

# Pathology of respiratory system

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**Lec 4**

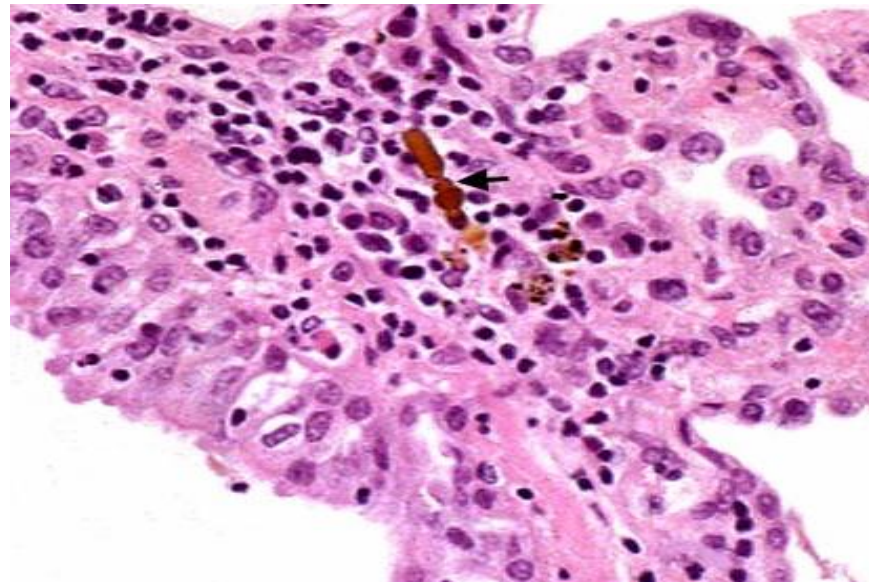
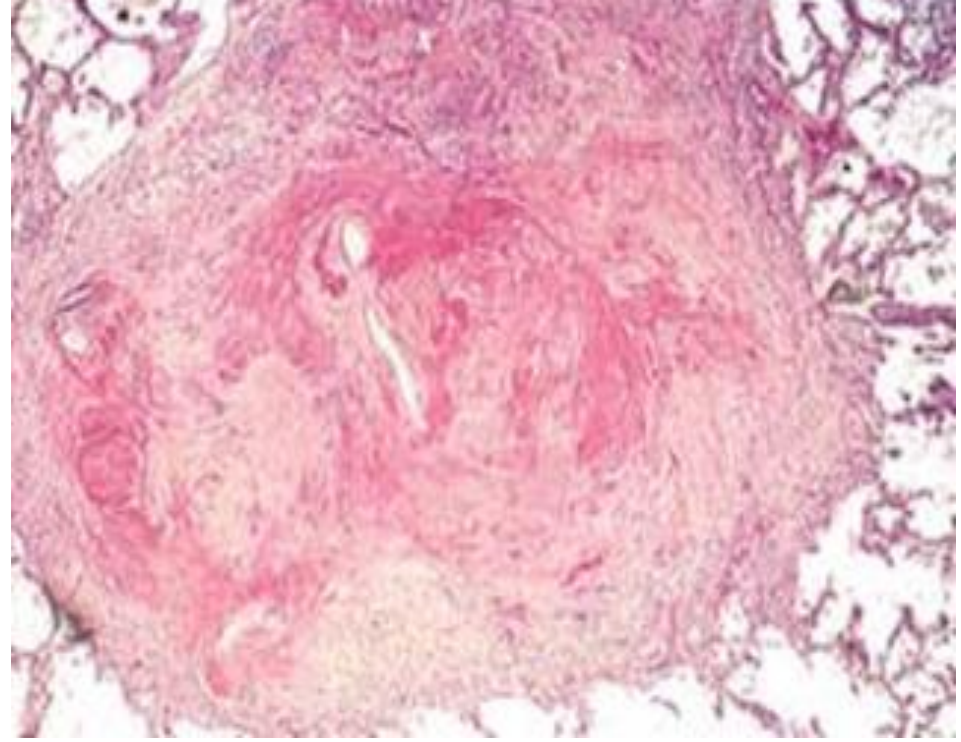
# ***Pneumoconioses:***

Def.:

Interstitial fibrosis of lung due to **inhalation of mineral dusts**

**It is a NON-neoplastic lung reactions to this minerals.**

- **The three most common of these result from exposure to:**
- **coal dust,**
- **silica, and**
- **asbestos;**
- **nearly always due to exposure in the workplace.**
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# Pathogenesis

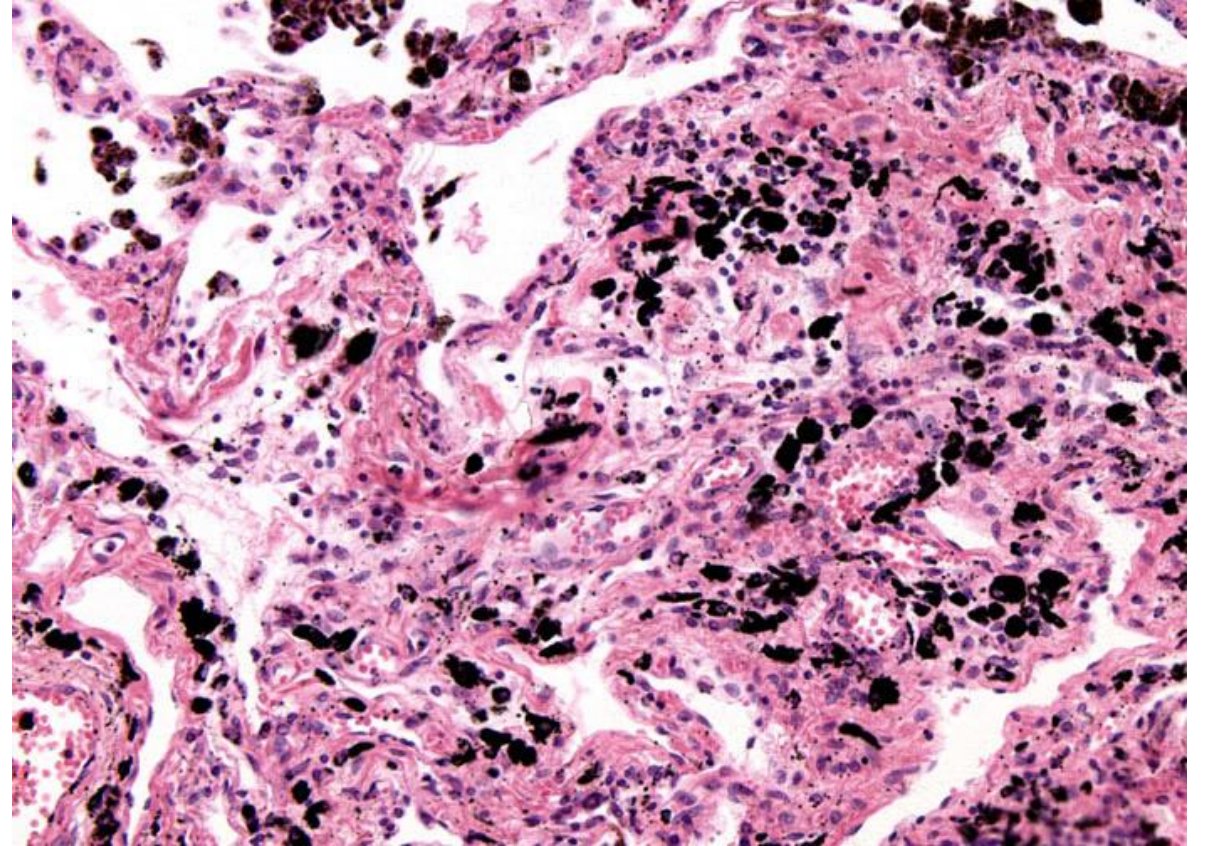
- The reaction of the lung to mineral dusts depends on the size, shape, solubility, and reactivity of the particles;
- *particles that are 1 to 5  $\mu\text{m}$  are the most dangerous, because they lodge at the bifurcation of the distal airways.*
- **Coal dust** is relatively **inert**, and large amounts must be deposited in the lungs before the disease is clinically apparent.
- **Silica, asbestos** are more reactive than coal dust, resulting in fibrotic reactions at lower concentrations.
- *The pulmonary alveolar macrophage play central role in the initiation and progression of lung injury and fibrosis.*
- Tobacco smoking worsens the effects of all inhaled mineral dusts, but particularly asbestos particle.

# **1. Coal Workers' Pneumoconiosis**

- The spectrum of lung findings in coal workers includes:
- a. **Asymptomatic anthracosis**, in which pigment accumulates without cellular reaction (NO fibrosis). It is also commonly seen in all urban dwellers and tobacco smokers. Inhaled carbon pigment is engulfed by alveolar or interstitial macrophages, which then accumulate in the connective tissue.
- b. **Simple coal workers' pneumoconiosis** is characterized by formation of **fibrotic nodules**
- c. **Progressive massive fibrosis** develops in 10% of those with the above; it occurs through the **coalescence of the fibrotic nodules**.

**There is NO increased frequency of bronchogenic carcinoma**

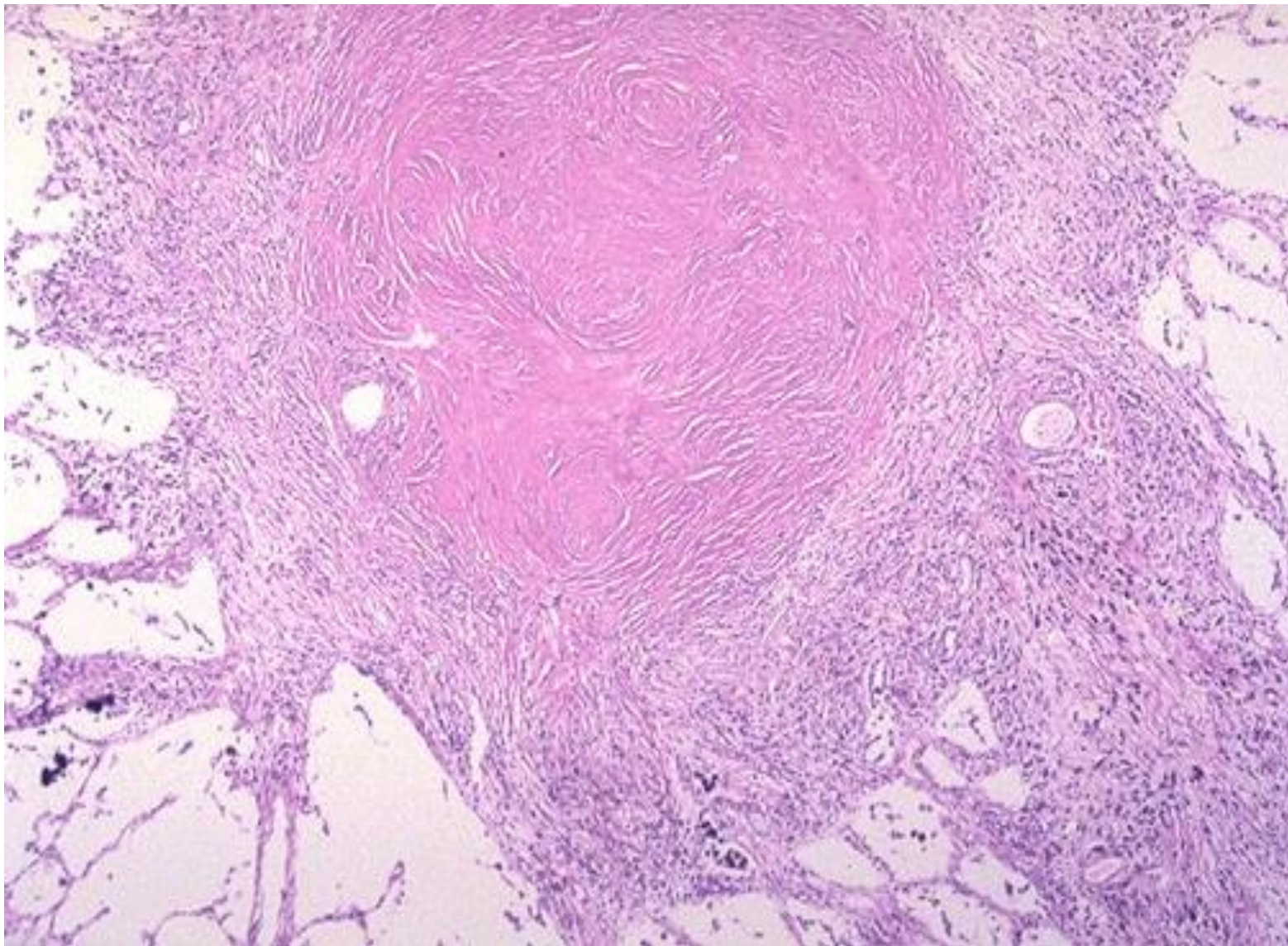
# Anthracosis



Asymptomatic anthracosis : pigment accumulates in the lungs without cellular reaction. It is also commonly seen in all urban dwellers and tobacco smokers. Inhaled carbon pigment is engulfed by alveolar or interstitial macrophages, which then accumulate in the connective tissue along the lymphatics

## 2- Silicosis:

- Silicosis is the most common chronic occupational disease in the world.
- It is caused by inhalation of silica crystals mostly quartz
- The condition is characterized by the formation of **silicotic nodules** involving the upper zones of the lungs.
- Silicosis is associated with an **increased susceptibility to tuberculosis because crystalline silica inhibits the ability of pulmonary macrophages to kill phagocytosed mycobacteria**
- **Silica from occupational sources is carcinogenic in humans.** However, this subject continues to be controversial.



Silicotic nodule

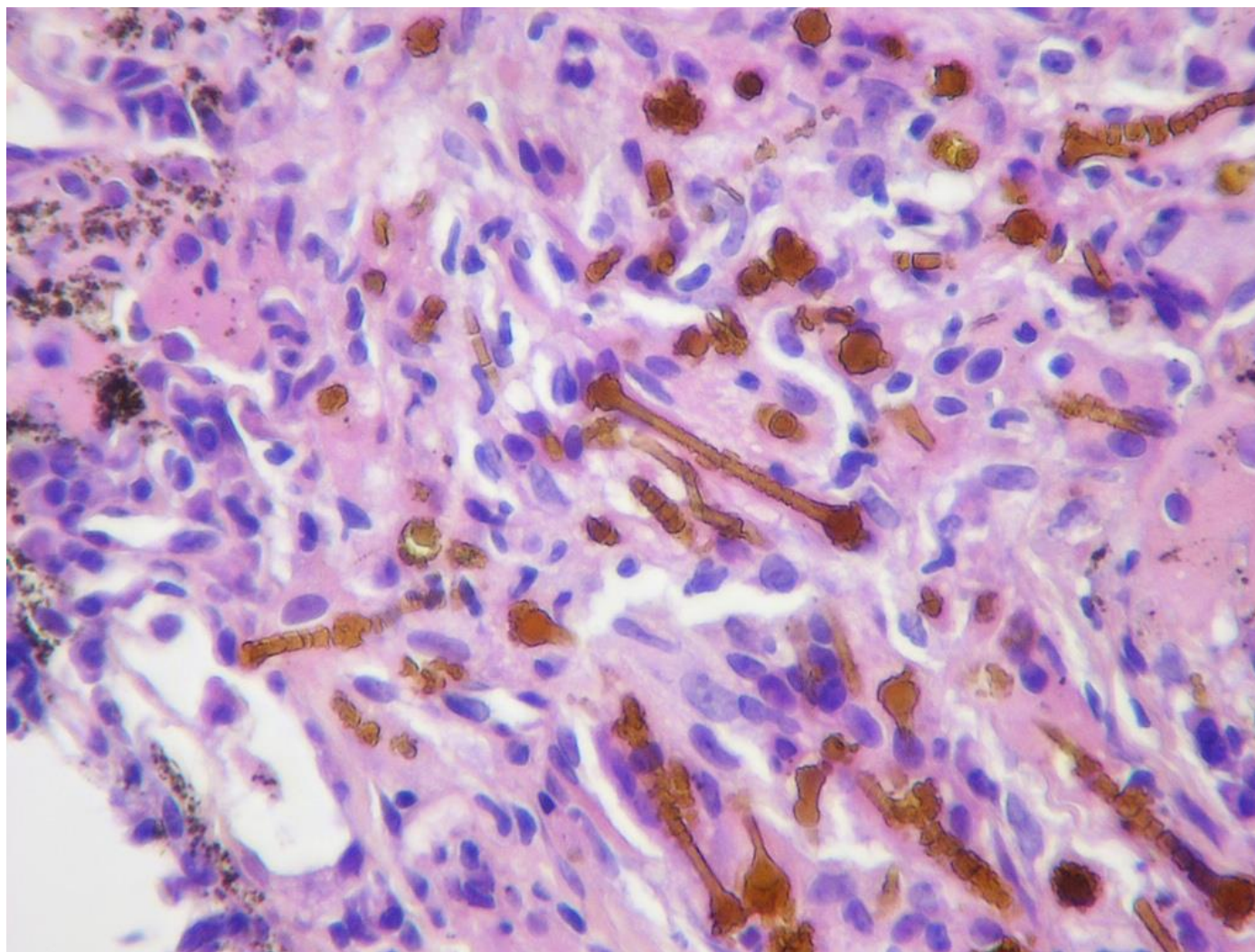


- **3. Asbestosis and Asbestos-Related Diseases**

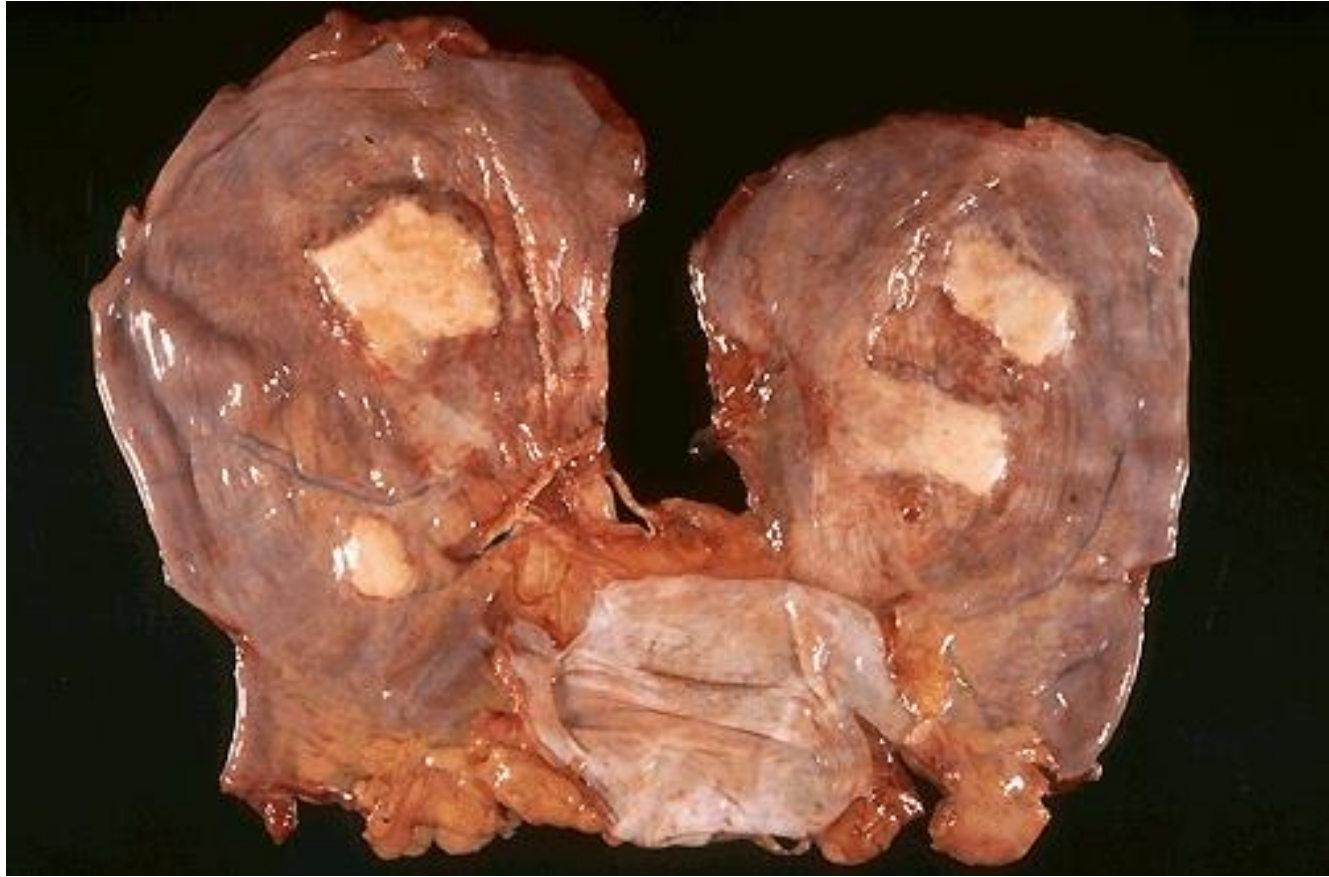
- Asbestos is a family of silicate crystals with a fibrous spatial arrangement.
- It cause a wide spectrum of diseases depending on concentration, size, shape, and solubility
- **Occupational exposure to asbestos is associated with :**
  - 1-Localized fibrous plaques or, rarely, diffuse pleural fibrosis
  - .2- recurrent pleural effusions.
  - 3- Parenchymal interstitial fibrosis
  - 4-Lung carcinoma
  - 5-Mesothelioma(pleural, peritoneal)
  - 6-Laryngeal, ovarian, and perhaps other extrapulmonary neoplasms, including colon carcinoma

- **Pathogenesis:** Once phagocytosed by macrophages, asbestos fibers
- activate the inflammation and stimulate the release of proinflammatory factors and fibrogenic mediators

- **Asbestosis** : diffuse pulmonary interstitial fibrosis & characteristically shows the presence of **asbestos bodies**, which are seen as golden brown, beaded rods. They consist of asbestos fibers coated with an iron-protein material.
- **Pleural plaques** are the most common manifestation of asbestos exposure and are well-circumscribed patches of dense collagen that develop most frequently on the parietal pleura and over the domes of the diaphragm.
- The risk of bronchogenic carcinoma is increased about five times for asbestos workers.
- The risk for mesotheliomas, normally a very rare tumor, is more than 1000 times greater.
- Concomitant cigarette smoking greatly increases the risk of bronchogenic carcinoma but not that of mesothelioma.
- The carcinoma & mesothelioma associated with asbestos exposure have a particularly poor prognosis.



Asbestos bodies : golden brown, beaded rods.  
They consist of asbestos fibers coated with an iron-protein material.



Asbestosis with **fibrous pleural plaque** well-circumscribed patches of **dense collagen** that develop most frequently on the parietal pleura and over the domes of the diaphragm.

# Respiratory Pathology Outline

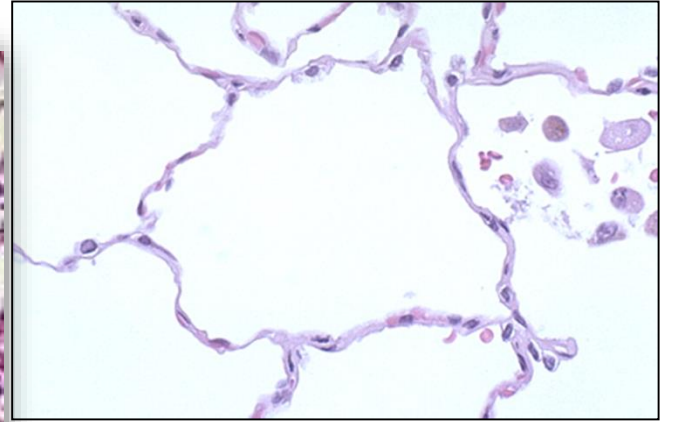
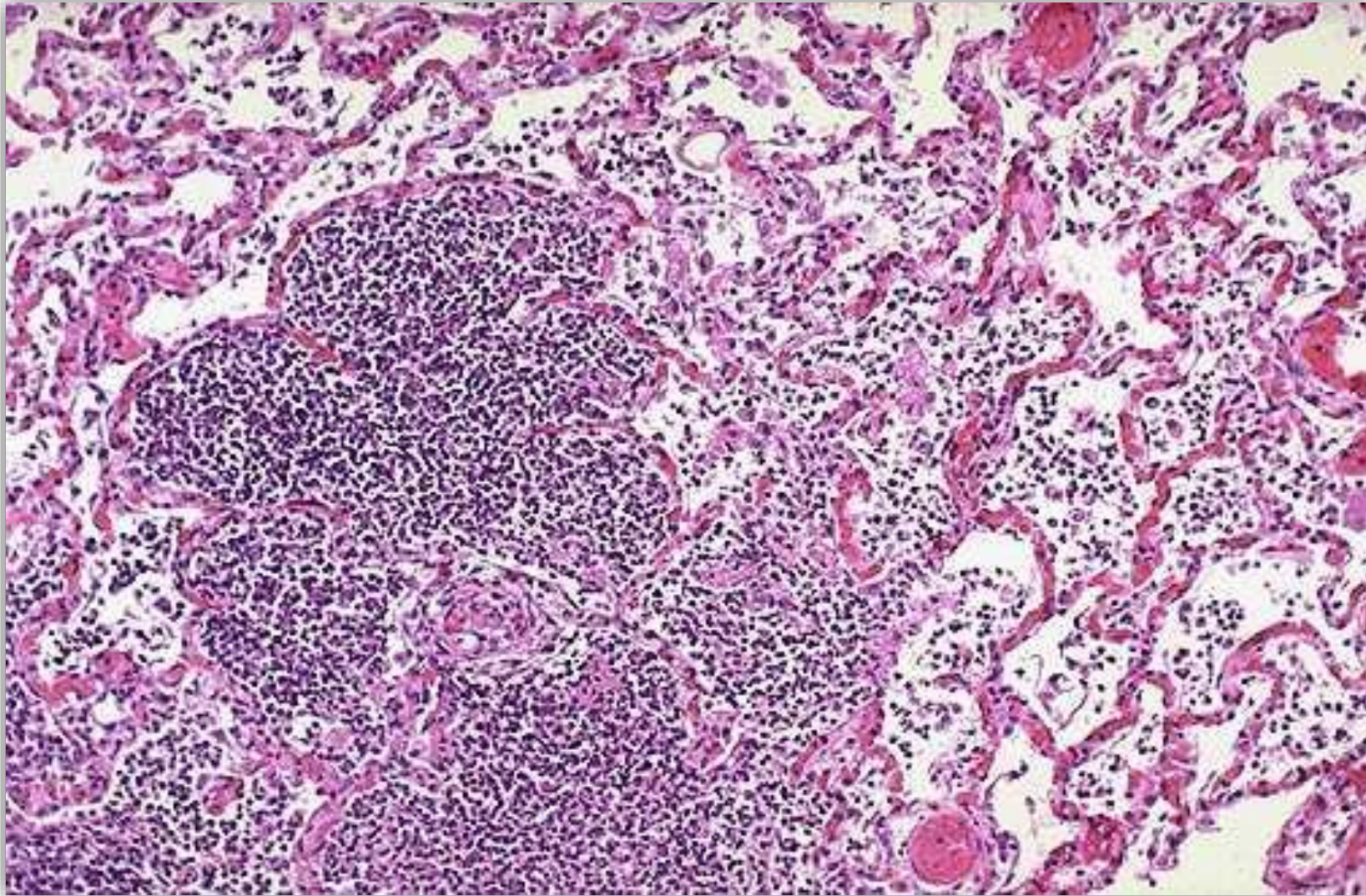
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- Congenital anomalies
- Atelectasis
- Obstructive lung diseases
- Restrictive lung diseases
- **Infections(pneumonia)**
- Carcinoma

# Pneumonia

- **Definition:** Inflammation of lung parenchyma .Characterized by consolidation .
- **Consolidation:**  
Replacement of the alveolar air by inflammatory exudates.

# Pneumonia:





- **Predisposing factors:** Pneumonia can result whenever :
  - **1- Decrease in systemic resistance** of the host
  - or
  - 2- impairment of **local defense** mechanisms.
- **Factors that decrease systemic resistance:**  
include:
  - chronic diseases,
  - immunologic deficiencies,
  - treatment with immunosuppressive agents,
  - and leukopenia.

**Local pulmonary defense mechanisms** may also be compromised by many factors, including:

- **1- Loss or suppression of the cough reflex**, as a result of altered sensation (e.g., coma), anesthesia, neuromuscular disorders, drugs, any of which may lead to *aspiration* of gastric contents.
- **2-Dysfunction of the mucociliary apparatus**, which can be
  - caused by cigarette smoke, inhalation of hot or corrosive gases, viral diseases, or genetic defects of ciliary function (e.g., immotile cilia syndrome).
- **3-Accumulation of secretions** in conditions such as cystic fibrosis and bronchial obstruction (e.g due to lung tumor).
- **3- Interference with the phagocytic and bactericidal activities of alveolar macrophages** by alcohol, tobacco smoke, or oxygen intoxication.
- **4-Pulmonary congestion and edema.**

## **Classification of pneumonia:**

1- According to **anatomical (and radiographic) patterns**

2- According to **setting in which the pneumonia is occurred**

### **3-Microbiological classification**

It Could be :

bacterial (90% due to streptococcal pneumoniae),

Viral,

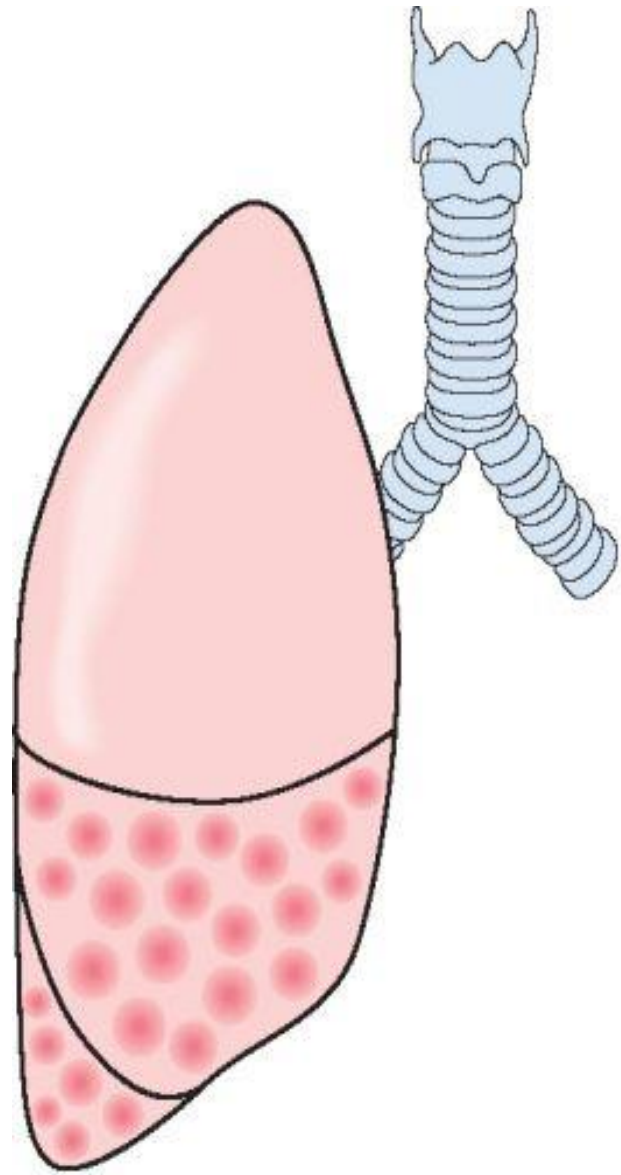
fungal and

Mycoplasma.

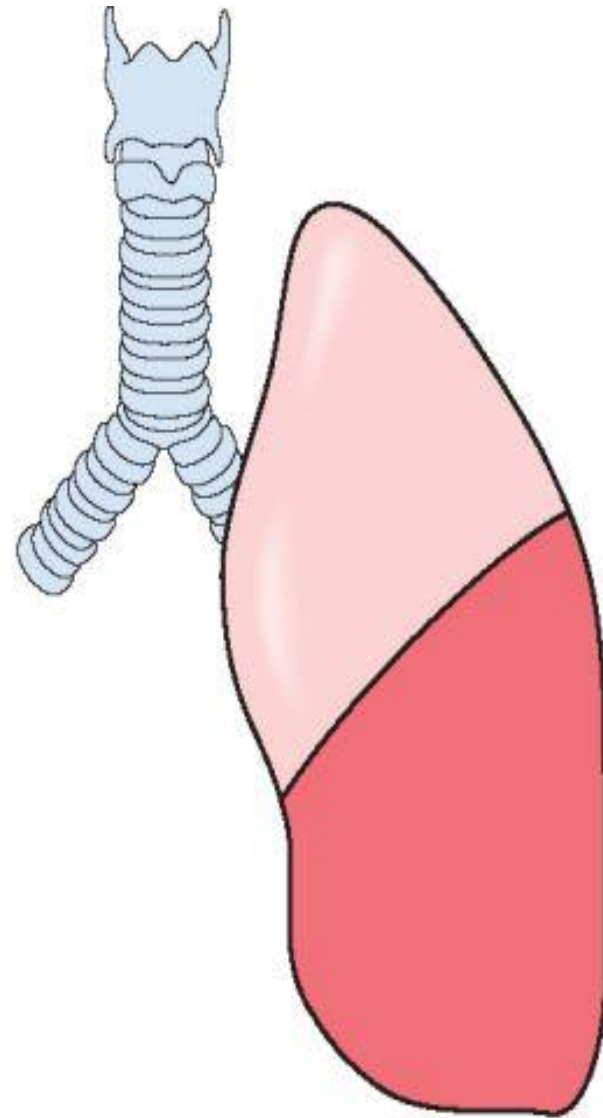
- Classification of pneumonia:

- I. According to anatomical (and radiographic) patterns:

- 1) Bronchopneumonia showing a **patchy distribution** of inflammation that generally involves more than one lobe. The initial infection is of the bronchi and bronchioles with extension into the adjacent alveoli.
- 2) Lobar pneumonia, which affect the airspaces of **part or all of a lobe**; these are homogeneously filled with an exudate that can be visualized on radiographs as a lobar or segmental consolidation.
- **Streptococcus pneumoniae is responsible for more than 90% of lobar pneumonias.**



Bronchopneumonia



Lobar pneumonia

Bronchopneumonia. Gross section of lung showing patches of consolidation (arrows).



Lobar pneumonia  
gray hepatization,  
gross photograph.  
The lower lobe is  
uniformly  
consolidated.

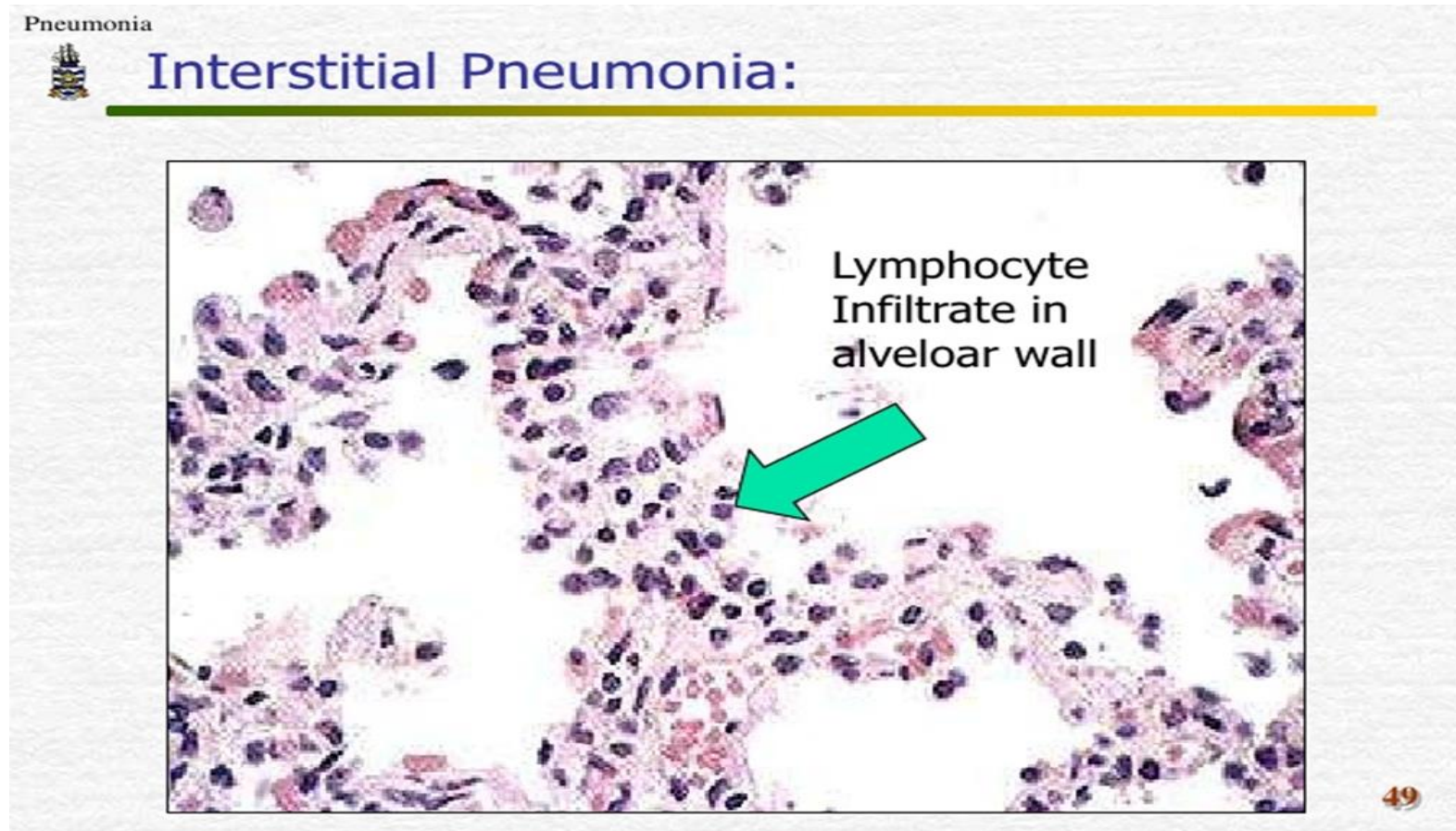


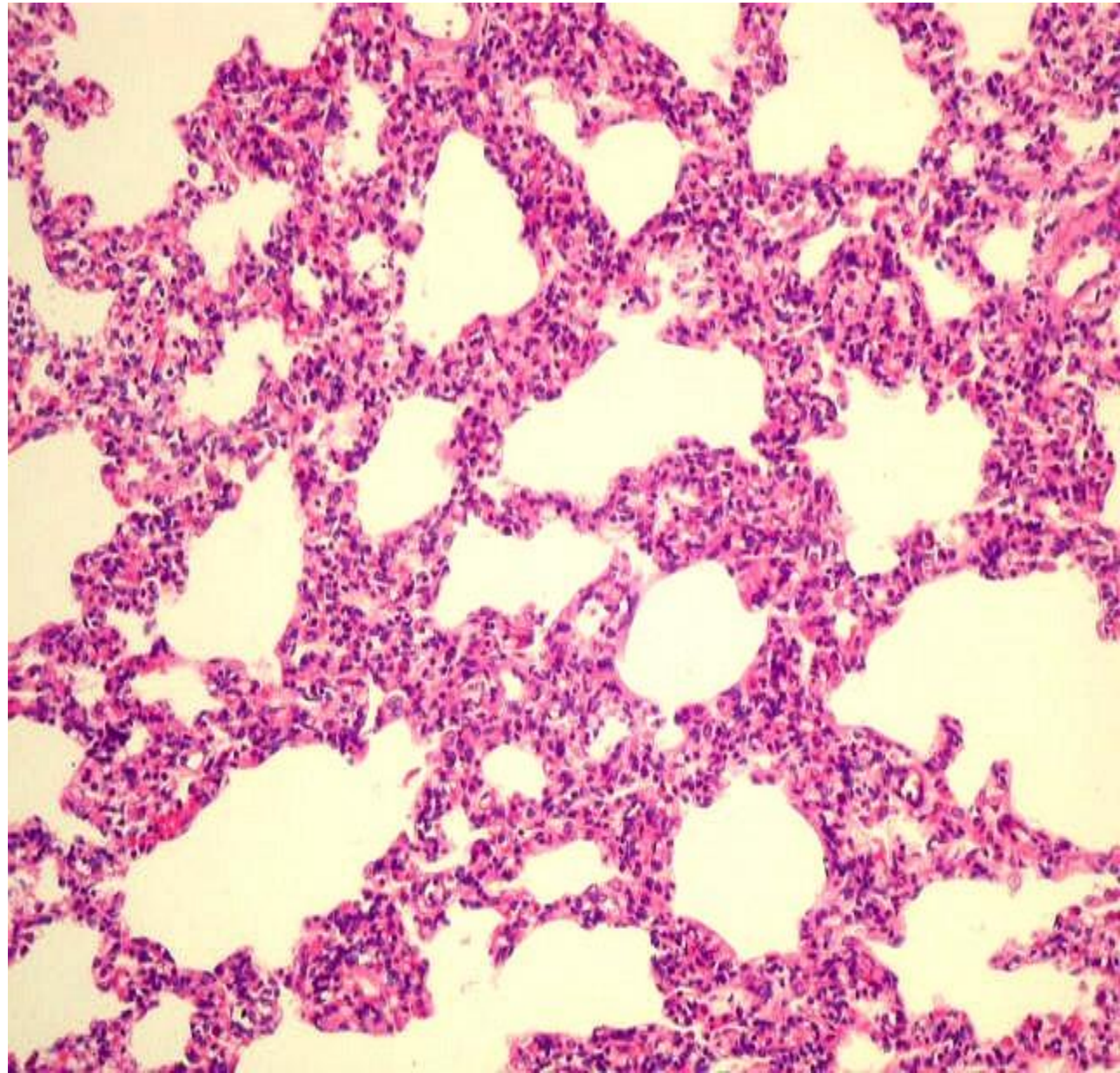
- **The anatomic distinction between lobar pneumonia and bronchopneumonia is often become blurred because:**
  1. **many organisms can produce either of the two patterns of distribution**
  2. **confluent bronchopneumonia can be hard to distinguish radiologically from lobar pneumonia.**

**Most important from the clinical standpoint are identification of the causative agent and determination of the extent of disease.**



- 3) **Interstitial Pneumonia(atypical)**:
- Inflammation in the of walls of alveoli and connective tissue of lung interstitial tissue.
- Typically **NO ALVEOLAR EXUDATE**





**2- According to setting in which the pneumonia is occurred:**

**1- Community acquired acute pneumonia**

**2- Community acquired atypical pneumonias**

**3- Nosocomial Pneumonia (hospital-acquired)**

**4- Aspiration Pneumonia**

**5- Necrotizing pneumonia & Lung Abscess**

## 1- Community acquire acute pneumonia:

characterized by

- This is the ***commonest type.***
- Due to **bacterial pneumonia** follows viral upper respiratory tract infection.
- Most important causative agent is **Pneumococci,** and **klepsilla**
- ***Increased risk in patients with congestive heart failure, chronic obstructive lung diseases, D.M, AIDS, absent spleen.***

## **2- Community acquired Atypical pneumonias:**

Differ from acute pneumonia by:

- ***Sputum production is modest.***
- ***No signs of consolidation.***
- ***WBC count was only moderately increased.***
  - Most important causative agent is ***Mycoplasma pneumoniae.***

### **Diagnostic tests:**

- **PCR** (for detect DNA of Mycoplasma),

### 3- Nosocomial Pneumonia (hospital-acquired):

- Defined as "*pulmonary infections acquired in the course of a hospital stay*".
- They are common in hospitalized persons with severe illness, immune suppression, or prolonged antibiotic therapy.
- Gram negative rods and Staphylococcus aureus are the most common cause.
- As soon as you step into a hospital, expect to be greeted by MRSA.
- Two things live in hospitals: 1) bacteria resistant to antibiotics  
2) sick people

#### **4- Aspiration Pneumonia:**

- Occurs in markedly debilitated patients or those who **aspirate gastric contents** either while unconscious (e.g., after a stroke) or during repeated vomiting.
- The resultant pneumonia is **partly chemical**, resulting from the extremely irritating effects of the gastric acid, and **partly bacterial**.
- This type of pneumonia is often **necrotizing** with a **fulminant(sever and sudden) clinical course**.
- In those who survive, **abscess formation** is a common complication.

## **5- Necrotizing pneumonia & Lung Abscess**

- Necrotizing pneumonia often coexists or evolves into lung abscess, making the distinction between the two somewhat subjective.



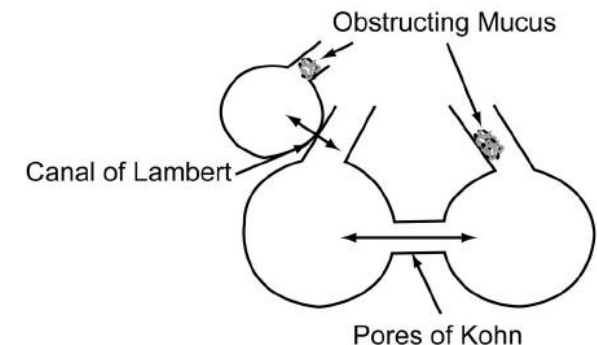
## Lobar pneumonia:

means inflammation limited to part or all parts of one lobe (fill with exudate & consolidation).

**Predisposing factors**: URTI, The M.O. reach the alveoli through the bronchial tree & spread from alveoli to alveoli through **pores of Kohn**.

Typically bacterial 90-95% are caused by **pneumococci**.

- Others are klebsiella pneumonia , staphylococcus, strept., H. influenza
- viral.



- **Gross:** a complete lobe is involved (consolidated)
- **Microscopically:** For descriptive purposes divided in to 4 stages

- There are **four stages** of evolvement of lobar pneumonia, they are:

**1- Congestion stage:** initial congestion due to **vascular engorgement** and Intra-alveolar proteinaceous fluid with neutrophils + many bacteria.

**2- Red hepatization:** marking a stage of massive neutrophilic exudation with hemorrhage (grossly resembling liver)

- **Grossly:** lobe is **red, firm, airless, look like a liver.**
- **Mic.:** The inflammatory exudate composed of **RBC+ neutrophils+ fibrin.**

**3- Gray hepatization** : characterized by red cell disintegration but persistence of fibrinopurulent exudates.

- **Grossly:** grey –brownish, dry surface & firm.
- **Mic.:** The exudate within alveoli is fibrino-suppurative (WBC+FIBRIN+LYSED RBC).

**4- Resolution:**

marked by progressive **enzymatic digestion** of the exudates and macrophage resorption of the debris, or fibroblast ingrowth.

Exudate resolution usually restores normal lung structure and function, but organization with fibrous scarring can occur.

Normal

4. Resolution

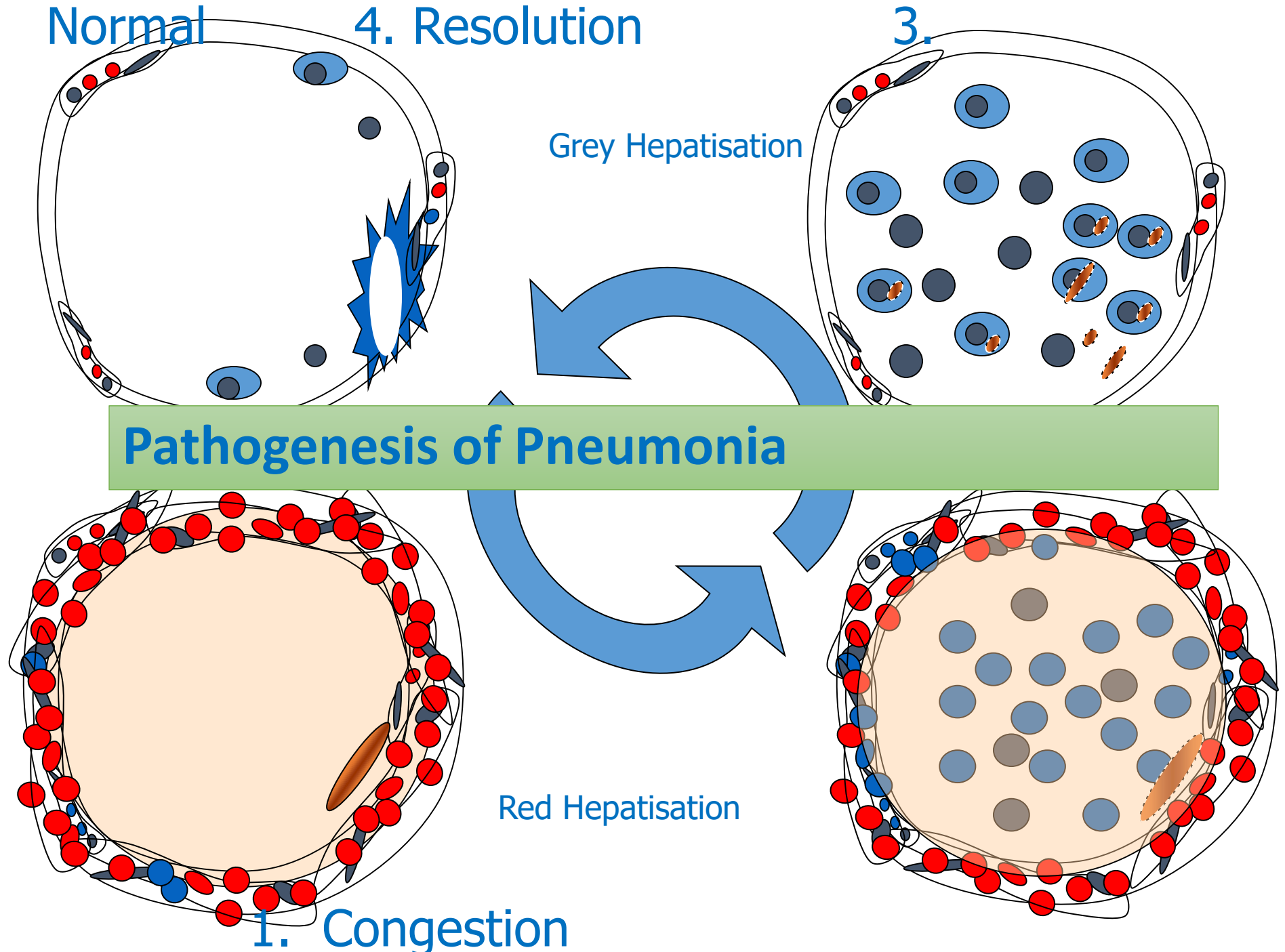
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Grey Hepatisation

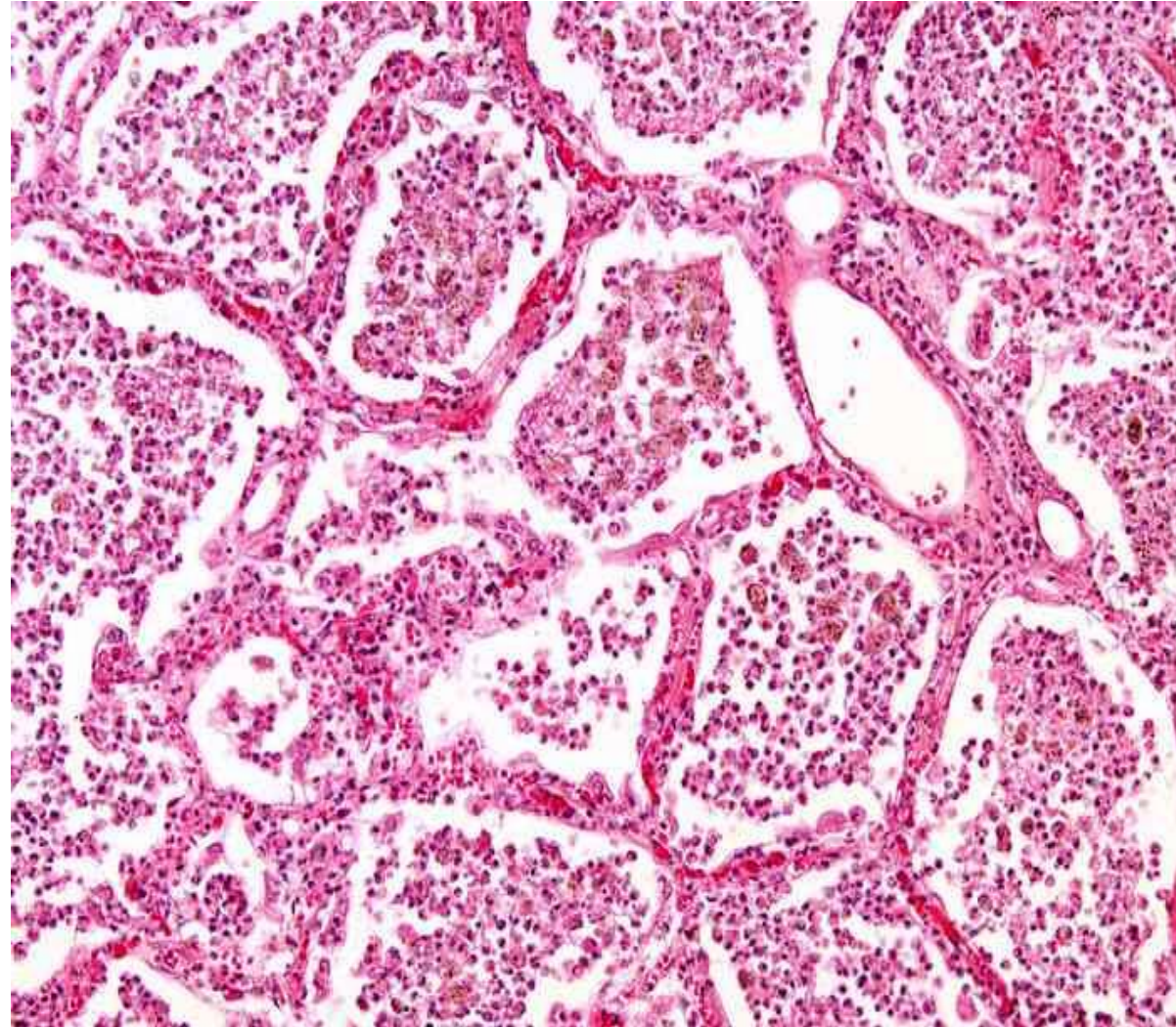
Pathogenesis of Pneumonia

Red Hepatisation

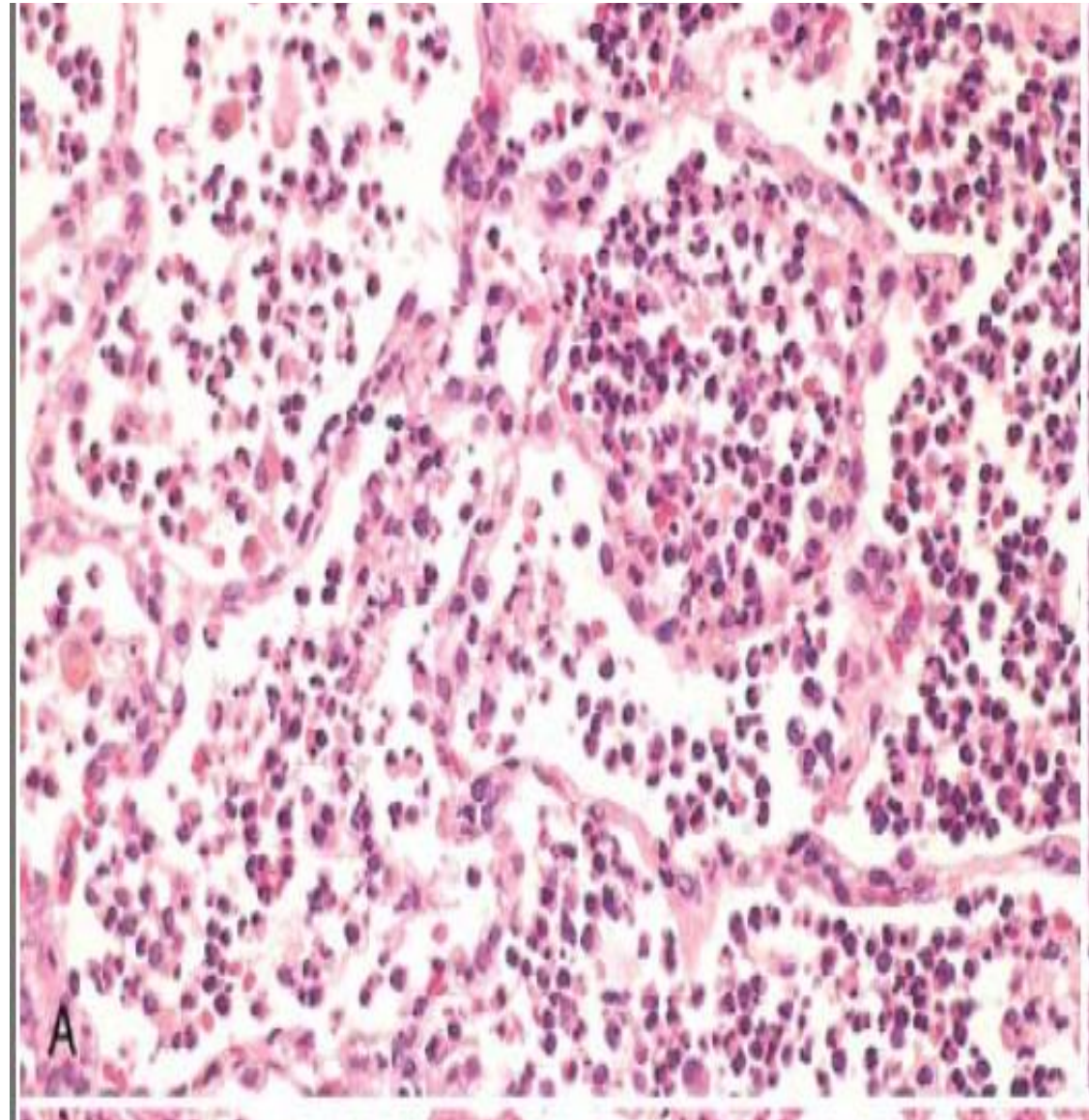
1. Congestion



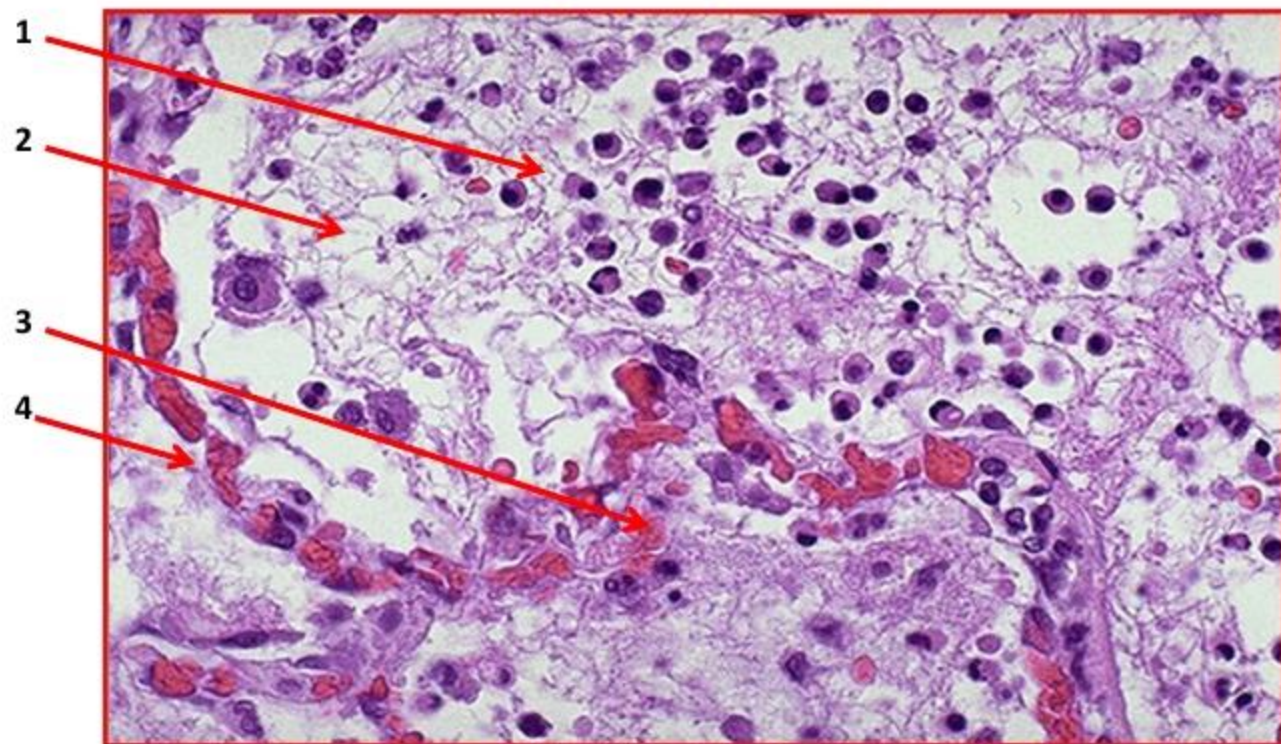
**Congestion  
stage of  
lobar  
pneumonia**



Red  
hepatization



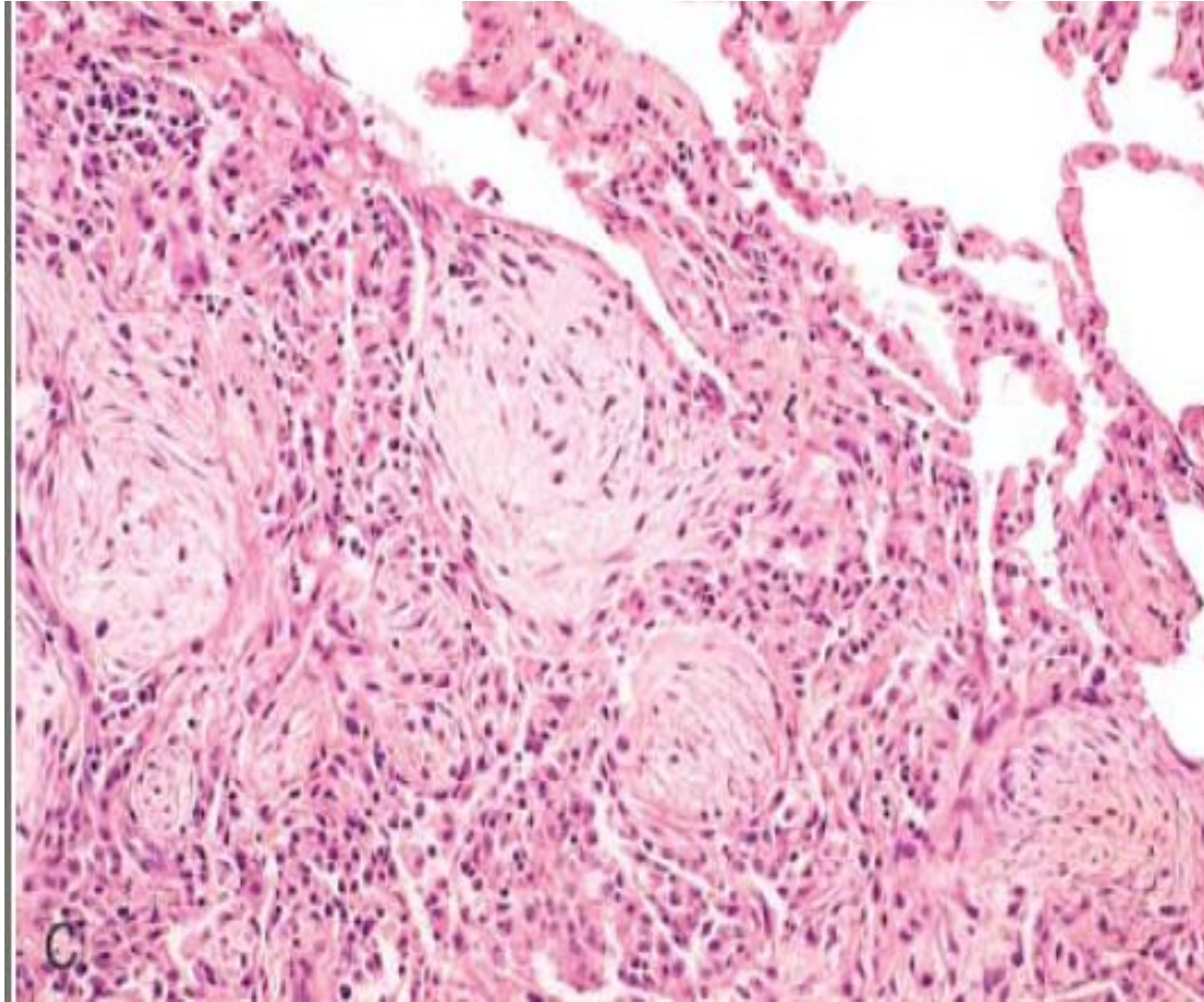
## Lobar Pneumonia, Grey Hepatization



- 1) Inflammatory cells
- 2) Clumps of fibrin and edema
- 3) Fragmented RBCs
- 4) Congested alveolar capillaries



# Grey hepatization



## 2. Bronchpneumonia:

means initial infection in the bronchi & bronchioles with extension into adjacent alveoli.

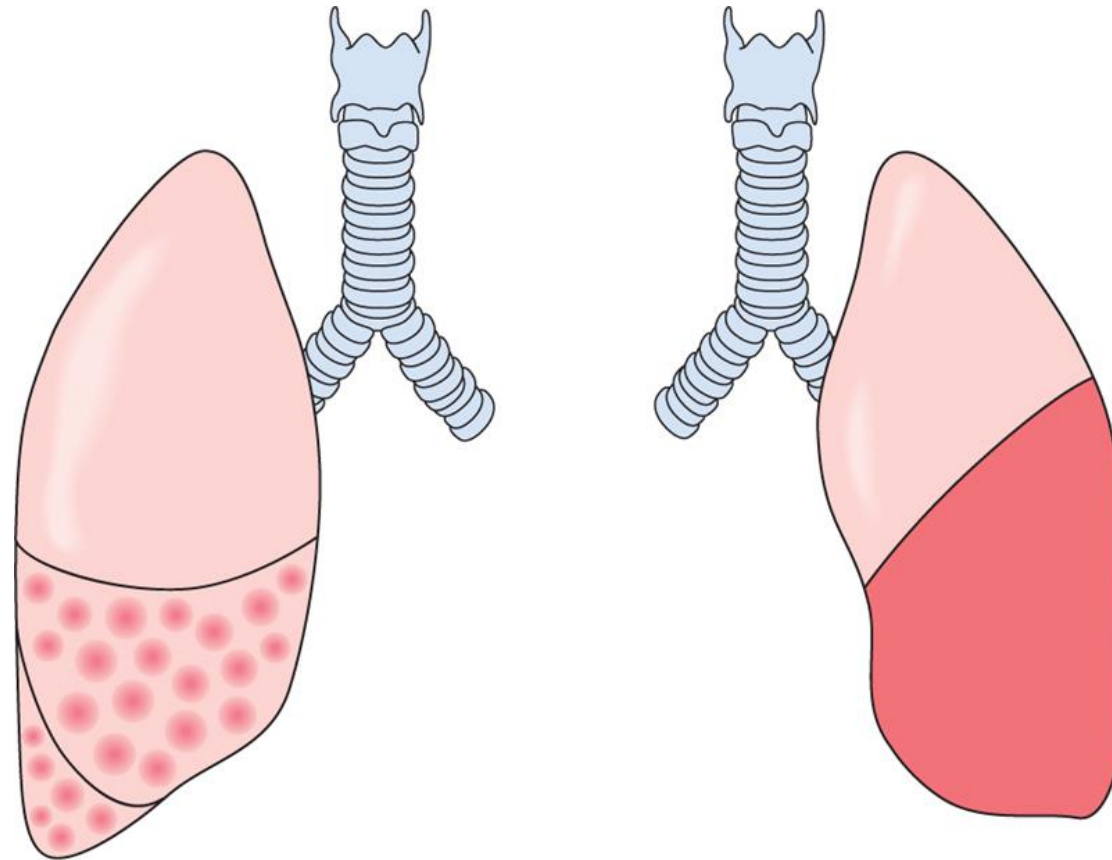
- Caused by staphylococcus, streptococcus, pneumococcus, hemophilus influenzae.
- The consolidation is **patchy** centered around inflamed bronchi that involve more than one **lobe(Multifocal & may be bilateral.)**
- **Predisposing factors:**
  - 1- **Both extreme of age**(in infancy and old age groups caused by low resistance.
  - 2- **Debilitating disease**(Occur Can complicate long term heart failure.)
  3. Pre existing respiratory diseases e.g. chronic bronchitis , emphysema, measles , influenza

- **Grossly**, the lungs exhibit focal areas of palpable consolidation. Lesions are multiple & may be bilateral, affect basal segments of lower lobes.
- **MIC**, there is acute (neutrophilic) suppurative exudation filling bronchi, bronchioles, and the extend to involve the alveoli; this will also eventually resolve.



bronchopneumonia

# Compare Lobar & Bronchopneumonia

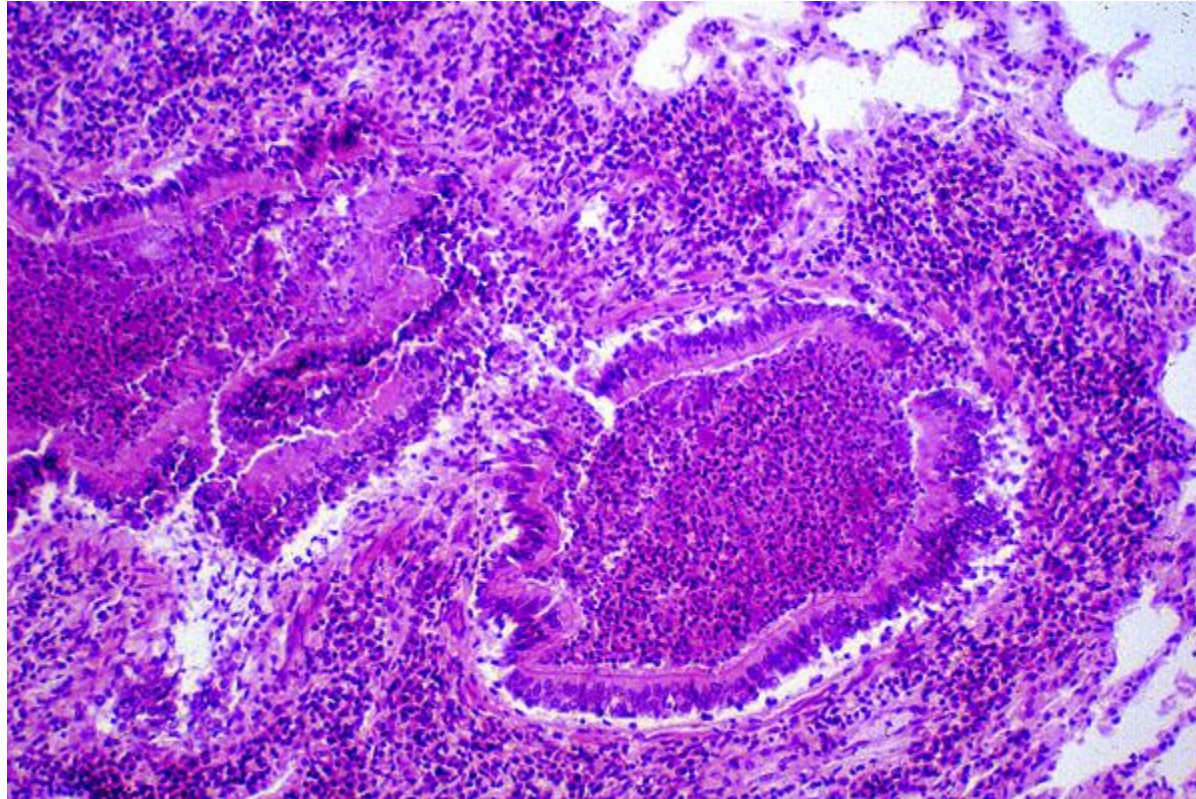


Bronchopneumonia

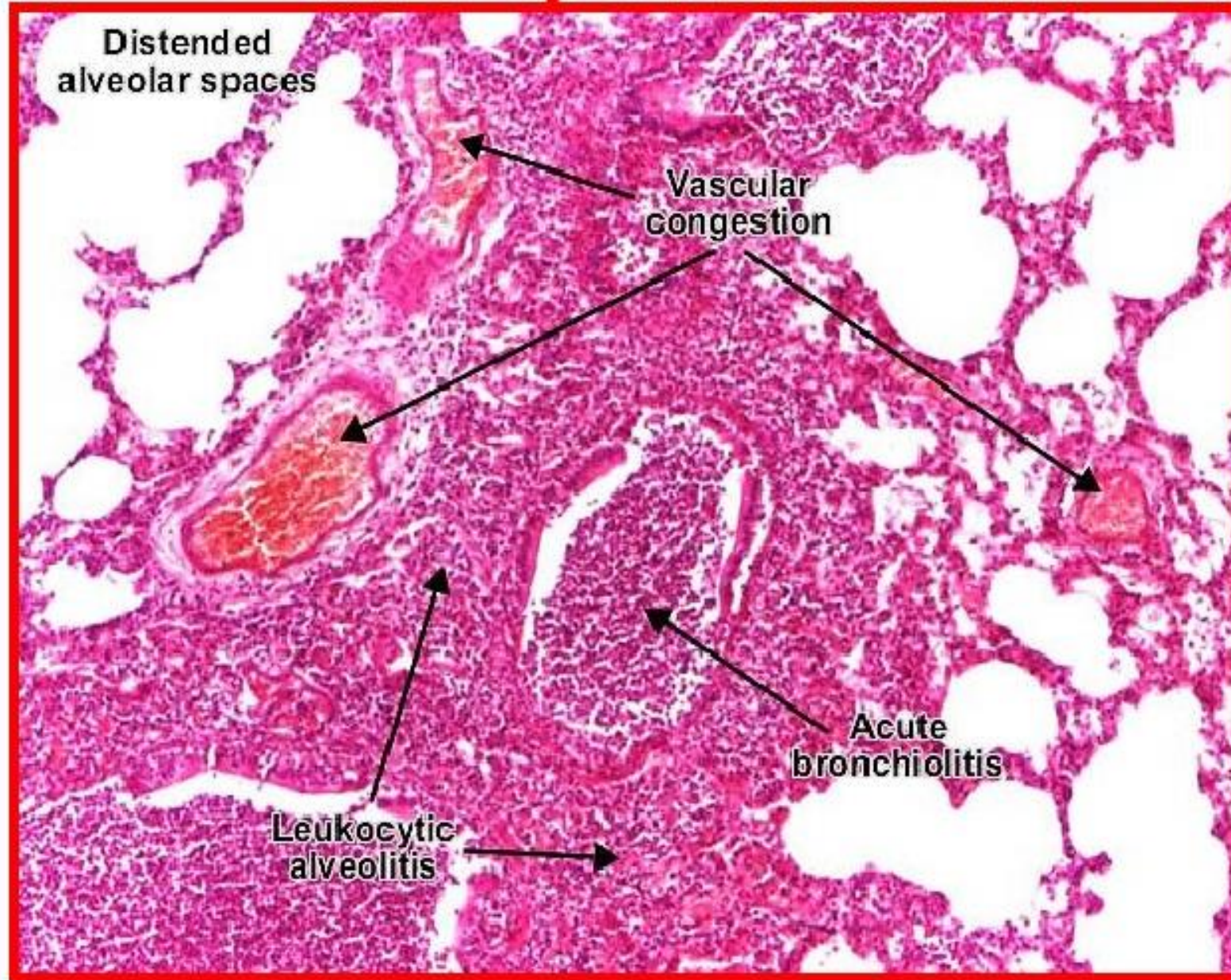
Lobar pneumonia

Kumar et al: Robbins & Cotran Pathologic Basis of Disease, 8th Edition.  
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Histologically, acute bronchopneumonia consists of a suppurative, neutrophil-rich exudate that fills bronchioles and alveolar spaces



# Bronchopneumonia



## Symptoms of pneumonia (in general):

- Abrupt onset of high fever with shaking chills, pleuritic chest pain, cough with mucopurulent (rusty) sputum. Occasionally hemoptysis.



## ***Diagnostic tests:***

- **Sputum examination**: for Gram stain (numerous neutrophils contain diplococci). (nonspecific because due to presence of normal flora)
- **Blood culture**: More specific, 20-30% is positive in early cases.
- **CXR**: The whole lobe is radiopaque in lobar pneumonia, whereas there are focal opacities in bronchopneumonia.
-

## Complications :

90% of cases will end up with resolution, otherwise complication includes:

Abscess formation, specially if the m.o is klebsiella and pneumococcal infection.

Spread of infection to the pleural cavity → empyema (pus inside the pleural cavity).

Organization (fibrosis) of the exudate → part of the lobe will turn solid.

1. Bacteremic dissemination cause meningitis, infective endocarditis, arthritis

