PNEUMONIA

pneumonia

Definition - Alc inflammatn of lung parenchyma distal to the terminal bronchiole

Pathogenesis:- Entry of microorganism into lung ~~1)~~ Inhalatn of microbes
~~2)~~ Aspiratn of org. from nasoloropharynx.
~~3)~~ hematogenous spread from distant focus of infectn.
~~4)~~ Direct spread from adjoining site of infectn.

Predisposing conditions ~~1)~~ Altered consciousness ~~2)~~ depressed cough & glottic reflexes.
~~3)~~ Impaired mucociliary transport.
~~4)~~ Impaired alveolar macrophage functn.
~~5)~~ Endobronchial obstructn
~~6)~~ Immuno compromised states.

LOBAR PNEUMONIA.

Alc bacterial infectn of a part of lobe, entire lobe or even two lobes of one or both lung.

Etiology - 1) Pneumococcal pneumonia (90%)
2) Staphylococcal pneumonia
3) Streptococcal pneumonia - β hemolytic Streptococci
4) Pneumonia by GNB - hemophilus influenzae, Klebsiella pneumonia, Escherichia coli, proteus.

Morphological features :-

4 stages - 1) Stage of congestn
2) Red hepatisatn
3) Grey hepatisatn
4) Resolutn

* Stage of Congestn - (1-2)

Gross: Enlarged, heavy, dark red & congested.

C/S - blood stained frothy fluid.

Histo -
1) Dilatatn & congestn of capillaries in the alveolar walls.
2) Pale eosinophilic edema fluid in air spaces.
3) few red cells & neutrophils in the intra-alveolar fluid.
4) Numerous bacteria ⇒ alveolar fluid.

* Red hepatisatn - early consolidatn - (2-4)

Gross : Red, firm, consolidated
C/S - Airless, red-pink, dry granular has liver-like consistency.
⇒ Suppurative pleurisy.
Histo -
1) Edema fluid of preceding stage is replaced by strands of fibrin.
2) Cellular exudate of neutrophils & extravasatn of RBC cells.
3) Neutrophils show ingested bacteria
4) Alveolar septa less prominent due

* Grey hepatisation - (4-8 days)

Gross :- firm & heavy

C/s - dry, granular, grey in app. with liver like consistency.

Fibrinous pleurisy is prominent.

Histo -

(1) fibrin strands are dense & more numerous.

(2) cellular exudate of neutrophils & macrophages begin to appear

(3) Cellular exudate is often separated from septal wall by a thick clear space

(4) Org - less numerous and appear as degenerated form

* Resolution - (8-9th)

Gross - Solid fibrinous exu. ⇒ liquified

C/s - grey red or dirty brown frothy yellow creamy fluid

Histo -

1) Macrophages are the predominant cells.

2) Granular & fragmented strands of fibrin. ⇒ enzymatic digestn

3) Alveolar capillaries - engorged.

4) Progressive removal of fluid content as well as cellular exudate from air spaces by expectoration + lymphatics ⇒ Resolution of normal lung parenchyma

Complications :-

1.) Organisation (3%) ⇒ carnification

2.) Pleural effusion (5%)

3.) Empyema (< 1%)

4.) Lung abscess

5.) Metastatic pyaemia

COPD

COPD

Chronic, partial or complete obstruction to the airflow at any level from trachea to the smallest airways. resulting in functional disability of the lungs.

COPD ⟶ Bronchiolitis

- Chronic Bronchitis
- Emphysema
- Bronchial Asthma
- Bronchiectasis

Emphysema :-

emphysema

Permanent dilatation of air spaces distal to terminal bronchioles & the destruction of the walls of the air spaces.

Classification :-

1. Centriacinar
2. Panacinar
3. Paraseptal
4. Para-cicatrical
5. Mixed.

Pathogenesis :-

Etiologic factors :- tobacco smoke, air pollutants

Protease Antiprotease theory :-

- α1 antitrypsin ⇒ α1 protease inhibitor → inhibits protease secreted by neutrophils. ↓ digest the lung parenchyma
- PiMM (normal)
  PiZZ (deficient)
  Pinull null - null type.

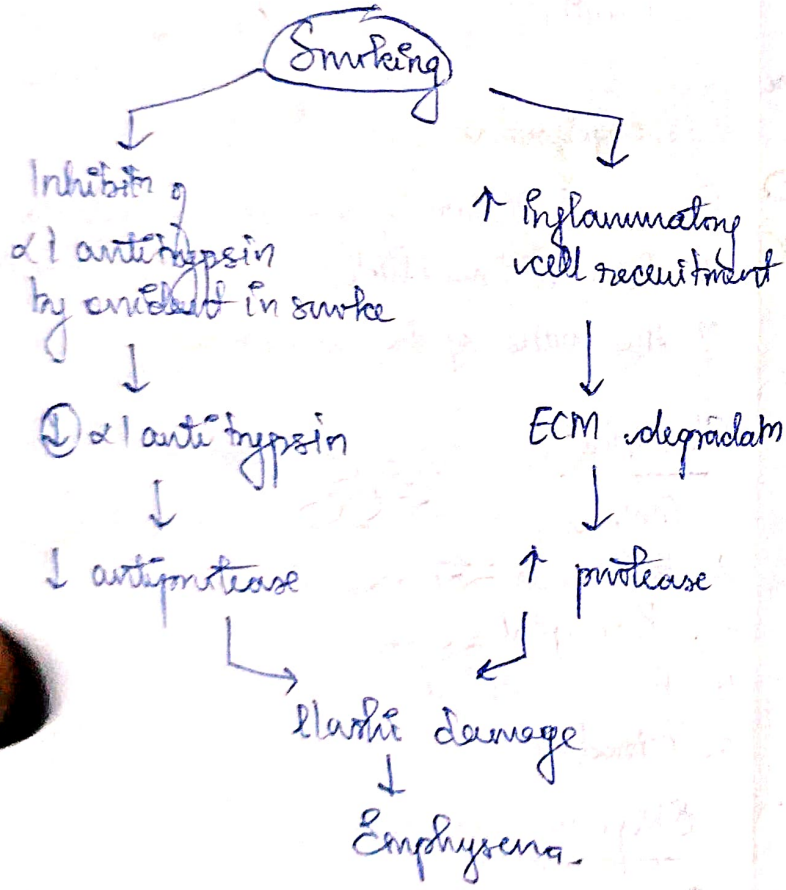
Imbalance b/w proteases (elastase) & anti-proteases (anti-elastase)

⇒ By ① anti-elastase activity ⇒ deg. of α1 antitrypsin.

⇒ By ② activity of elastase

⇒ Oxidant in cigarette ⊖ α1 antitrypsin.

⇒ Smokers have more phagocytes & neutrophils in their lungs than non-smokers.



Morpho :-

Gross :- Voluminous, pale with little blood. Edges - rounded

Mild cases - dilatation of air spaces.

Advanced cases - subpleural bullae & blebs.

↓ Rupture of alveoli

↓ rupture of adjacent air spaces.

Micro :- 1) lung show abnormally large alveoli separated by thin septa. Destruction of alveolar walls & loss of attachments of the alveoli. Pores of Kohn are enlarged.

2) Advanced - bullae

3) Inflammatory changes in small airways.

Bronchial Asthma

Chronic inflammatory disorder of the bronchial tree in which breathing is periodically rendered difficult by widespread narrowing of the bronchi. Clinically characterised by recurrent episodes of wheezing, breathlessness, tightness of chest & cough.

Ethopathogenesis & Types -

1) Atopy (extrinsic) - type I HSR

2) Non-atopic (intrinsic)

Morpho :-

Gross :- Overdistended & overinflated.

C/S - occlusion of bronchi & bronchioles by viscid mucus plugs.

Micro :-

1) Mucus plugs - normal or degenerated resp. epithelium forming twisted strips Curschmann's spirals.

2) Sputum - numerous eosinophils & diamond shaped crystals Charcot leyden crystals

3). Bronchial wall shows thickened BM, submucosal edema & inflammatory infiltrate.. lymphocyte & plasma cells.
Hypertrophy of submucosal gland

Bronchiectasis

Abnormal & irreversible dilatation of the bronchi & bronchioles developing 2° to inflammatory weakening of the bronchial wall

CM - persistant cough with expectoration of copious amount of foul smelling purulent sputum.

Pathogenesis:- → Endobronchial obstruction
→ Infection

① Hereditary & congenital factors:-
i) Congenital bronchiectasis - developmental defect of bronchial system.
ii) Cystic fibrosis.
iii) Hereditary immune dysfunction diseases.
iv) Immotile cilia syndrome - Kartagener syndrome.
v) Atopic BA.

② Obstruction - foreign bodies, endobronchial tumors, compression

③ 2° complication - necrotizing pneumonias

Morpho:-

Grossly:- B/L involvement of lower lobes
i) Cylindrical
ii) Fusiform
iii) saccular
iv) varicose