Viral & mycoplasma pneumonias (Prim. atypical pneumonia)

Patchy inflammatory changes, largely confined to interstitial tissue of the lungs, without any alveolar exudate.

Occur in all ages

Most cases mild & transient

Rarely severe & fulminant

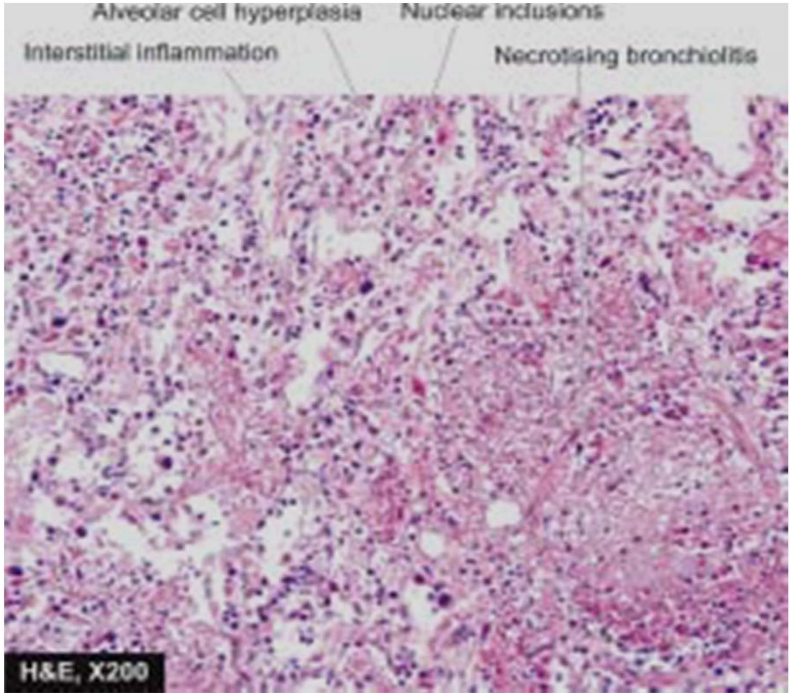
Aetiology

- Caused by variety of agents
- Most commonly by Respiratory syncytial virus (RSY)
- Others are *Mycoplasma pneumoniae,* influenza and parainfluenza viruses, adenoviruses, rhinoviruses, coxsackieviruses, cytomegaloviruses(CMV).
- Occasionally, psittacosis *(Chlamydia),* Q fever *(Coxiella)*
- *Infection of respiratory tract with these agents common*
- *Remains confined to URT, in the form of common cold*
- *May extend down into interstitium in cases of* malnutrition, chronic debilitating diseases and alcoholism

Morphology

- **G/A:** Lungs heavy congested and subcrepitant. C/S exudes frothy or bloody fluid. Pleural reaction mild.
- **M/E:**
- **i) Interstitial inflammation:** Thickening of alveolar walls due to congestion, oedema and mononuclear inflammatory infiltrate by lymphocytes, macrophages and some plasma cells.
- **ii) Necrotising bronchiolitis:** Foci of necrosis of the bronchiolar epithelium, inspissated secretions in the lumina and mononuclear infiltrate in the walls and lumina.

- **iii) Reactive changes:** Lining epithelial cells of bronchioles and alveoli proliferate may form multinucleate giant cells and syncytia in the bronchiolar and alveolar walls. Occasionally, viral inclusions (intranuclear and/or intracytoplasmic) found, in pneumonitis caused by CMV.
- **iv) Alveolar changes:** In severe cases, the alveolar lumina contain oedema fluid, fibrin, scanty inflammatory exudate and coating of alveolar walls by pink, hyaline membrane similar to respiratory distress syndrome. Alveolar changes are prominent when bacterial infection supervenes.


Alveolar cell hyperplasia
Nuclear inclusions
Interstitial inflammation
Necrotising bronchiolitis
H&E, X200

Complications

- Superimposed bacterial infections and its complications
- Most cases recover completely.
- Late interstirial fibrosis and permanent damage

Pneumocystis carinii Pneumonia

- A protozoon widespread in environment, causes pneumonia by inhalation of organisms as an opportunistic infection in neonates and immunosuppressed people
- Almost 100% cases of HIV/AIDS develop opportunistic infection during the course of disease, most commonly *Pneumocystis carinii pneumonia*
- Other immunosuppressed groups are patients on chemotherapy for organ transplant and tumours, malnutrition, and agammaglobulinaemia

Morphology

G/A:

- Lungs, dry, consolidated, grey.

M/E:

- i) Interstitial pneumonitis with thickening and mononuclear infiltration of the alveolar walls.
- ii) Alveolar lumina contain pink frothy fluid containing the organisms.
- iii) By Gomori's methenamine-silver (GMS) stain, characteristic oval or crescentic cysts, 5 μm in diameter surrounded by numerous tiny black dotlike trophozoites of *P. carinii demonstrable in the frothy* fluid.
- iv) No significant inflammatory exudate is seen in the air spaces.

Aspiration pneumonia

- Results from inhalation of various agents into the lungs.
- Food,
- Gastric contents,
- Foreign body and
- Infected material from oral cavity

Factors predispose to inhalation pneumonia:

- unconsciousness, drunkenness, neurological disorders affecting swallowing, drowning, necrotic oropharyngeal tumours, premature infants and congenital tracheo-oesophageal fistula

Morphology

- Right lung affected more commonly.
- **Sterile** foreign matter as acidic gastric contents produce *chemical pneumonitis.*
- There is haemorrhagic pulmonary oedema with presence of particles in the bronchioles.
- **Non-sterile** aspirate cause widespread *bronchopneumonia* with multiple areas of necrosis and suppuration. A granulomatous reaction with foreign body giant cells may surround the aspirated vegetable matter

Lung abscess

- Localised area of necrosis of lung tissue with suppuration.
- ***Primary*** *lung abscess develops in otherwise normal* lung. The commonest cause is aspiration of infected material.
- ***Secondary*** *lung abscess develops as a complication of* some other disease of the lung or from another site.

Aetiopathogenesis

- Microorganisms commonly isolated from the lungs are streptococci, staphylococci and various gram-negative organisms.
- **Mode of entry in lungs:**
- Aspiration of infected foreign material
- Preceding bacterial infection
- Bronchial obstruction
- Septic embolism
- **Miscellaneous:** i) Infection in pulmonary infarcts.
- ii) Amoebic abscesses due to infection with *Entamoeba histolytica.*
- iii) Trauma to the lungs.
- iv) Direct extension from a suppurative focus in the mediastinum, oesophagus, subphrenic area or spine.

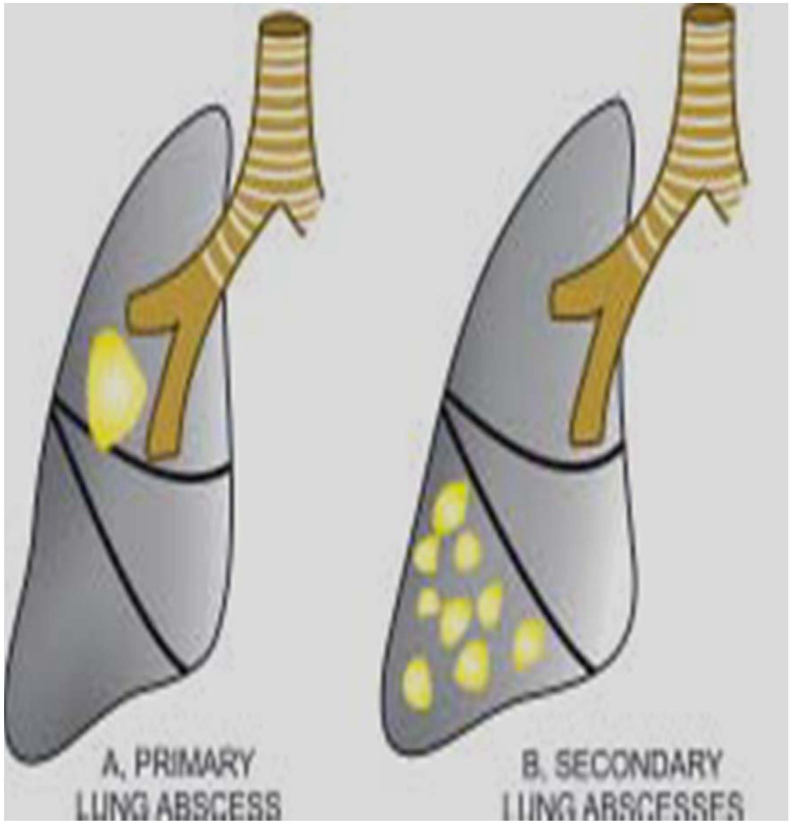
Morphology

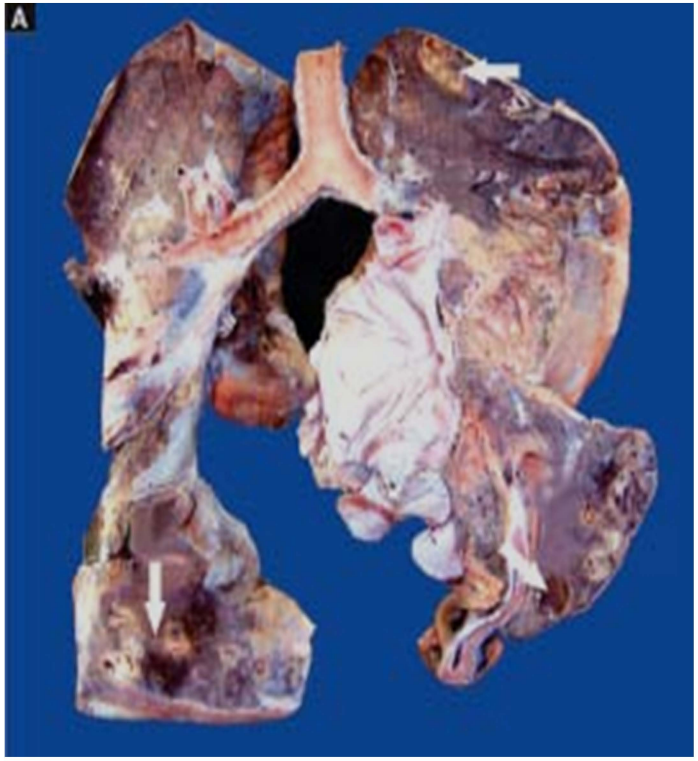
- **G/A:**
- Variable size few mm to 5-6cms
- Cavity has exudate
- Acute abscess has poor ragged wall
- Chronic develops thick fibrous wall.
- **M/E:**
- Destruction of lung parenchyma with suppurative exudate in lung cavity.
- Cavity initially surrounded by acute inflammation in the wall, later by chronic inflammatory cell infiltrate composed of lymphocytes, plasma cells and macrophages

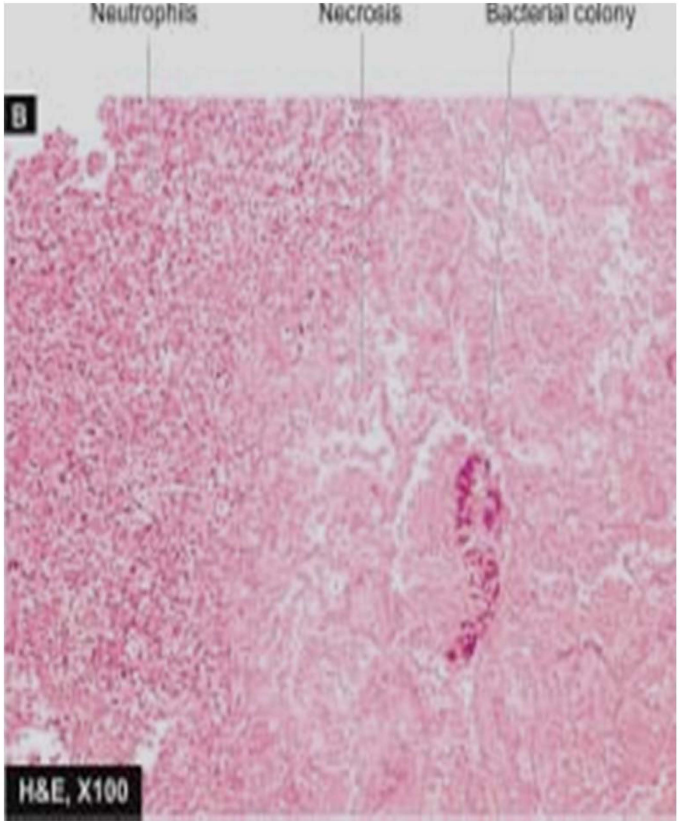
- In more chronic cases, considerable fibroblastic proliferation forming a fibrocollagenic wall.

Clinical features:

- fever, malaise, loss of weight, cough, purulent expectoration, haemoptysis in half the cases. Clubbing of the fingers, Secondary amyloidosis in chronic long-standing cases.


A, PRIMARY
LUNG ABSCESS
B, SECONDARY
LUNG ABSCESSES


A


Neutrophils
Necrosis
Bacterial colony
B
H&E, X100