulmonale is late complication

- Cause of death:
  - respiratory acidosis and coma
  - right sided heart failure
  - massive lung collapse secondary to pneumothorax

- Diagnosis
  - History, physical examination, clinical presentation and spirometry
**CHRONIC BRONCHITIS**

- Persistent cough with sputum production for at least 3 months in at least 2 consecutive years, in absence of any other identifiable cause
- Long term inflammation of bronchi by chronic irritation from agents
- Common among habitual smokers and inhabitants of smog-laden city

**CHRONIC BRONCHITIS**

- Earliest feature is hypersecretion of mucus in large airways associated with hypertrophy of submucosal gland in trachea and bronchi
- Chronic bronchitis persist: marked increase in goblet cells of small airway (bronchi, some bronchioles) leading to excessive mucus production
- Cigarette smoke predisposes to infection, that interferes ciliary action of respiratory epithelium or direct damage to epithelium, and inhibits ability of bronchial and alveolar leukocytes to clear bacteria or virus
Increase in size and numbers of submucosal mucus glands of bronchi

CHRONIC BRONCHITIS

- Clinical feature
  - persistent cough productive of sputum
  - continued smoking $\rightarrow$ hypoxia, cyanosis
  - long standing severe chronic bronchitic
    leads to cor pulmonale with cardiac failure
<table>
<thead>
<tr>
<th></th>
<th>Predominant Bronchitis</th>
<th>Predominant Emphysema</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>40-45</td>
<td>50-75</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>Mild; late</td>
<td>Severe; early</td>
</tr>
<tr>
<td>Cough</td>
<td>Early; copious sputum</td>
<td>Late; scanty sputum</td>
</tr>
<tr>
<td>Infections</td>
<td>Common</td>
<td>Occasional</td>
</tr>
<tr>
<td>Respiratory insufficiency</td>
<td>Repeated</td>
<td>Terminal</td>
</tr>
<tr>
<td>Cor pulmonale</td>
<td>Common</td>
<td>Rare; terminal</td>
</tr>
<tr>
<td>Airway resistance</td>
<td>Increased</td>
<td>Normal or slightly increased</td>
</tr>
<tr>
<td>Elastic recoil</td>
<td>Normal</td>
<td>Low</td>
</tr>
<tr>
<td>Chest radiograph</td>
<td>Prominent vessels; large heart</td>
<td>Hyperinflation; small heart</td>
</tr>
<tr>
<td>Appearance</td>
<td>Blue bloater</td>
<td>Pink puffer</td>
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</tbody>
</table>
ASTHMA

- Chronic inflammatory disorder of airways
- Inflammation causes an increase in airway responsiveness (bronchospasm) to a variety of stimuli eg. exercise, cold, allergen, fever
ASTHMA

- Sign and symptom
  - reversible acute symptomatic attacks
to severe dyspnea, cough andwheezing triggered by suddenepisodes of bronchospasm
BRONCHIECTASIS

- Permanent, irreversible dilation of bronchial caused by destruction of muscle and elastic tissues of bronchial wall, resulting from chronic necrotizing infection
- Fever from infection, persistent cough
- Expectoration of copious amounts of foul smelling purulent sputum
- Dyspnea
- Occasional life-threatening hemoptysis

Congenital condition
- cystic fibrosis
- intralobar sequestration of lung
- primary ciliary dyskinesia
- kartagener syndrome

Post-infectious condition
- necrotizing pneumonia
  - bacteria: TB, S. aureus, Pseudomonas, H. influenzae
  - virus: adenovirus, influenza, HIV
  - fungi: Aspergillus spp.
- Bronchial obstruction from tumor, foreign body, mucus impaction
- Other: rheumatoid arthritis, SLE, inflammatory bowel disease, lung transplantation
Repeated or severe infection induces widespread damage to bronchial wall (supporting smooth muscle, elastic tissue) together with mucous obstruction

- further fibrosis
- permanent dilatation of bronchi
  (bronchiectasis)

Dilated airways sometimes up to 4 times normal size
BRONCHOGENIC CARCINOMA

- Arise form bronchial epithelium

WHO classification

- Squamous cell carcinoma (25-40%)
- Adenocarcinoma (25-40%)
- Small cell carcinoma (20-25%)
- Large cell carcinoma (10-15%)

BRONCHOGENIC CARCINOMA

- Cause:
  - Tobacco smoking
  - Industrial Hazards: asbestos and uranium exposure
  - Air pollution
  - Genetic
  - Scarring: previous TB, lung abscess
BRONCHOGENIC CARCINOMA

Squamous cell carcinoma

Adenocarcinoma

Small cell carcinoma

Large cell carcinoma
# Paraneoplastic Syndromes

- **Antidiuretic hormone (ADH):** hyponatremia
- **Adrenocorticotropic hormone (ACTH):** Cushing syndrome
  - small cell carcinoma
- **Parathyroid hormone-related peptide:** hypercalcemia
  - squamous cell carcinoma
- **Calcitonin:** hypocalcemia
- **Gonadotropins:** gynecomastia
DISEASES OF PLEURA

- Pleural effusion
- Pneumothorax
- Pleural tumor : mesothelioma

PLEURAL EFFUSION

- Accumulation of fluid in pleural cavity
- Pleural cavity filled with no more than 15 ml. of serous, relatively acellular, clear fluid lubricated the pleural surface
PLEURAL EFFUSION

- Increased accumulation of pleural fluid occurs in:
  - increased hydrostatic pressure eg. congestive heart failure
  - increased vascular permeability eg. Pneumonia
  - decreased osmotic pressure eg. nephrotic syndrome
  - increased intrapleural negative pressure eg. Atelectasis
  - decreased lymphatic drainage eg. mediastinal carcinomatosis

PLEURAL EFFUSION

- Clinical
  - dyspnea, pleural pain
- Physical examination
  - decreased movement of chest
  - dullness to percussion
  - decreased breath sound
  - decreased vocal resonance
PNEUMOTHORAX

- Air or gas in pleural cavities
- It causes compression and atelectasis of lung and may be marked respiratory distress
- Progressive increasing pressure may be sufficient to compress vital mediastinal structure and contralateral lung as “Tension pneumothorax”
PNEUMOTHORAX

- Classified as 3 types
  - spontaneous pneumothorax
  - traumatic pneumothorax
  - therapeutic pneumothorax

Pneumothorax  Tension pneumothorax
PNEUMOTHORAX

- Clinical features
  - sudden onset shortness of breath, dyspnea, chest pain, cyanosis
  - if severe, hypoxia and lead to coma
- Physical examination
  - decrease breath sound
  - hyperresonance to percussion