Histology - Pseudopalisading

Hypocellular - Antoni-A

Hypocellular - Antoni-B

Hypocellular

Verocay Bodies

Lungs

1) Bronchial asthma - Reversible bronchospasm

2) Bronchiectasis

3) Emphysema [pink puffers]

4] Chronic Bronchitis (blue bloaters)

COPD

Bronchial asthma -

Chronic inflammation → ↑ Responsiveness of Bronchial mucosa.

Reversible - Bronchoconstriction

Etiology: Chromosome - 5q associated with atopy & bronchial asthma.

→ most strongly m/c a/w — IL-13 gene polymorphism

NT-33 → Smooth muscle proliferation, fibroblast proliferation

↓

Airway Remodelling

a { ↑ Chitinase like glycoprotein → Severity Disorder.

↓

YLK-40

Serum ↑ ; Lung Tissue ↑.

Bronchial asthma.

Atopic

[m/c] - extrinsic

Pediatrics

H/o - allergy ⊕

H/o family ⊕

Serum. IgE ↑

Allergic asthma.

Allergic Bronchopulmonary aspergillosis

Occupational asthma.

Non atopic.

Intrinsic.

Adults.

No { allergy.
Family H/o.

Sr. IgE - (N)

→ Drugs

m/c Aspirin

→ cold

→ Viral infections

→ exercise induced

NB: Sputum ccc

1) Curschmann Spirals - mucus plugs - Tracheal shadow.

2) Charcot-Leyden Crystals ⟸ Eosinophilic membrane protein → Galactin-10

3) Creola Bodies → Individual epithelial cells.

Bronchiectasis:-

Bronchi/Bronchiole - dilatation irreversible

due to necrotizing inflammation.

Reversible: overinflation, -

Initiating event - infection, obstruction - anything

TB Tumor mass.

Congenital a) Cystic fibrosis

b) Primary ciliary Dyskinesia.

→ AD.

→ Dynein protein mutant (ciliary movement)

→ 50% Kartagener Syndrome

- Situs Inversus
- Sinusitis.
- Bronchiectasis.
- Infertility.

Complications -

Bronchiectasis

↓

Lung Abscess → Amyloidosis.

↙ ↓ ↓

Cerebral abscess. Renal abs. Suppurative arthritis.

3) Chronic Bronchitis -

Productive Cough
>3 months for >2 consecutive years. without any identifiable cause.

→ CB - strongly associated with - Smoking

→ Mucous gland - Hypertrophy } Reid index - 0.4
Bronchial epithelial thickness } C.B R.I >0.5

→ Metaplasia → dysplasia → Lung Ca.

4) Emphysema : Irreversible destruction of elastic tissue of acini

Trachea
↓
Bronchi.
↓
Bronchiole
↓
Terminal Bronchiole
↓
Respiratory Bronchiole } Acinus.
↓
Alveolar duct
↓
Alveoli.

Pathogenesis -

Imbalance

Protease ⟹ Anti-protease imbalance.

Macrophage, neutrophils } ↑ elastase.

Lung: α1-antitrypsin def.

Centri-acinar

m/c Type { Clinical, Histological, Overall.

→ Cigarette.

→ Coal Workers pneumoconiosis

↓

macrophages

↓

Elastase (mmp)

↓

α_1 AT digestion.

- alveolar duct, alveolar sac

Pan-acinar

Congenital deficiency of α_1-antitrypsin

↓

Neutrophil

↓

elastase

↓

Pan-acinar

→ entire acinus is affected.

Acute Interstitial pneumonia (ARDS with unknown etiology)

Histologically Similar to ARDS

Diffuse alveolar damage

→ Hyaline membrane disease.

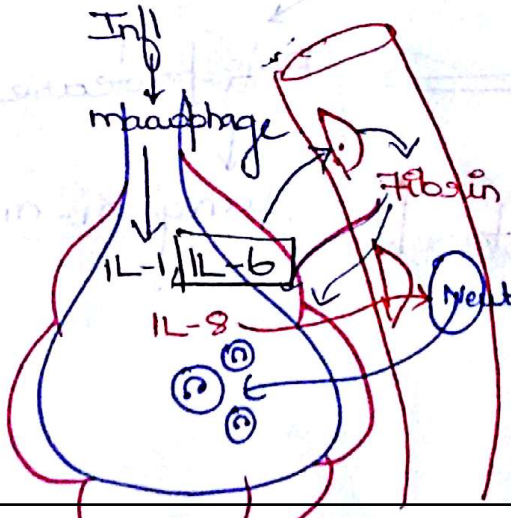
→ Shock lung.

Hyaline membrane

FIBRIN +

Type-I pneumocytes

Pathogenesis -



Thrombus + endothelial damage.

Even with Supply of 100% O_2 → No gaseous exchange due to Hyaline membrane

↓

No improvement → ∴ Shock lung.

m/c Presentation : Hypoxia > Diffuse alveolar damage.

Pneumonia

Inflammation + infection of lung parenchyma.

Typical

Fever - ↑

CBC - ↑TLC.

CXR - Consolidation.

Histology - Congestion + Consolidation

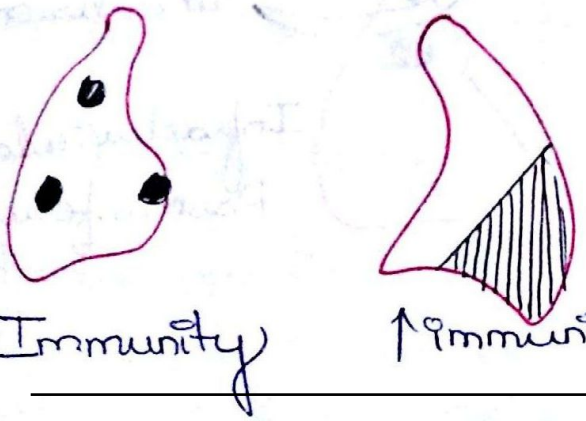
↓

d/t alveolar exudates.

m/c Caused -

Streptococcus pneumoniae

Broncho — Lobar



↓Immunity ↑Immunity.

Atypical.

mild/absent.

Walking pneumonia.

Histology - Congestion without Consolidation

No alveolar exudates

Interstitial mononuclear inflammatory cells.

m/c associated - Viral infection

m/c a/w — mycoplasma infection

→ Giant cells.

Total - 10 days

Laennec Stages — Lobar pneumonia

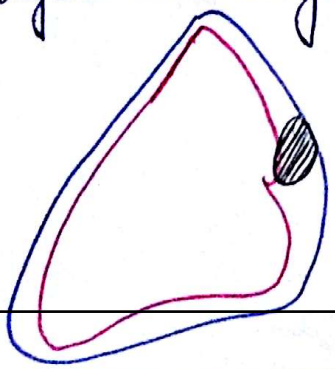
1) Stage of Congestion - Bacterial ↑↑ — 1-2 days
2) Red Hepatization - RBC + WBC + ↑ Bact. — 3-4 days
3) Gisray Hepatization - Fibarineus exudate. — 5-7 days
4) Stage of Resolution (m/c outcome) — 8-10 days

Tuberculosis

Virulence Factor - Cord Factor.

Types

1° — Pediatrics
exogenous
LN enlargement (m/c)
U/L - Hilary lymphnodes
pleural effusion
Consolidation
erythema nodosum.
Phlyctenular Conjunctivitis.



2° — Reactivation TB
Adults / elderly
endogenous
Rarely
Cavitation.
- pulmonary fibrosis
Supraclavicular
Pulls lesion
Infraclavicular
Assmann focus

Simon's focus + Lymphatic Lymphnode - Ghon's Complex

Ranke's Complex : Calcification + fibrosis of ghon's complex

Other 2° TB

Simon's focus - TB nodule elsewhere in the body
(Both 2° > 1°).

Miliary Tuberculosis : (Hematogenous Spread)

1) Liver → Simmonds focus.
2) Brain Cortex → Rich focus.
3) Pulmonary Vein - (Sub-intimal) - Weigert's focus.

Congenital TB-

m/c site - 1° complex of Liver.
m/c overall.

Pneumoconiosis

→ Dangerous particle size - 1-5 µm (∵ macrophages cannot phagocytose < 5µm)

1) Coal Worker Pneumaconiosis
2) Silicosis - m/c occupational.
3) Asbestosis.

Ferruginous Bodies : Iron + glycoprotein + organic + Inorganic dust duct fibres

Asbestosis > Silicosis, CWP

Lobar pneumonia

Total - 10 days

Stages

1) Stage of Congestion - Bacterial ↑↑ — 1-2 days
2) Red Hepatization - RBC + WBC + ↑ Bact. — 3-4 days
3) Gray Hepatization - Fibrinous exudate. — 5-7 days
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Virulence Factor - Cord Factor.

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1° | 2° Reactivation TB

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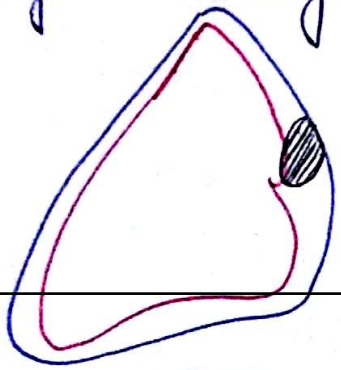
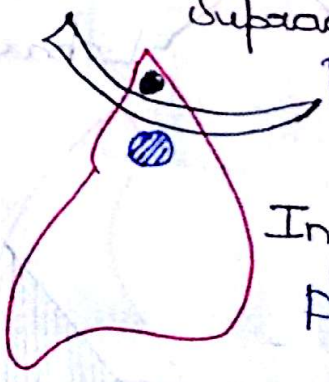


Ghon's focus.

Adults / elderly
endogenous
Rarely
Cavitation.
- pulmonary fibrosis



Subaadavicular
Puhl's lesion
Infraclavicular
Assman focus

Ghon's focus + Lymphatic Lymphnode - Ghon's Complex.

Rathke's Complex: Calcification + fibrosis of ghons complex.

Other 2° TB

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→ Dangerous particle Size - 1-5 µm (∵ macrophages cannot phagocytose < 5µm)

1) Coal Worker Pneumaconiosis
2) Silicosis - m/c occupational.
3) Asbestosis.

Ferruginous Bodies: Iron + glycoprotein + organic + Inorganic dust dust fibres.

Asbestosis > Silicosis

Asbestos

Straight — Serpentine

Crocidolite
Amphasite } most potent.

Chrysotile.

Pathogenic.

→ mesothelioma.

→ Asbestos ⟶ Risk same even after leaving disease (for all pneumaconiosis)

→ 5-10 years.

Diffuse Calcific pleural plaque - Characteristic

Asbestos Carcinogenesis.

mc a/w — Bronchogenic Ca. — 15-20 years

most specific — Mesothelioma. — 25-45 years

Adeno Ca > Squamous Ca.

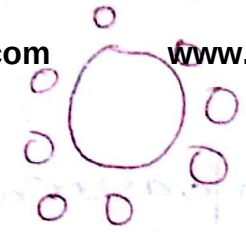
Adeno Ca of lung is a/w with Scarring of lung tiss

Mesothelioma.

Adenoca

Lumen surrounded by Tumor cells.

Papillary glandular pattern



IHC Thyroid Transcription Factor-1

Napsin-A.

Cytokeratin 5/6.

Calretinin - Best

Electron microscopy [EM]

Small, nonbranching, microvilli

Large Branching microvilli

Lung Cancer

m/c lung tumor - Mets

m/c Site of metastasis - Lung

Mets

Pediatric

Mets - mc osteosarcoma > WT.

Adults

- Breast Ca.

Malignancy - Carcinoid.

Adenoca (m/c female, non-smokers)

Benign - Inflammatory myofibroblastic tumor.

Non-smokers > Smokers.

Hamartoma

Squamous Cell Ca

→ Central.

→ Smoking m/c associated with Sq. cell. Ca

Smoking is Strongly a/w → Small cell Ca

→ m/c - a/w - Hypercalcemia,
Cavitation.

Histology - Epithelial/Keratin pearls.

Concentric eosinophic Rings.
- well differentiated Sq. cell. Ca

m/c a/w - Pancoast Syndrome → Sq. cell Ca.

Superior Vena Cava Syndrome → Small cell Ca.

II] Adenocarcinoma:

→ Slow growing

→ peripheral in location

→ poorer prognosis (due to early metastasis)

Lung → another Lung metasis [aerogenous spread]

Types) 1) Non-mucinous

2) Mucinous. (m/c associated with aerogenous spread)

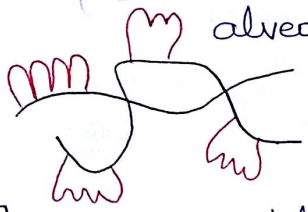
→ Bronchioalveolar adenoCa:

Clara cells (m/c) > Type II pneumocytes

↓

Terminal Bronchiole

→ Best prognosis – Nometastasis. (∵ they grow along preexi alveolar septa)



* Lepidic pattern

(Butterfly overlence)

* Lepidic cells – Myxoma – stellate mesenchymal cells.

III) Small cell Ca – Worst prognosis

NET – worse prognosis.

→ Neurosecretory granules.

Azzopaudi effects – Basophilic staining of BV walls.

m/c Paraneoplastic in small cell Ca:

SIADH > Cushing.

m/c – Cause of ectopic ACTH production – Carcinoid.

Lung Ca – m/c site of mets – Adrenal 75%.