Respiratory system

LUNG ABSCESS

- localised area of necrosis of lung tissue with suppuration (discharge present)
- 2 types : 1° & 2° (develops as a complication of some other disease)

Pathogenesis

* Streptococci, staphylococci & various gram negative organisms isolated from lung abscess. They are introduced into lungs by :

→ Aspiration of infected foreign material

→ Preceding bacterial infection : As a complication of bronchopneumonia

→ Bronchial obstruction : An abscess may be form distal to the obstructed bronchus

→ Septic embolism

→ Miscellaneous :

- Infection in pulmonary infarcts
- Amoebic abscess
- Trauma to the lungs

PATHOLOGY

- M/c site : lower part of right upper lobe
  upper part of the right lower lobe

Gross : Size → few mm → large cavities, 5-6 cm in diameter
contains exudate
Poorly defined ragged wall - Acute

Develops fibrous wall - Chronic

M/C : Destruction of lung parenchyma with suppurative exudate in lung cavity

Initially - Cavity surrounded by acute inflammation

Then replaced by chronic inflammatory cell infiltrate (LC, PC, MP)

More chronic : fibrocollagenic wall

CF : fever, malaise, loss of wt, cough, hemoptysis

clubbing of fingers & toes

2° Amyloidosis may occur.

BRONCHIECTASIS

Abnormal & irreversible dilatation of bronchi & bronchioles in a focal or diffuse manner developing 2° to inflammatory weakening of bronchial walls.

Characteristic clinical manifestations : Persistent cough + expectoration (spitting out / coughing out) of copious amounts of foul smelling - purulent sputum.

Etiopathogenesis : due to endobronchial destruction & infections

- Hereditary & congenital factors :
  - ★ congenital bronchiectasis
  - ★ CF
  - ★ 1° immunodeficiencies
  - ★ Atopic bronchial asthma (rarely)

✓ AI & rheumatological causes :

- ★ RA
- ★ IBD
- ★ Sjogren syndrome

- Obstruction

  + Post obstructive bronchiectasis

    ↳ causes of endobronchial obstruction : foreign bodies, tumours, post-inflammatory scarring

- As a complication

PATHOLOGY

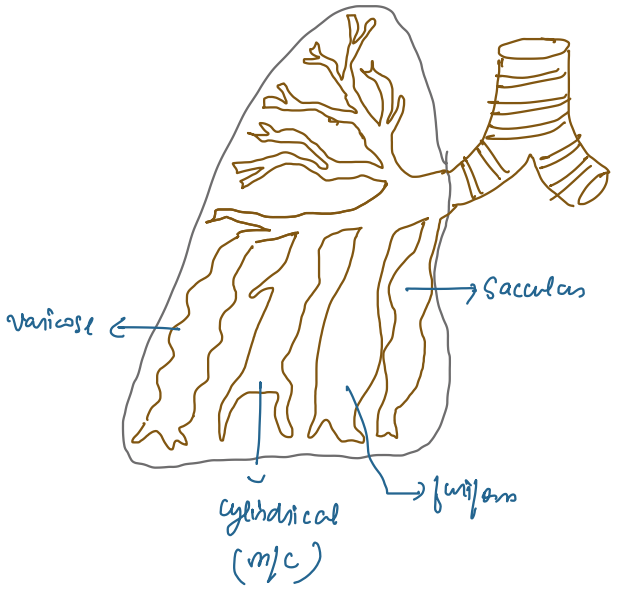
→ Affects distal bronchi & bronchioles

Gross : Bilateral involvement of lower lobes

vertical air passages of left lower lobe >> right

Dilated airways, extending upto pleural surface.

subcategorised into 4 diff types :



Cut surface of the affected lower lobes → honey combed appearance

→ Bronchi : Extensively dilated, walls are thickened, lumen filled with mucus / muco-pus.

Intervening lung parenchyma - reduced & fibrotic

m/c : Bronchial epithelium normal / ulcerated / squamous metaplasia

Bronchial wall : infiltration by acute / chronic inflammatory cells,

destruction of normal muscle, elastic tissue → replacement by fibrosis

Intervening parenchyma shows fibrosis

c/f : Chronic coughs + foul smelling sputum prodn + haemoptysis + recurrent pneumonia + sinusitis

COPD

→ Chronic bronchitis

Cor pulmonale

As the alveoli are lined by mucous → O_2 diffusion ↓

↓

↓ O_2 saturation : BLUE BLOATERS

Hypoxia :

Hypoxia

↓

All BV dilate to deliver more O_2 to the body - VD

↓

But pulmonary vessels constrict - pulmonary VC

↓

↑ pulmonary resistance

↓

↑ Pulmonary hypertension → right heart failure

↓

Cor pulmonale (mc seen in chronic bronchitis)

Emphysema

"Permanent dilatation of airspaces distal to the terminal bronchioles & the destruction of the walls of the dilated airspaces"

CLASSIFICATION

- Based on portion of the acinus involved
  - * Centriacinar
  - * Panacinar
  - * Paraseptal (distal acinar)
  - * Irregular
  - * Mixed

ETIOPATHOGENESIS

COPD → Emphysema
COPD → Chronic bronchitis

2 etiological factors : tobacco smoke & air pollutants (main)
Occupational exposure, infection, genetic influences (others)

Pathogenesis – destruction of the alveolar walls
↓
due to the deficiency of α-1 antitrypsin (α_1 Antiprotease)

α1-antitrypsin / α1-protease inhibitor inhibits proteases (mainly elastases which are derived from neutrophils). Elastase - digests lung parenchyma

- Mechanism of alveolar wall destruction : imbalance b/w proteases & antiproteases
  - ↑ed activity of elastase
  - ↓ed activity of anti-elastase

C/F : Severe exertional dyspnoea, use of accessory muscles, barrel shaped, cough occurs late, recurrent resp infections, weightloss, features of right heart failure

Types :-

1. Centriacinar emphysema

- Initial involvement of respiratory muscles, ie proximal part of the acinus

Gross - more severe in upper lobe of the lungs

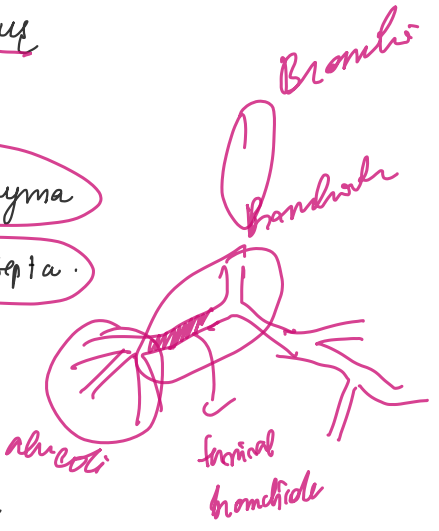
c/s - distended airspaces surrounded by rim of normal lung parenchyma

- lobules are separated from each other by fine fibrous tissue septa.

- Large amt of black pigment often seen. → Associated with Smoking
↳ tobacco

m/s - distension & destruction of resp bronchiole

- Terminal bronchioles shows chronic inflammation & are narrowed



2. Panacinar (Panlobular) emphysema

- All portion of acinus are effected

Gross : Involves lower zone of lung more frequently & more severly than upper zone.

- Confined to a few lobules, or may be more widespread

m/s : All portions of acini are distended

Associated with α1 AT deficiency

3. Paraseptal (distal acinar) emphysema

- Involves distal part of acinus
- Localised along the pleura & along perilobular septa
- Its mechanism is due to inflammatory destruction of alveolar walls
- Gross : subpleural portion of the lung shows air-filled cysts, 0.5 - 2 cm in diameter

Associated with inflammatory destruction of alveolar walls

4. Irregular emphysema

- Seen surrounding scars from any cause
- Smoke & coal dust in miners are major factors

Smoke/coal dust associated

5. Mixed emphysema

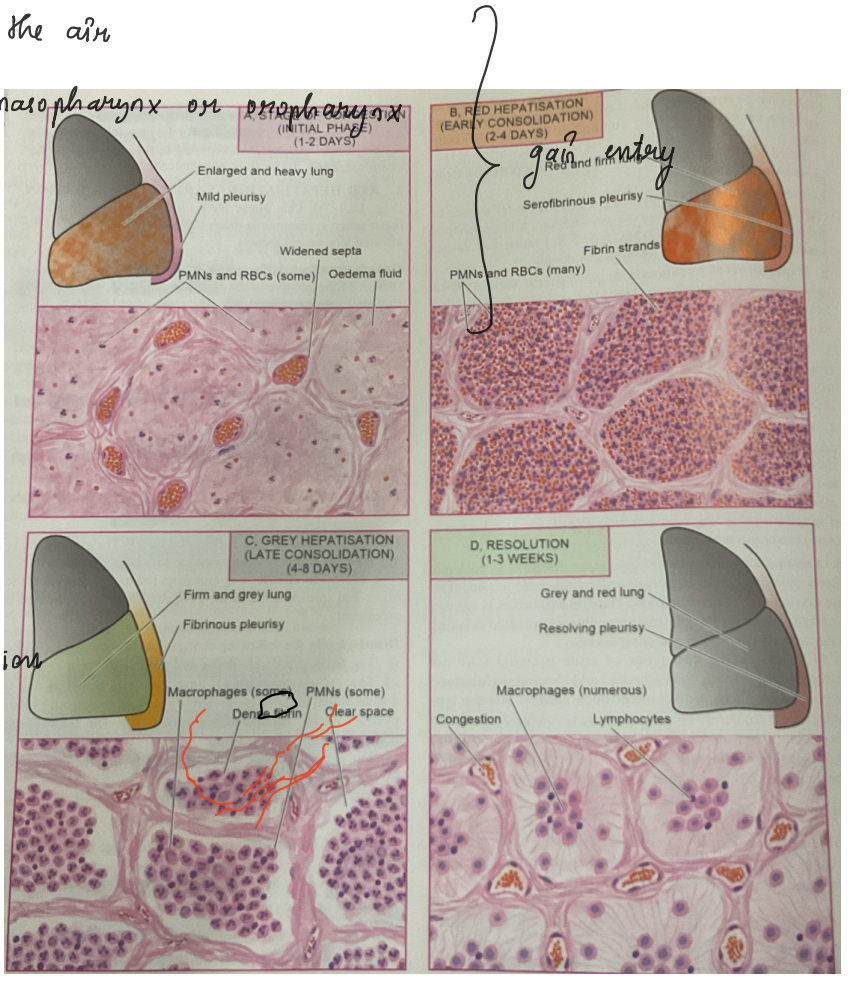
- Same lung may show more than one type of emphysema

Pneumonia

"Acute infection of the lung involving alveoli or interstitium, or both, by one or more copathogens"

PATHOGENESIS

- Inhalation of the microbes present in the air
- Aspiration of organisms from the nasopharynx or oropharynx
- Hematogenous spread
- Direct spread



gain entry

Predisposing factors:

* Altered consciousness (eg. alcoholism)
* Depressed cough & glottic reflexes
* Impaired alveolar macrophage function
* Endobronchial obstruction
* Immunocompromised states

Atypical bacterial pathogens include *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and *Legionella* species. Atypical organisms cannot be generally cultured or identified on Gram stain.

Based on etiologic agent, following common types of bacterial pneumonias are described:

1. **Pneumococcal pneumonia** Approximately 30-60% cases of CAP are caused by *Streptococcus pneumoniae*, a lancet-shaped diplococcus. Out of various types, type 3 *S. pneumoniae* causes particularly virulent form of bacterial pneumonia.

2. **Staphylococcal pneumonia** *Staphylococcus aureus* causes pneumonia by haematogenous spread of infection from another focus or after viral infections. It also includes strains of methicillin-resistant *Staphylococcus aureus* (MRSA).

3. ***Moraxella* pneumonia** *Moraxella catarrhalis* is one of the common organisms identified in bacterial pneumonia in children (etiologic agent in otitis media) and in the elderly (etiologic agent in COPD).

4. **Pneumonia by gram-negative aerobic bacteria** Other causes of bacterial pneumonia are gram-negative organisms like *Haemophilus influenzae*, *Klebsiella pneumoniae (Friedlander bacillus)*, *Pseudomonas aeruginosa* and *Moraxella catarrhalis*. *H. influenzae* commonly causes pneumonia in children below 3 years of age after a preceding viral infection. *Pseudomonas* is known to cause necrotising pneumonia.

5. ***Legionella* pneumonia** or Legionnaire disease is an epidemic illness caused by gram-negative bacilli, *Legionella pneumophila* that thrives in aquatic environment. The epidemic occurs in summer months by spread of organisms through contaminated drinking water or in air-conditioning cooling towers.

Morphologically, two types of acute bacterial CAP are distinguished—lobar pneumonia and broncho-(lobular-) pneumonia, each with distinct morphologic changes. Another type distinguished separately by some workers is *confluent pneumonia* that combines the features of both lobar and bronchopneumonia and involves larger (confluent) areas in both the lungs irregularly, while others consider this as a variant of bronchopneumonia.

LOBAR PNEUMONIA

Lobar pneumonia is an acute bacterial infection of a part of a lobe, the entire lobe, or even two lobes of one or both the lungs. The lower lobes are affected most commonly.

PATHOLOGY Laennec original description divided lobar pneumonia into 4 sequential pathologic phases: *stage of congestion* (initial phase), *red hepatisation* (early consolidation), *grey hepatisation* (late consolidation) and *resolution*. However, these classic stages seen in untreated cases are found much less often now-a-days due to early institution of antibiotic therapy and improved medical care. However, the sequence of pathologic changes described below represents the inflammatory response of lungs in bacterial infection.

1. STAGE OF CONGESTION: INITIAL PHASE (Fig. 18.7, A) The initial phase represents the early acute inflammatory response to bacterial infection that lasts for 1 to 2 days.

Grossly, the affected lobe is enlarged, heavy, dark red and congested. Cut surface exudes blood-stained frothy fluid.

Histologically, typical features of acute inflammatory response to the organisms are seen. These are as under:

i) Dilatation and congestion of the capillaries in the alveolar walls.
ii) Pale eosinophilic oedema fluid in the airspaces.
iii) A few red cells and neutrophils in the intra-alveolar fluid.
iv) Numerous bacteria demonstrated in the alveolar fluid by Gram's staining.

2. RED HEPATISATION: EARLY CONSOLIDATION (Fig. 18.7, B) This phase lasts for 2 to 4 days. The term hepatisation in pneumonia refers to liver-like consistency of the affected lobe on cut section.

Grossly, the affected lobe is red, firm and consolidated. The cut surface of the involved lobe is airless, red-pink, dry, granular and has liver-like consistency. The stage of red hepatisation is accompanied by serofibrinous pleurisy.

Histologically, the following features are observed (Fig. 18.8):

i) The oedema fluid of the preceding stage is replaced by strands of fibrin.
ii) There is marked cellular exudate of neutrophils and extravasation of red cells.
iii) Many neutrophils show ingested bacteria.
iv) The alveolar septa are less prominent than in the first stage due to cellular exudation.

3. GREY HEPATISATION: LATE CONSOLIDATION (Fig. 18.7, C) This phase lasts for 4 to 8 days.

Grossly, the affected lobe is firm and heavy. The cut surface is dry, granular and grey in appearance with liver-like consistency (Fig. 18.9). The change in colour from red to grey begins at the hilum and spreads towards the periphery. Fibrinous pleurisy is prominent.

Histologically, the following changes are present (Fig. 18.10):

i) The fibrin strands are dense and more numerous.
ii) The cellular exudate of neutrophils is reduced due to disintegration of many inflammatory cells as evidenced by their pyknotic nuclei. The red cells are also fewer. The macrophages begin to appear in the exudate.
iii) The cellular exudate is often separated from the septal walls by a thin clear space.
iv) The organisms are less numerous and appear as degenerated forms.

4. RESOLUTION (Fig. 18.7, D) This stage begins by 8th to 9th day if no chemotherapy is administered and is completed in 1 to 3 weeks. However, antibiotic therapy induces resolution on about 3rd day. Resolution proceeds in a progressive manner.

Grossly, the previously solid fibrinous constituent is liquefied by enzymatic action, eventually restoring the normal aeration in the affected lobe. The process of softening begins centrally and spreads to the periphery. The cut surface is grey-red or dirty brown and frothy, yellow, creamy fluid can be expressed on pressing. The pleural reaction may also show resolution but may undergo organisation leading to fibrous obliteration of pleural cavity.

Histologically, the following features are noted:

i) Macrophages are the predominant cells in the alveolar spaces, while neutrophils diminish in number. Many of the macrophages contain engulfed neutrophils and debris.
ii) Granular and fragmented strands of fibrin in the alveolar spaces are seen due to progressive enzymatic digestion.
iii) Alveolar capillaries are engorged.
iv) There is progressive removal of fluid content as well as cellular exudate from the airspaces, partly by expectoration but mainly by lymphatics, resulting in restoration of normal lung parenchyma with aeration.