Pulmonary Infections

By

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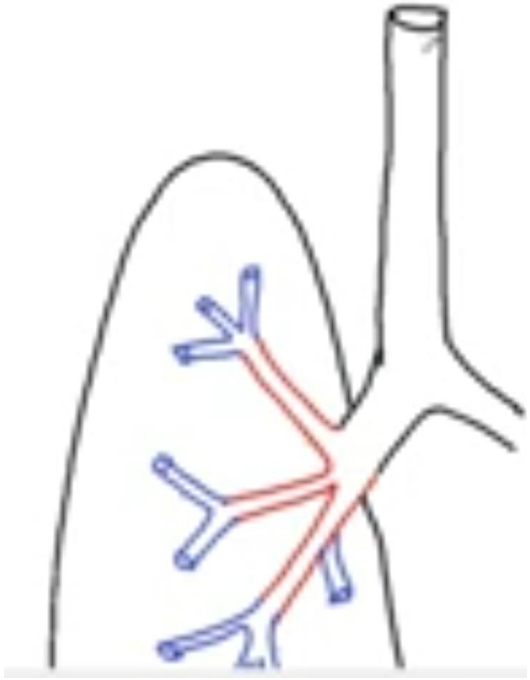
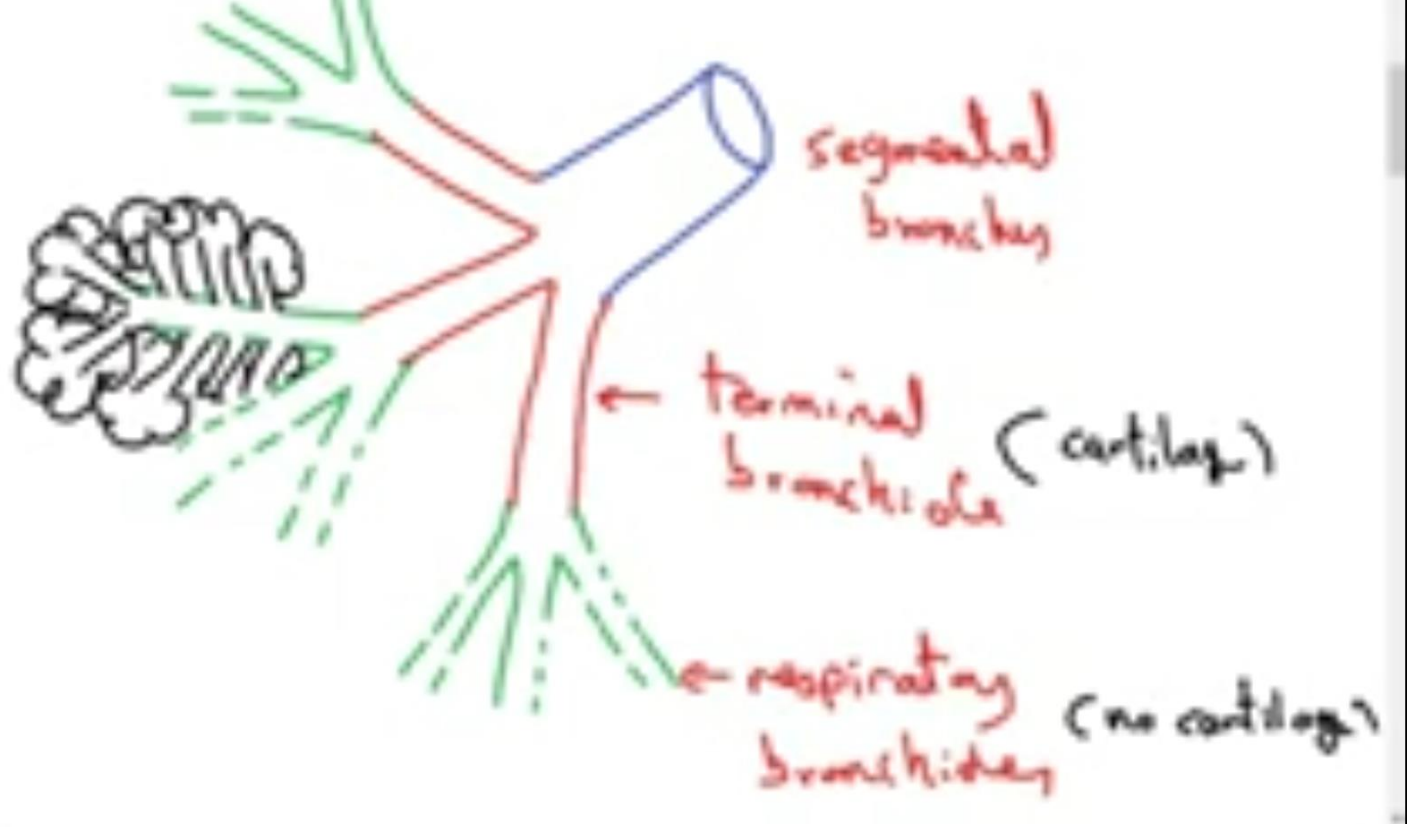
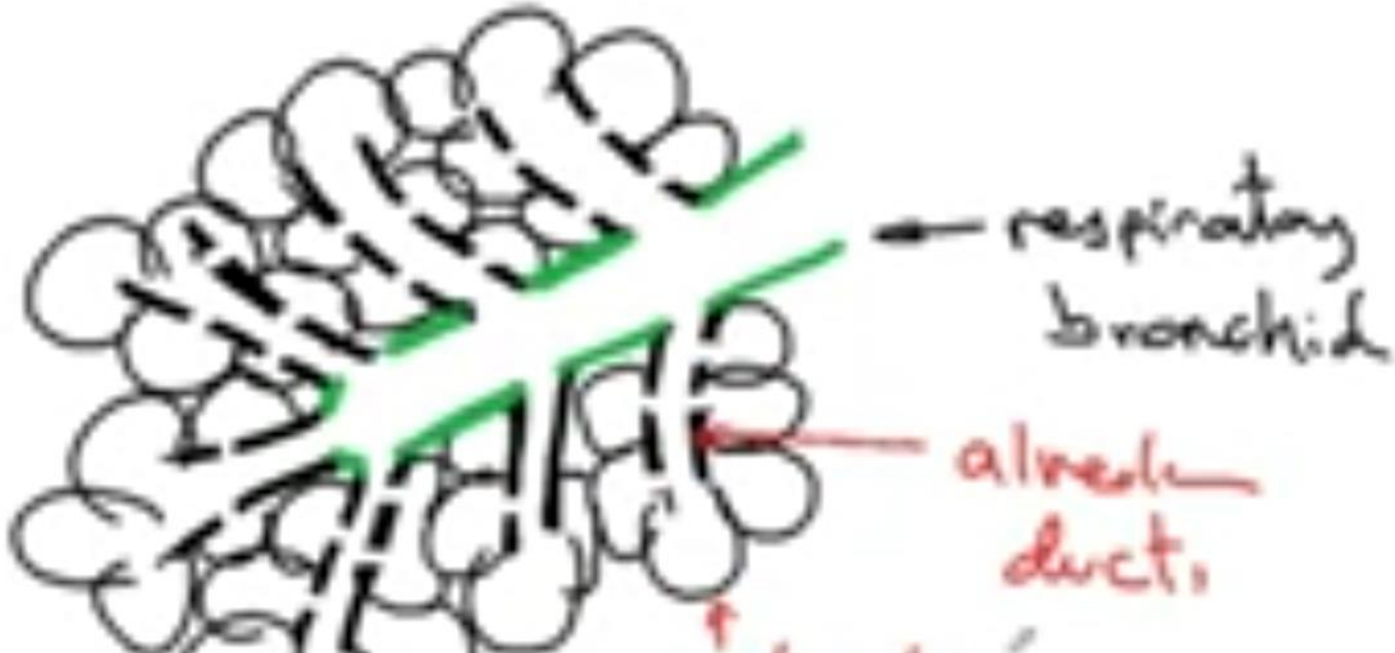
ILOs

- **Define pneumonia.**
- **Classify pneumonia, outline the anatomical and etiological classification of pneumonia.**
- **Describe the gross and microscopic picture of lobar pneumonia and mention its complication.**
- **Describe the gross and microscopic picture of bronchopneumoniaand mention its complication.**
- **Describe the gross and microscopic picture of interstitial pneumoniaand mention its complication.**




segmental bronchus
terminal bronchiole
(cartilage)
respiratory bronchiole
(no cartilage)


respiratory bronchiole
alveolar duct

Pneumonia

- Definition:
- Patchy or diffuse inflammation of the lung with consolidation.
- Predisposing factors:
- 1.Altered consciousness, Coma states
- 2.Following surgery, general anesthesia, tracheostomy, endotracheal or nasogastric tube.
- 3.Retention of secretions
- 4.Impairment of normal lung defense mechanisms.
- 5.Congestion & edema
- 6.Bronchial obstruction or dilatation.

Classifications of pneumonia

alveoli → lobar pneumonia

alveoli + bronchi → bronchopneumonia

interstitial → interstitial pneumonia

Classifications

- A. Anatomically:
- **Lobar pneumonia**.
- **Bronchopneumonia** (or Lobular pneumonia).
- **Interstitial pneumonia**.
- B.Etiologically:
- **I.BacterialPneumonia:**
- a.Lobarpneumonia.
- b.Bronchopneumonia(Lobularpneumonia).
- c.Tuberculousbronchopneumonia.
- **II.Viral andMycoplasmaPneumonia**
- **III.Othertypes:**
- a.Aspiration(Inhalation)pneumonia.

Types of Pneumonia

Inflammation Upper Lobe of the Lungs

Inflammation Middle Lobe of the Lungs

Inflammation Lower Lobe of the Lungs

Lobar Pneumonia

- Definition:
- It is an acute diffuse fibrinous inflammation affecting one or more lobes.
- Etiology (causative organism):
- Pneumococci.
- Occasionally other organisms as Staphylococci,Streptococci,
- Predisposing Factors:
- Lowering of the body resistance.
- More common in young adults and middle-aged persons.

Pathology of lobar pneumonia

- Upper lobes are commonly affected.
- It is divided into 4sequential pathologic phases:
- 1.**Congestion**(1stdayofillness)
- 2.**Redhepatization**(earlyconsolidation,2nd–4thday)
- 3.**Grayhepatization**(lateconsolidation,5th–8thday)
- 4.**Resolution**(9th–21stday)

1.Congestion(1st day of illness):

- **Definition**:

It represents the early acute inflammatory response to bacterial infection

Gross Picture:

The affected lobe is dark red, enlarged, heavy & congested.

On cut surface ,exudation of frothy fluid.

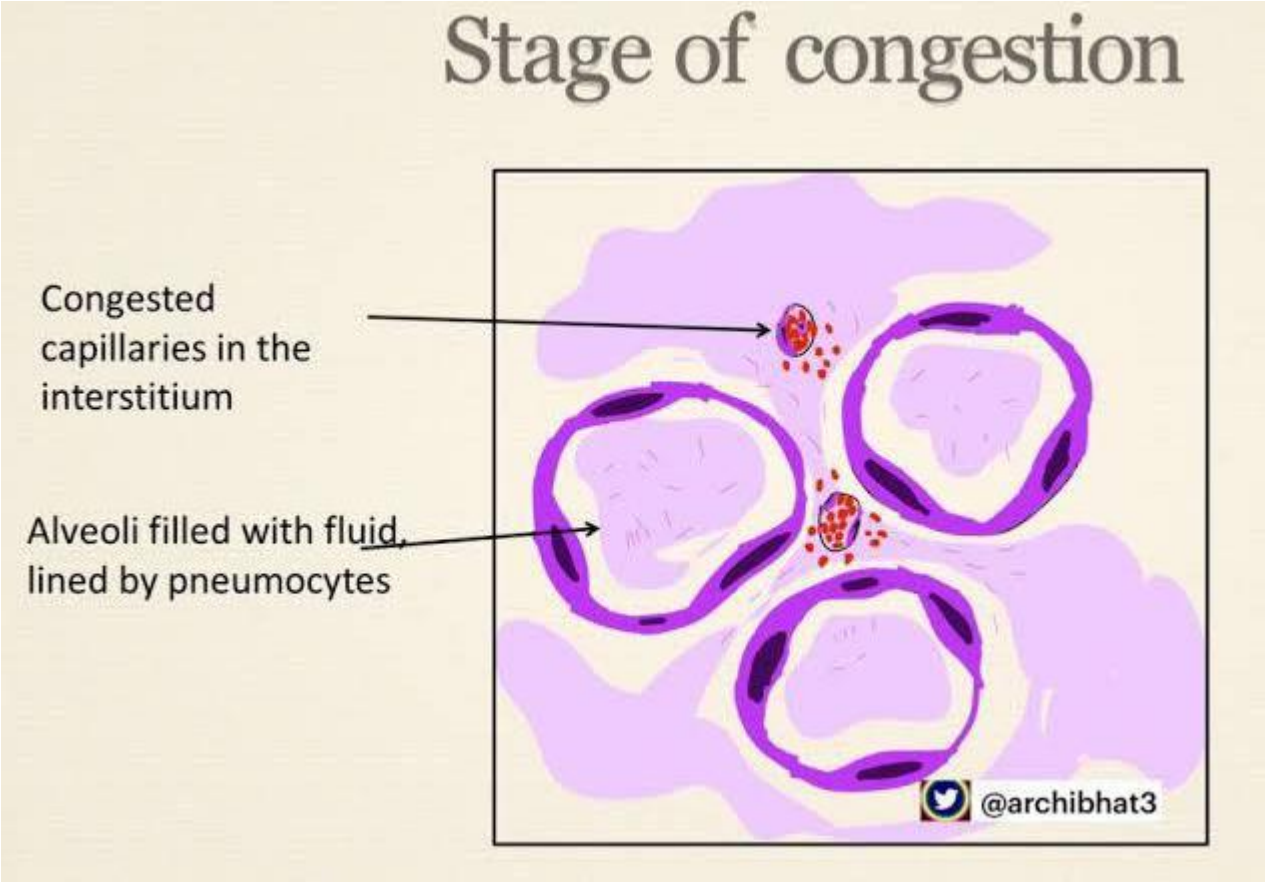
Consistency is wet sponge.

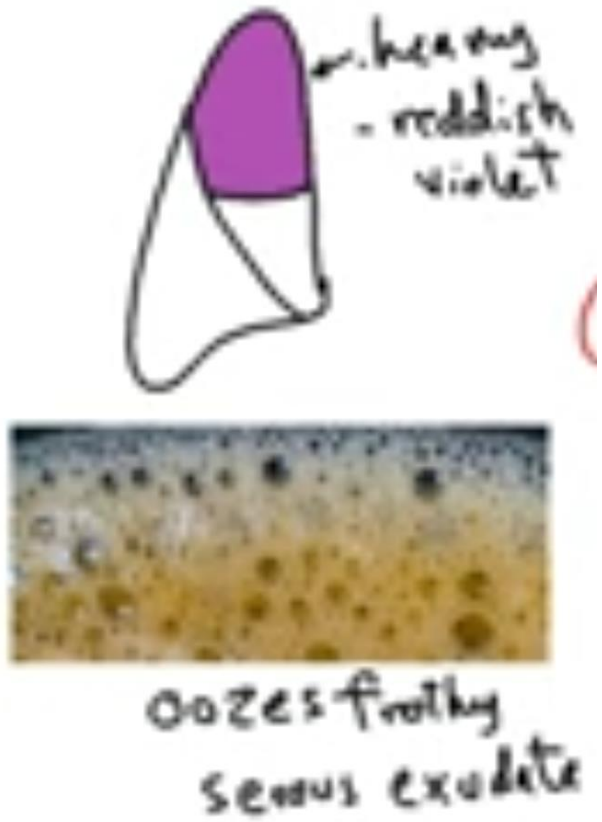
Microscopic Picture:

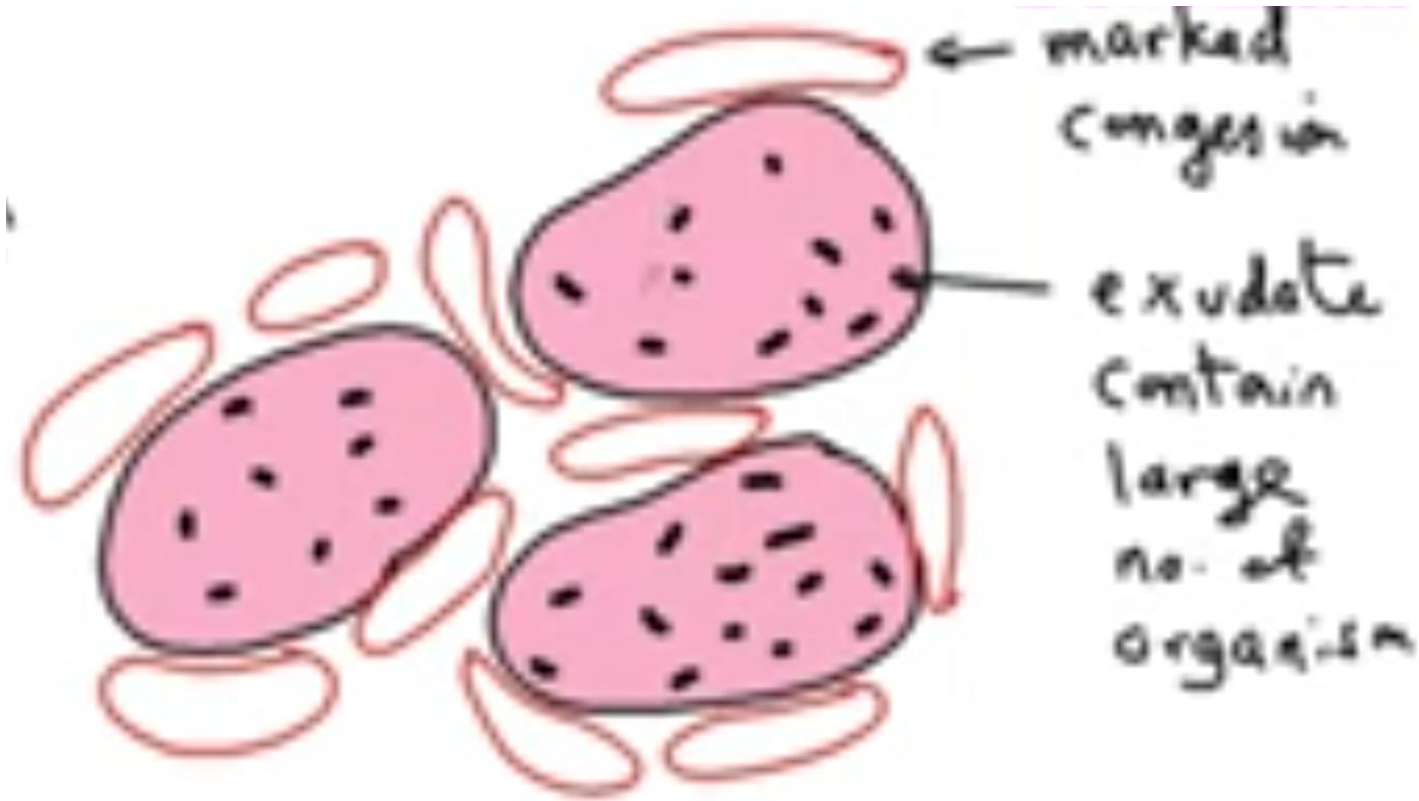
The alveolar capillaries are **dilated** and congested.

Alveolar wall is **thickened**

The alveolar spaces contain **serous exudate** ,large number of **pneumococci** and few numbers of polymorphs

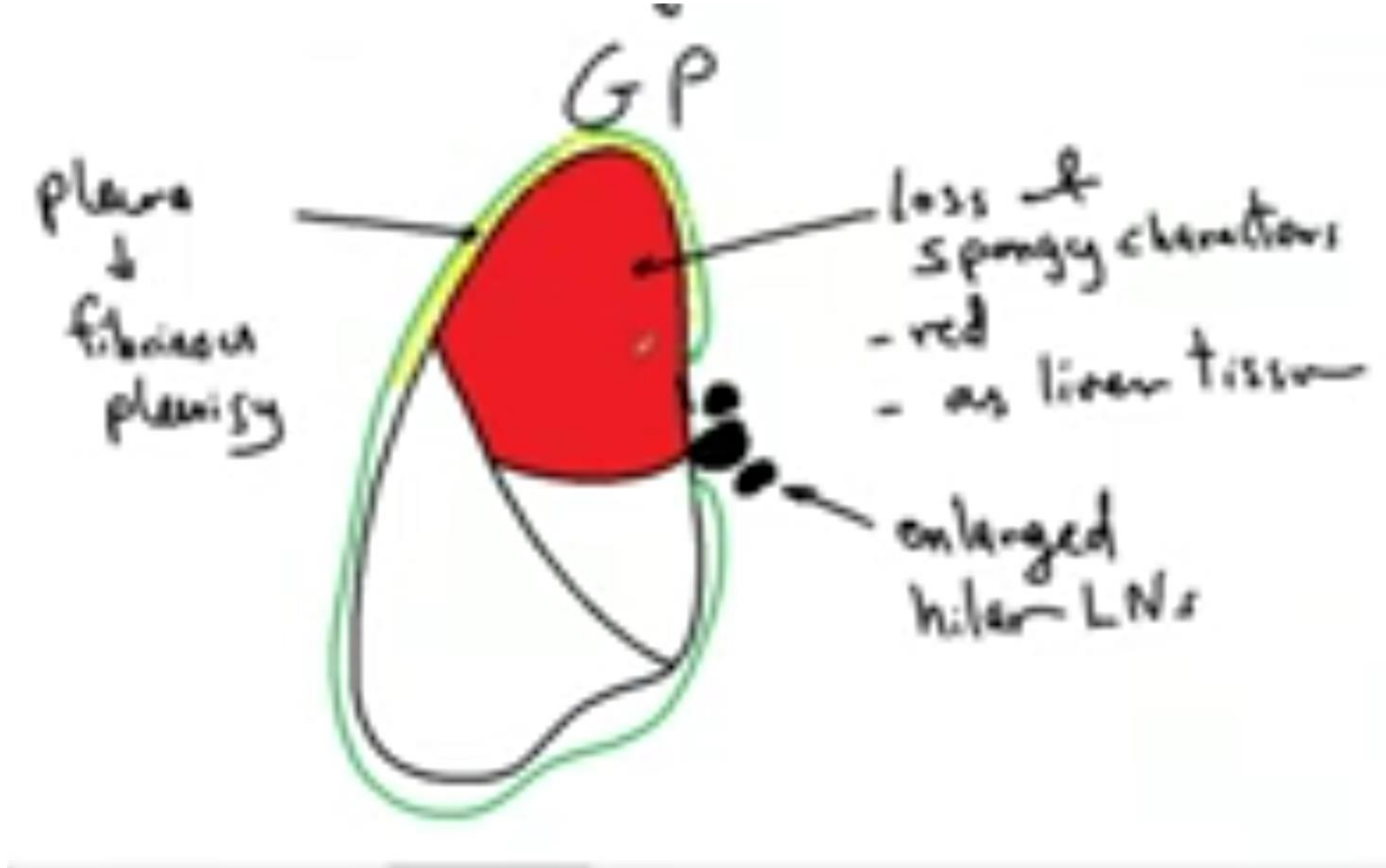



- heavy
- reddish
violet
oozes frothy
serous exudate


marked
congestion
exudate
contain
large
no. of
organism

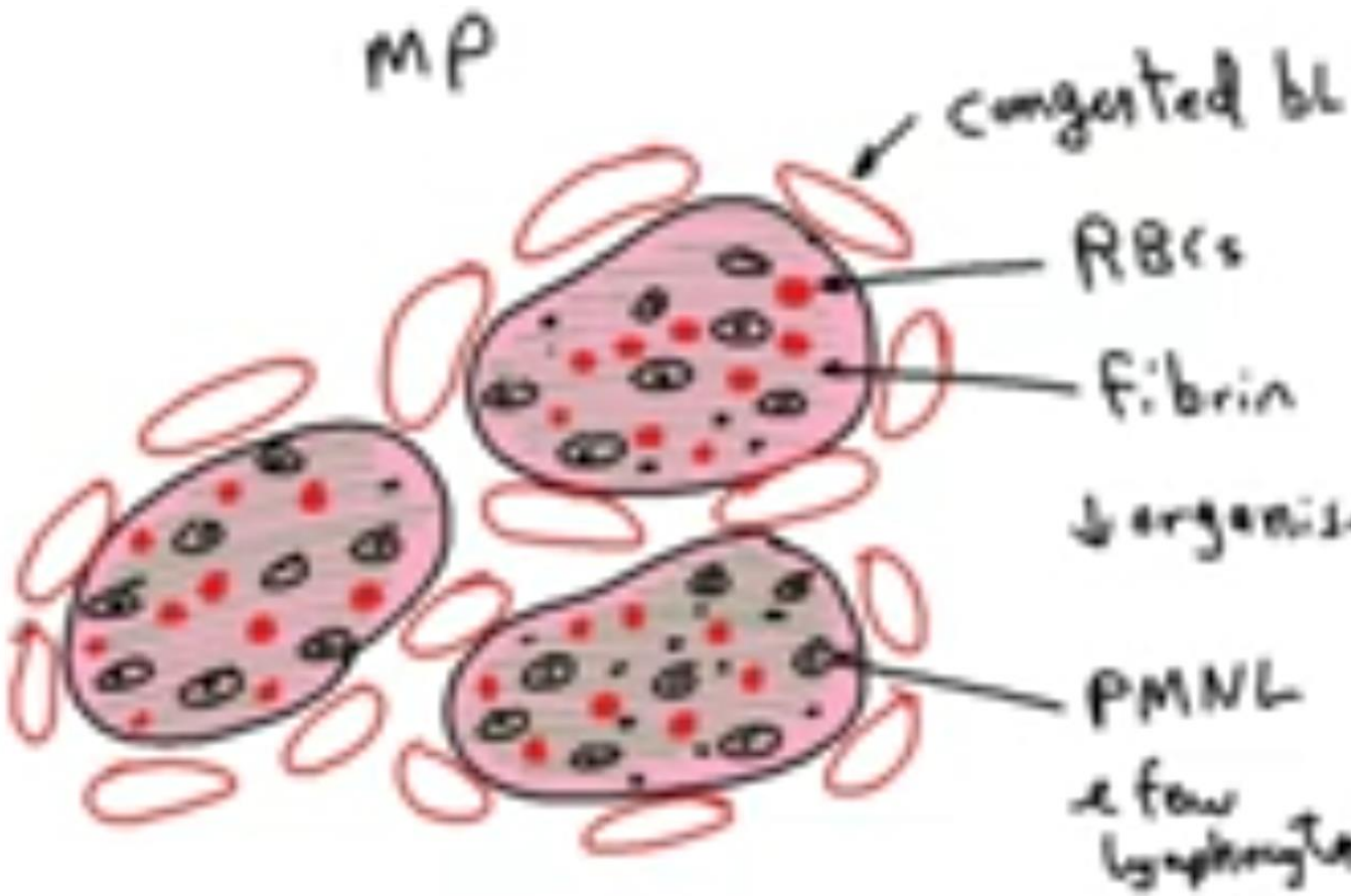
Red Hepatization (Early consolidation, 2nd – 4th day):

- **Definitio**n: The term hepatization in pneumonia refers to liver-like consistency of the affected lobe on cut section.
- Gross Picture:
- ➢ The affected lobe is red, enlarged, firm & consolidated.
- ➢ The cut surface of the involved lobe is airless, red-pink, dry, granular and has liver-like consistency.
- ➢ The stage of red hepatization is accompanied by sero-fibrinous pleurisy.
- ➢ The hilar lymph nodes are enlarged due to acute lymphadenitis.


GP
pleura
↓
fibrinous
pleurisy
loss of
spongy characteristics
- red
- as liver tissue
enlarged
hilar LNs

Microscopic Picture:

- ➢ The alveolar capillaries **are dilated** and congested.
- ➢ The alveolar walls are t**hickened**.
- ➢ The alveolar spaces contain
- excess fibrin in the form of
- a network entangling large
- number of red cells and
- few numbers of polymorphs,
- macrophages and pneumococci.



Gray hepatization (late consolidation, 5th – 8th day):

Gross Picture:

- The affected lobe is enlarged, firm and heavy.
- The cut surface is dry, granular and gray in appearance with liver-like consistency.
- Fibrinous pleurisy is prominent.
- The hilar lymph nodes are enlarged due to acute lymphadenitis.

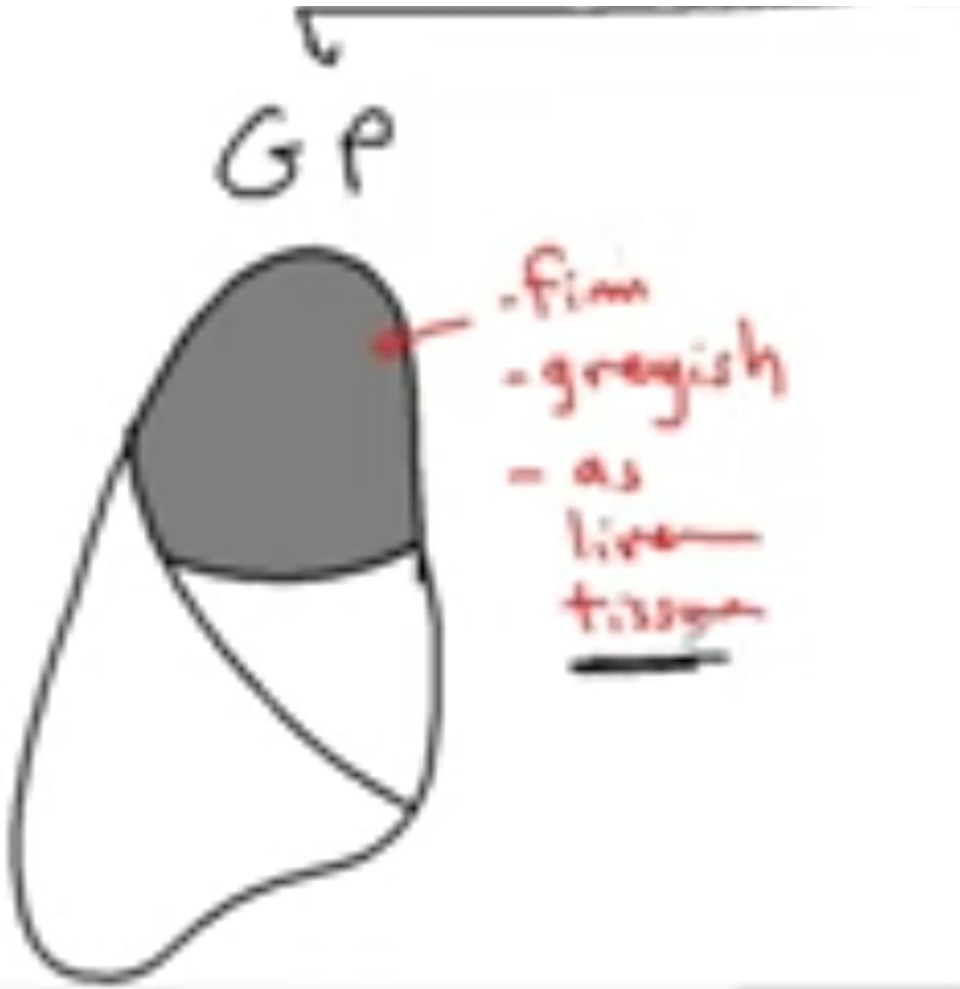
Microscopic Picture:

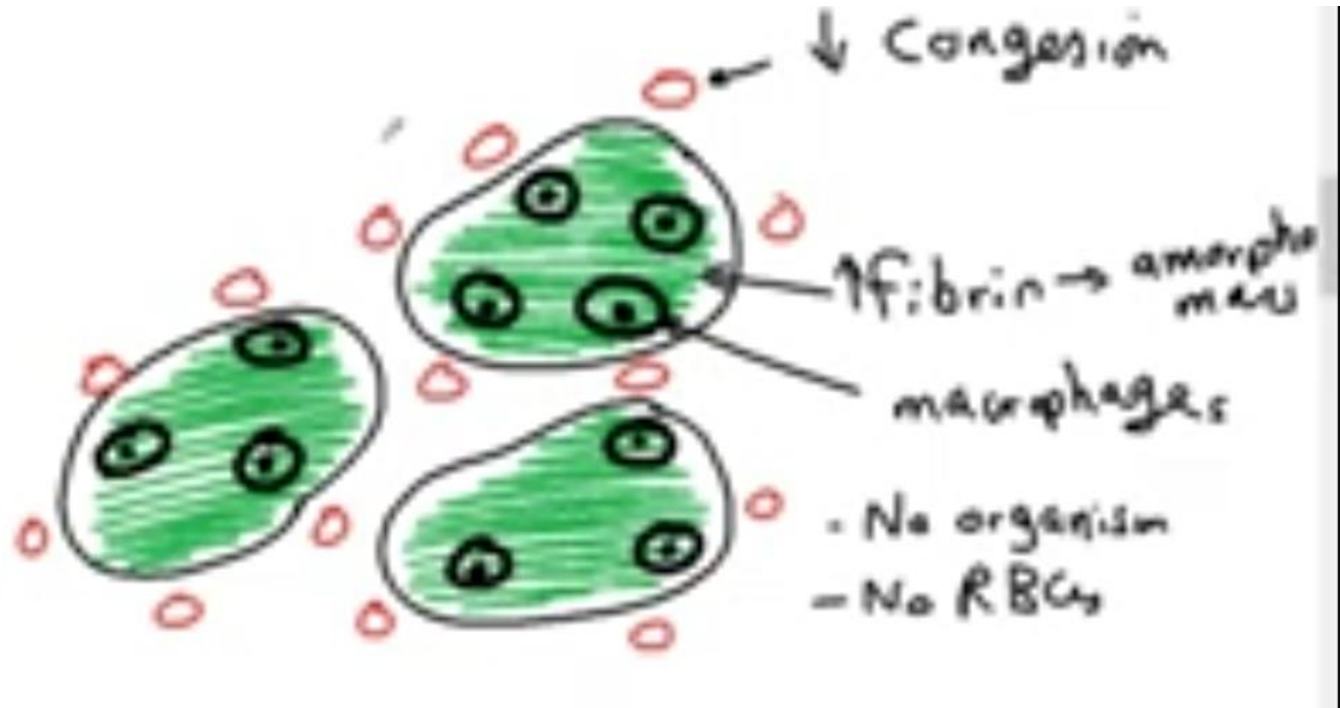
The alveolar capillaries have reduced congestion.

The alveolar walls have thinned (thinner than in red hepatization) due to marked distension of the alveoli.

➢The alveolar spaces contain dead bacteria ,hemolyzed red cells, large number of neutrophils and macrophages.

Fibrin shrinks & form clumped masses in the center of the alveoli


GP
-firm
-greyish
- as
liver
tissue


↓ Congestion
↑ fibrin → amorphous mass
macrophages
. No organism
- No RBCs

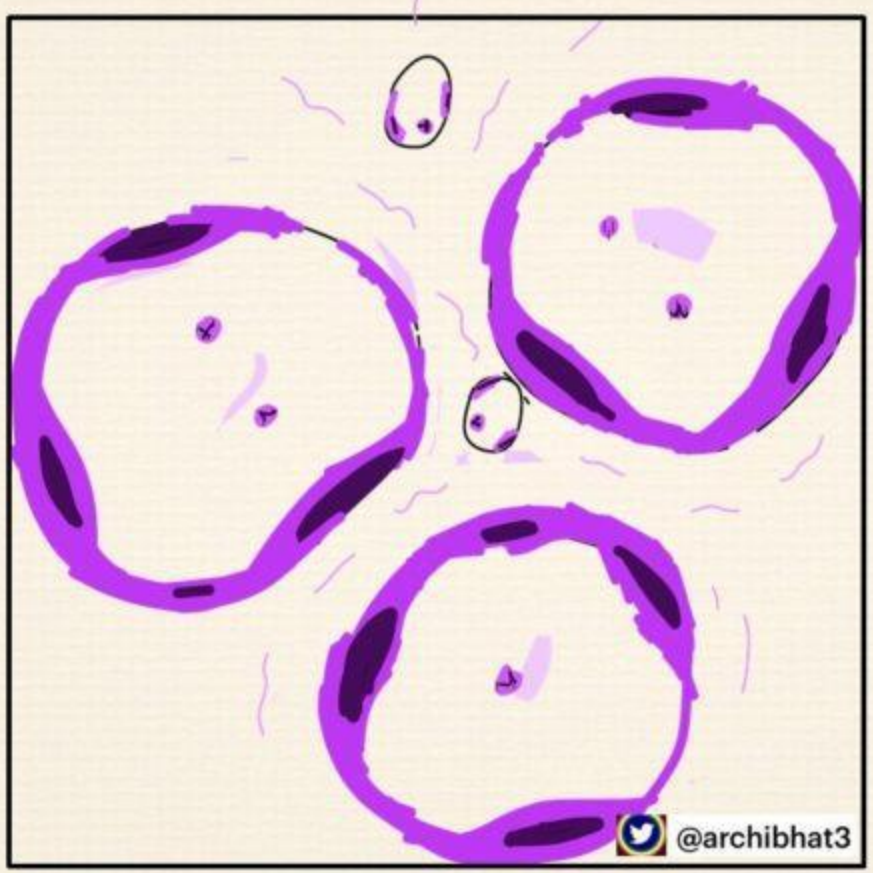
4-Resolution:

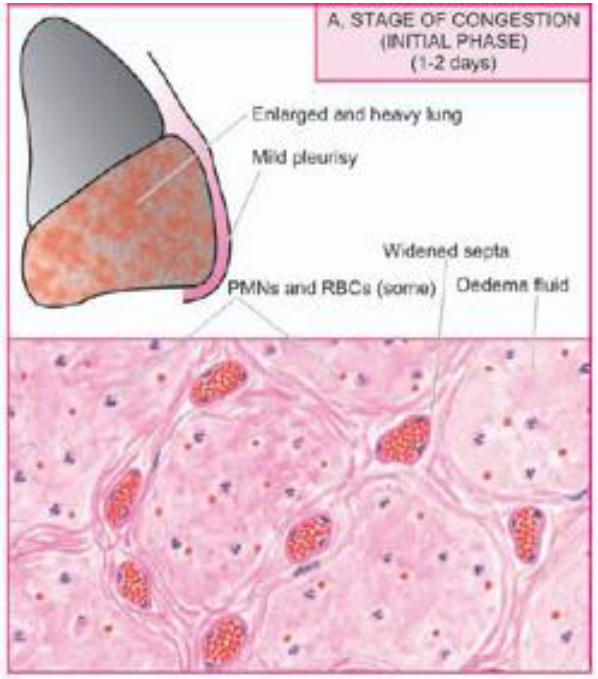
This stage begins by 9th day if no therapy is administered and is completed in 1 to 3 weeks

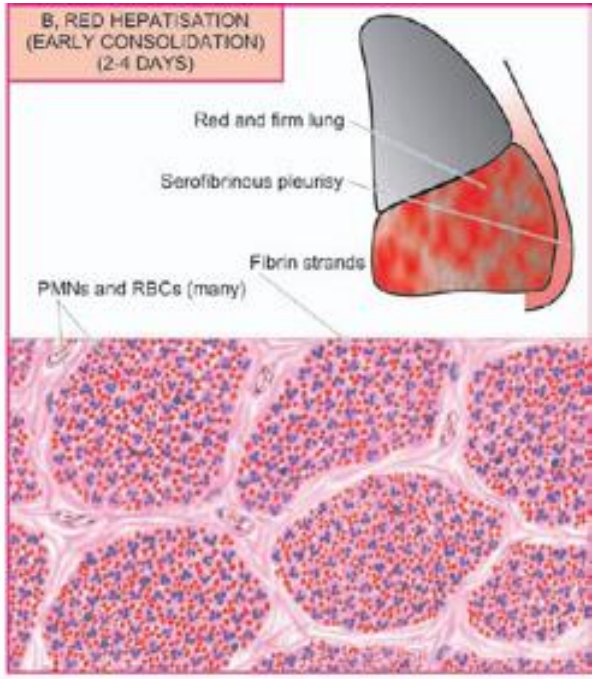
- Gross Picture:
- ➢ The previously solid fibrinous constituent is liquefied by enzymatic action.
- ➢ The affected lobe gradually decreases in size.
- ➢ The cut surface is yellow and soft in consistency.
- Microscopic Picture:
- **Macrophages** are the predominant cells in the alveolar spaces**, while neutrophils** diminish in number.
- ➢ **Fibrin** is liquefied by proteolytic enzymes from dead neutrophils
- ➢ **Lymphatic drainage** of liquefied exudate.
- ➢ Gradually restore normal aeration of the alveoli.
- ➢ The lung returns to normal except for fibrous adhesions from pleurisy

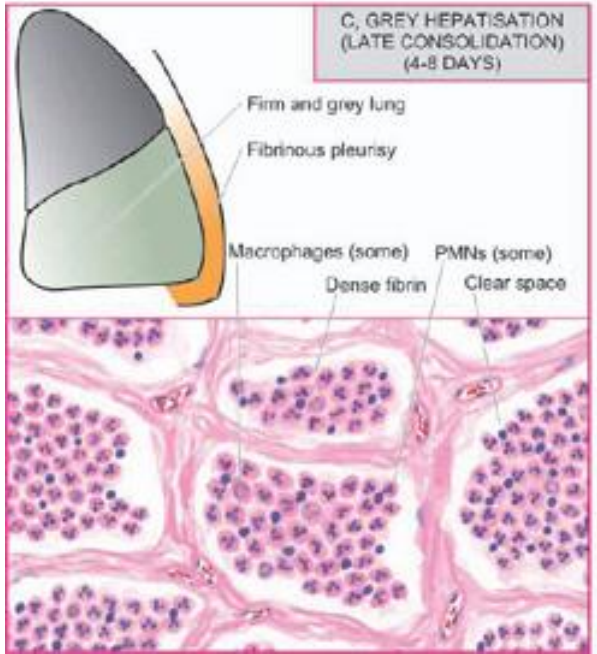
Stage of Resolution

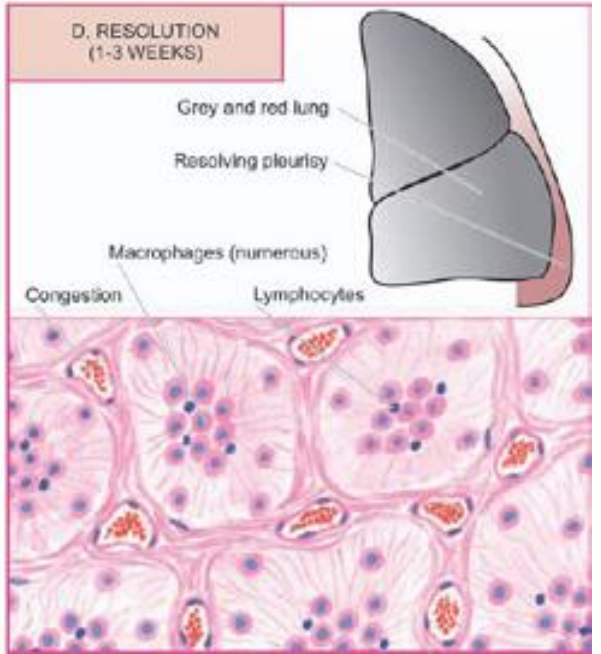
The alveolar spaces look nearly normal now!




A, STAGE OF CONGESTION
(INITIAL PHASE)
(1-2 days)
Enlarged and heavy lung
Mild pleurisy
Widened septa
PMNs and RBCs (some)
Oedema fluid


B, RED HEPATISATION
(EARLY CONSOLIDATION)
(2-4 DAYS)
Red and firm lung
Serofibrincus pleurisy
Fibrin strands
PMNs and RBCs (many)


C, GREY HEPATISATION
(LATE CONSOLIDATION)
(4-8 DAYS)
Firm and grey lung
Fibrinous pleurisy
Macrophages (some)
PMNs (some)
Dense fibrin
Clear space


D, RESOLUTION
(1-3 WEEKS)
Grey and red lung
Resolving pleurisy
Macrophages (numerous)
Congestion
Lymphocytes

Complications of Pneumonia:

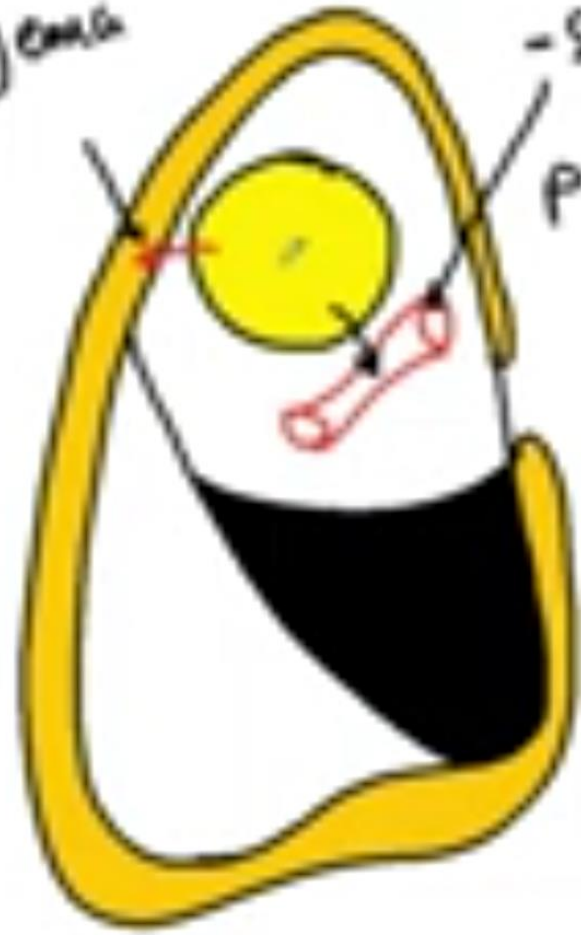
These develop in immunocompromised patients.

- 1.Delayed resolution.
- 2.Failure of resolution leading to lung fibrosis (cornification), resolution of the exudate does not occur but instead it is organized. There is in growth of fibroblasts from the alveolar septa resulting in fibrosed, tough, airless leathery lung tissue.
- 3.Post-pneumonic abscess and gangrene: rare complication
- 4.Local spread of infection causes empyema, pericarditis and mediastinitis.
- 5.Blood spread of infection causes arthritis, meningitis, osteomyelitis and septicemia.
- 6.Toxemia: occurs in 9th day when bacterial toxins are absorbed.
- 7.Toxic myocarditis leading to acute heart failure.

failure of resolution ↓ fibrosis (cavification)

empyema

- acute toxaemia
- septicaemia

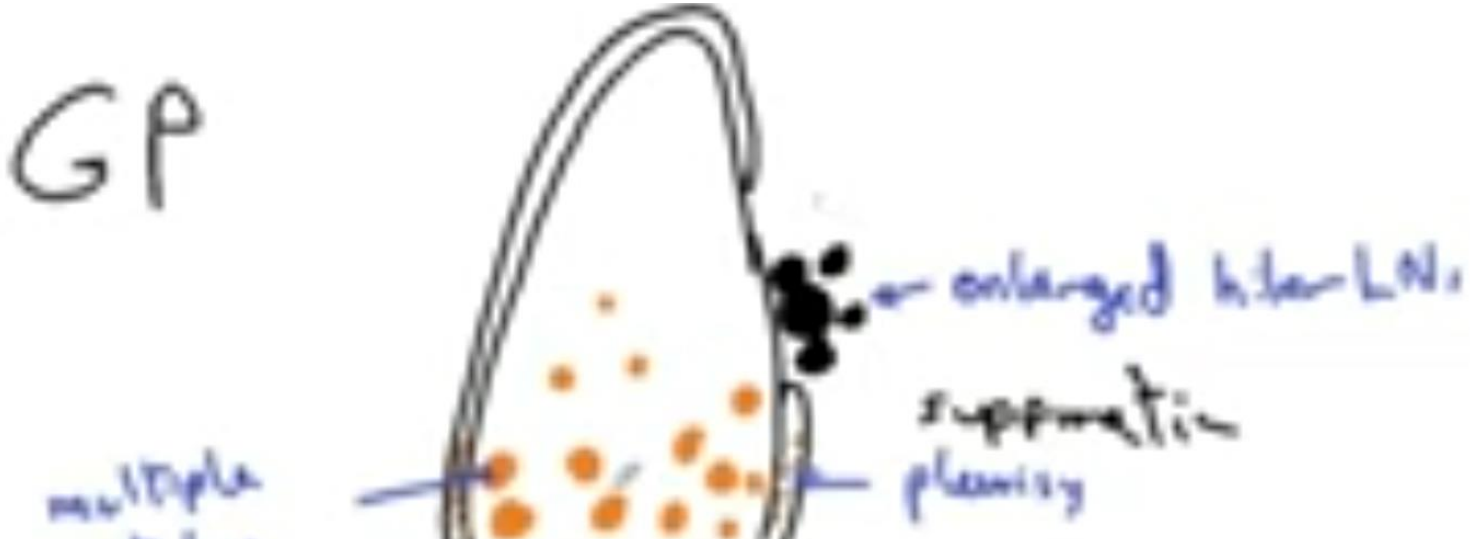


postpneumonic abscess ↓ DM ↑ postpneumonic gangrene

II. Septic Bronchopneumonia

- Definition:
- ➢ Patchy suppurative inflammation of the bronchioles and the
- surrounding lung alveoli, resulting in patchy consolidation of the lung.
- Etiology:
- ➢ Commonly caused by staphylococci, streptococci,
- pneumococci, Klebsiella pneumoniae, Hemophilus influenzae, and gram-negative bacilli like pseudomonas and coliform bacteria.
- ➢ The condition is frequent in infancy and old age, as a terminal
- event in chronic debilitating diseases and as a secondary infection following viral respiratory infections such as influenza, measles etc.

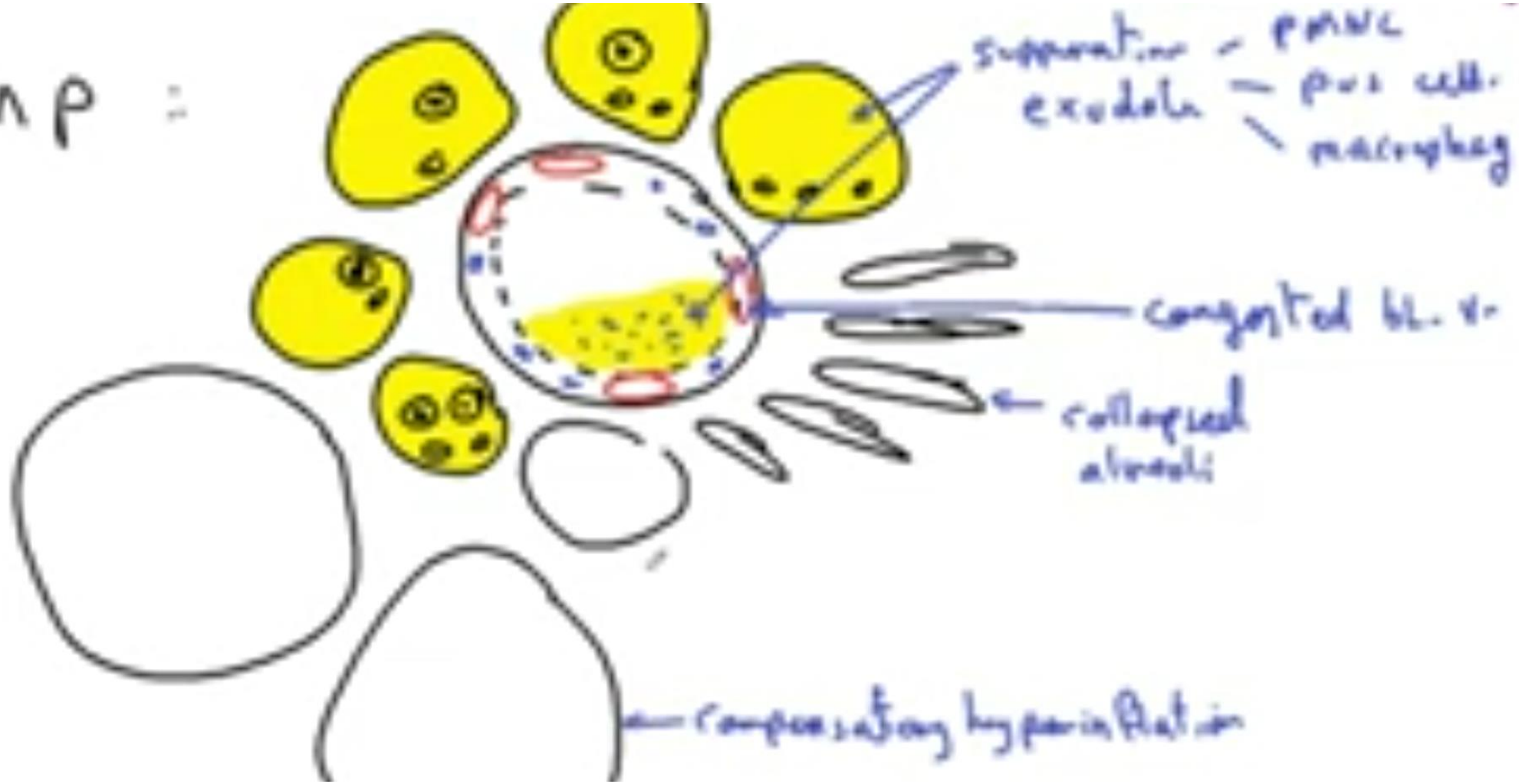
- Gross Picture:
- ➢ Both lungs (bilateral) are usually affected especially the lower lobes due to gravitation of the secretions.
- ➢ The lungs show multiple consolidated yellowish patches exuding pus on pressure.
- ➢ In severe cases, the lesions are numerous and coalesce causing confluent bronchopneumonia.
- ➢ On section, they are severely congested and their bronchi contain pus.
- ➢ The pleura show fibrinous pleurisy.
- ➢ The hilar lymph nodes are enlarged due to acute lymphadenitis.

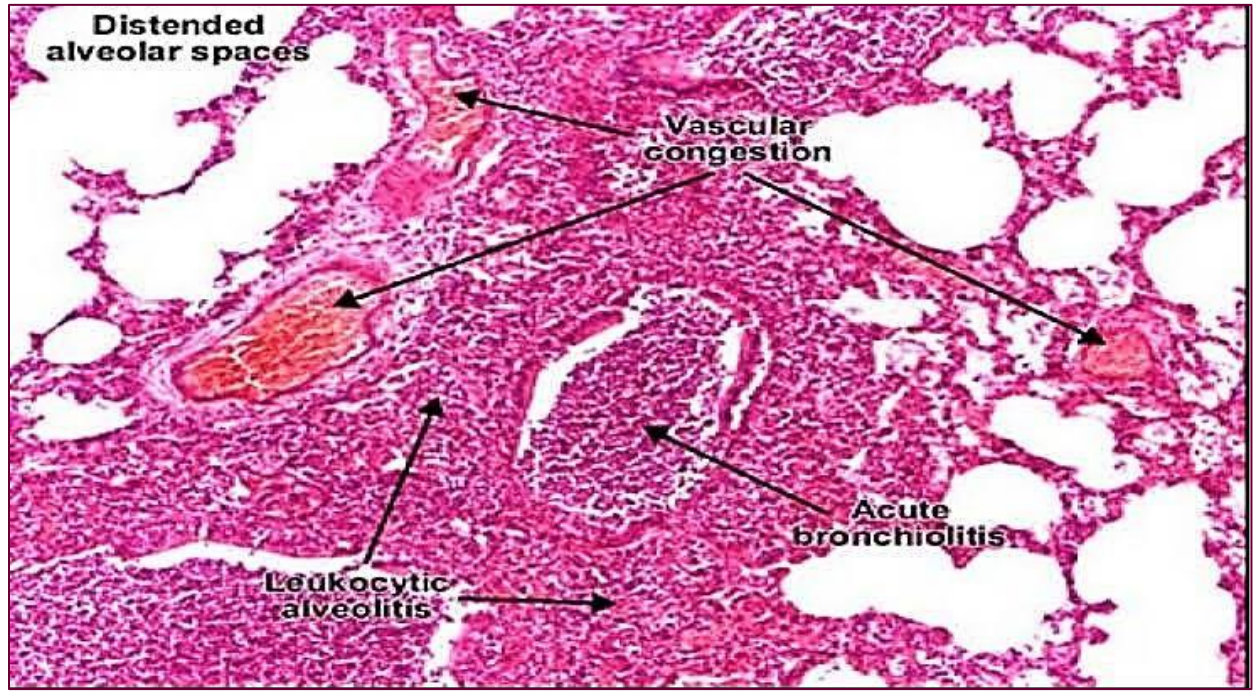


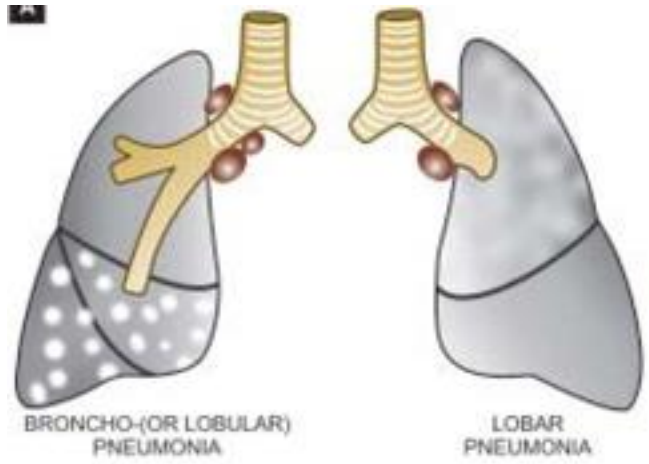
- Microscopic Picture:
- ➢ **The bronchioles show**:
- Wall of the bronchioles show congested capillaries, neutrophils and pus cells.
- The epithelial lining is destroyed and shedded.
- The lumen contains shedded epithelial cells,leucocytes and pus cells.
- ➢ **The adjacent alveoli show:**
- Alveolar inflammation: congested capillaries,neutrophils, pus cells and fibrin.
- alveolar collapse.

  alveolar dilation: compensatory emphysema.

MP :

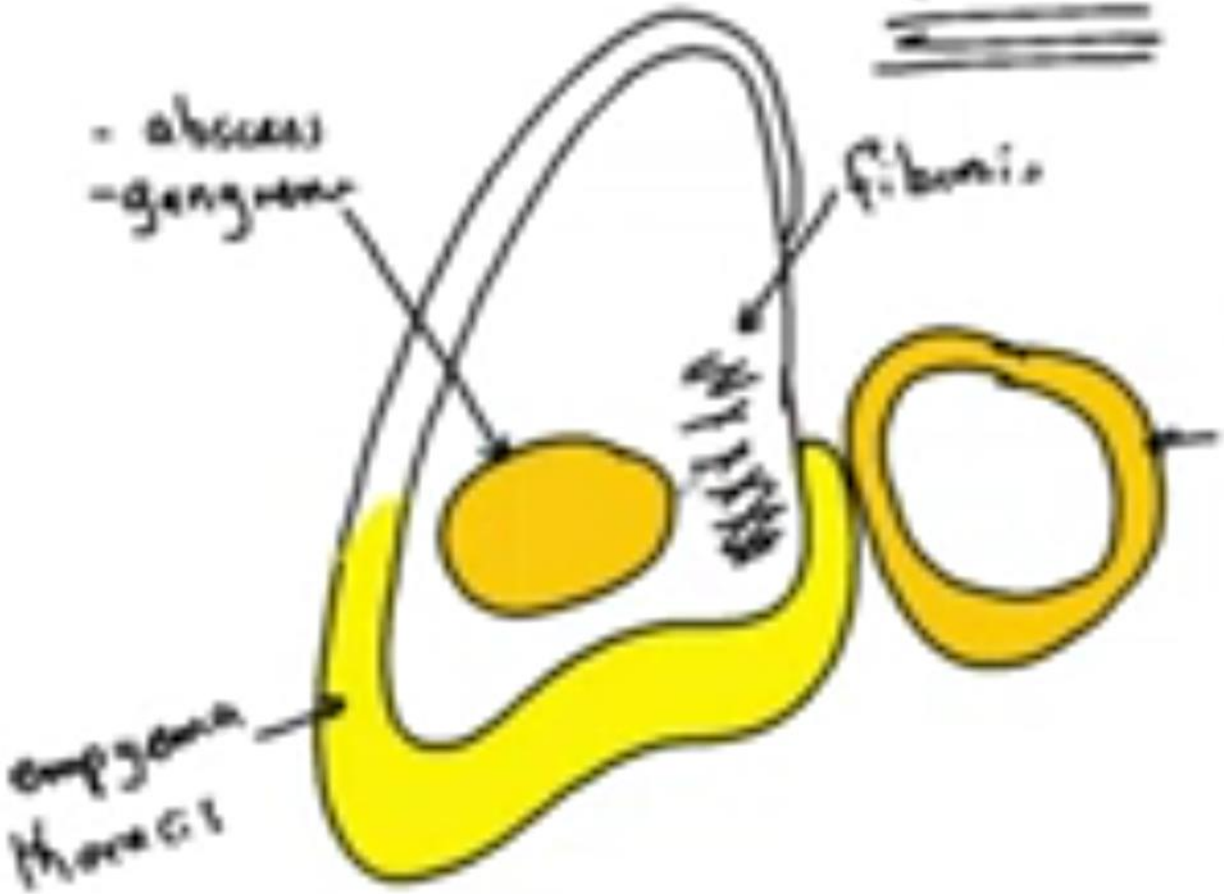

suppurative – PMNC
exudate – pus cells
– macrophag
congested bl. v.
collapsed alveoli
compensatory hyperinflation


Distended alveolar spaces
Vascular congestion
Acute bronchiolitis
Leukocytic alveolitis


BRONCHO-(OR LOBULAR) PNEUMONIA
LOBAR PNEUMONIA

- **Complications:**
- ➢ The complications of lobar pneumonia may occur in
- bronchopneumonia, in addition to bronchiectasis (persistent dilatation of medium-sized
- bronchi & bronchioles, accompanied by chronic suppurative inflammation of their walls) due to destruction of the bronchial walls.

Local



General

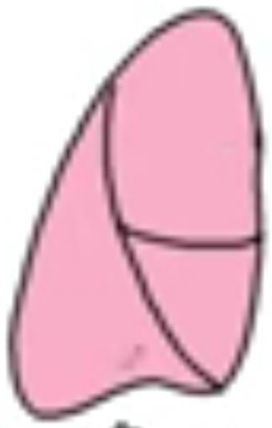
- Toxaemia
- septicaemia
- pyaemia
- Rt. side HF
↓
???
CVC & generalized oed

III. Interstitial Pneumonia

- Definition:
- ➢ They are characterized by patchy inflammatory changes,
- largely confined to interstitial tissue of the lungs without any
- alveolar exudate.
- Etiology:
- a. **Viral infections**: Primary respiratory involvement as influenza
- or Secondary respiratory involvement as in measles or herpes.
- b. **Other infections**: mycoplasma, pneumocystis carinii.
- c. **Chemicals and drugs**: cyclophosphamide, heroin, barbitone.
- d. **Physical irritation:** fumes of burns

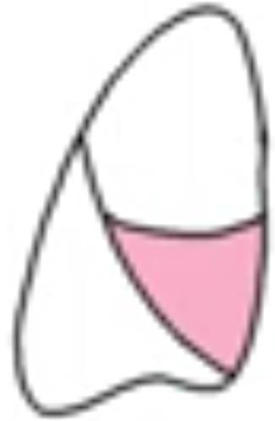
- **A. Mild Cases (Common in viral interstitial pneumonia):**
- Gross Picture:
- Both lungs show peri-bronchical congested patches. The
- patches ooze red frothy fluid on cutting.
- ➢ The covering pleura show fibrinous pleurisy.
- Microscopic Picture:
- ➢ Interstitial inflammation characterized by congested
- capillaries, oedema and infiltration by lymphocytes, plasma cells and macrophages.
- The alveoli are compressed without alveolar necrosis.
- Fate and Complications:
- a. Resolution is common.
- b. Interstitial fibrosis: uncommon.
- c. Secondary bacterial infection: septic bronchopneumonia

- GP



diffuse

patchy



one lobe or more

Bilateral

thickening of alveolar walls

- MP

formation of hyaline membrane

- oedema
- lymphocytes
- plasma cells
- macrophages

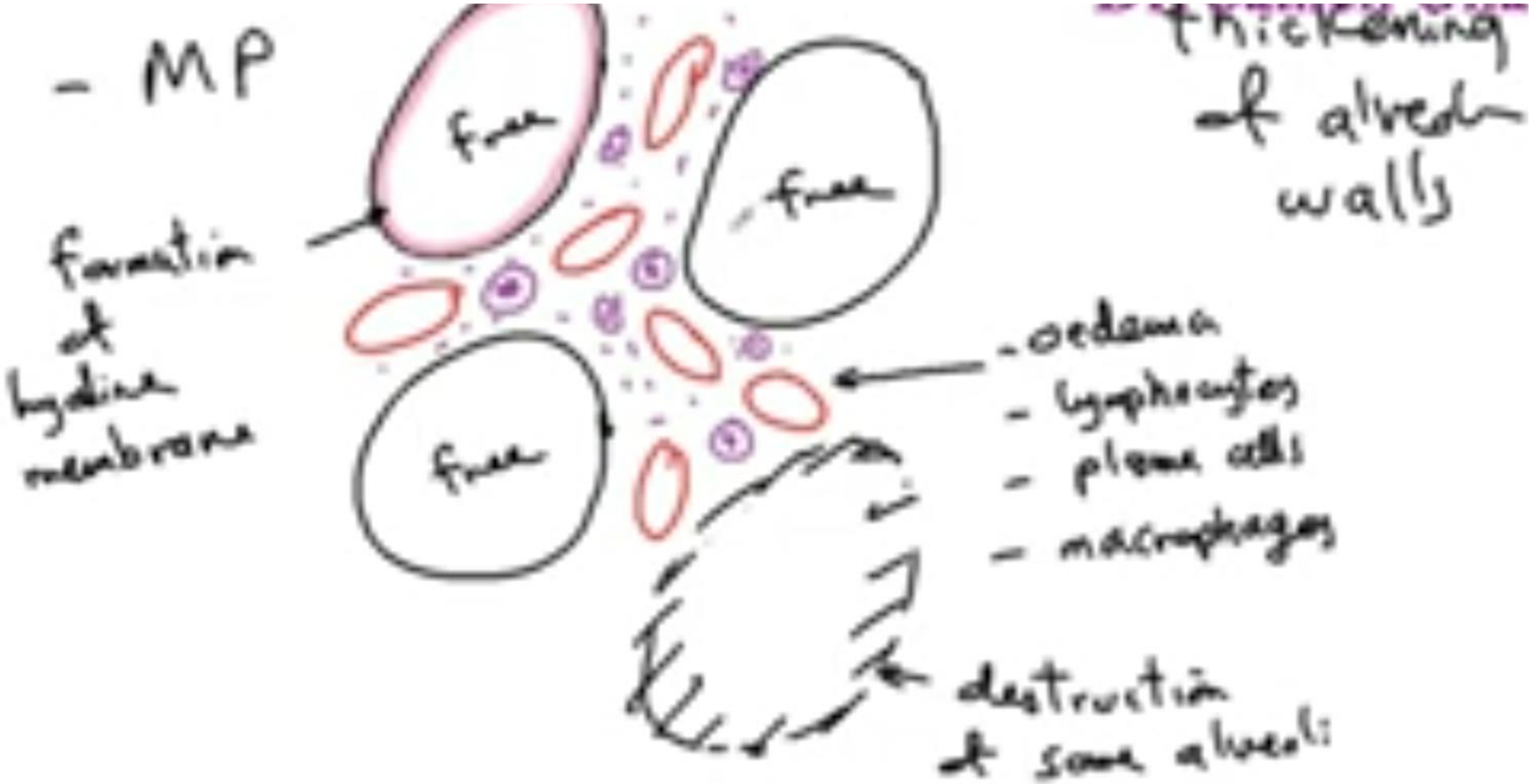
B. Severe Cases: Adult Respiratory Distress Syndrome (ARDS). It is also known as diffuse alveolar damage (DAD) or acute alveolar injury.

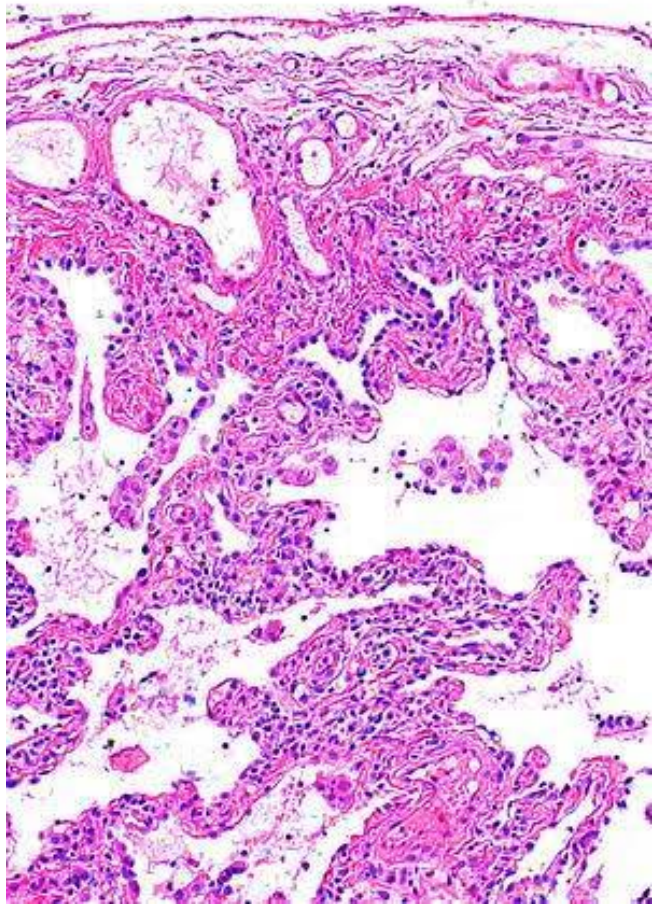
- Early stage (exudative stage):
- Characterized by diffuse alveolar damage, necrosis of alveolar epithelial cells with formation of hyaline membranes, fibrin deposition, hyperplasia of alveolar cells and inflammatory cells.
- Proliferative stage (fibrous phase):

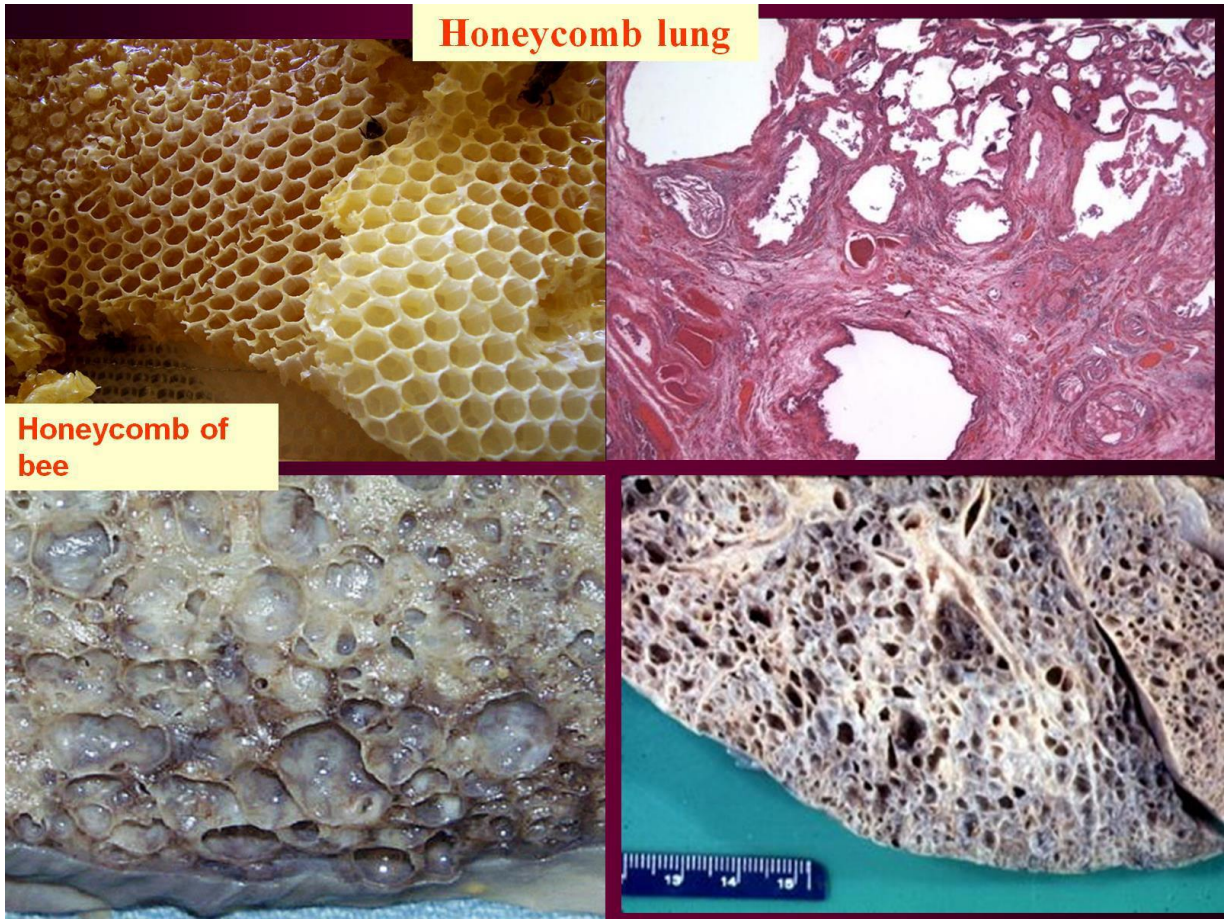
In surviving patients, repair occurs (proliferative phase) ending in interstitial fibrosis (fibrous phase) leading to microcystic changes called honeycomb lung.

Fate and Complications:

Death due to respiratory failure.


- MP
formation of hyaline membrane
fibrin
fibrin
fibrin
- oedema
- lymphocytes
- plasma cells
- macrophages
destruction of some alveoli
thickening of alveolar walls




Honeycomb lung
Honeycomb of bee