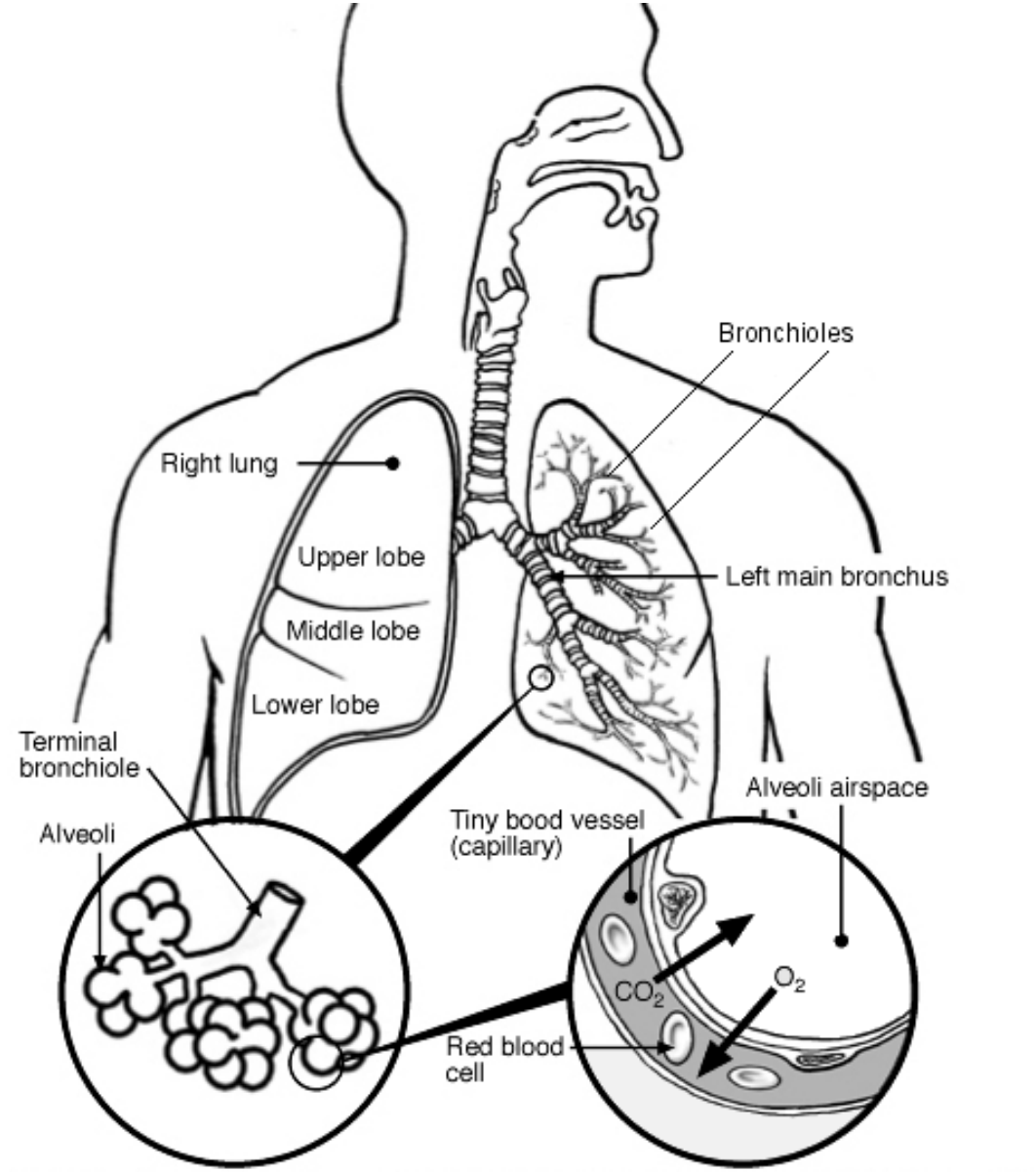
PULMONARY INFECTIONS PNEUMONIAS

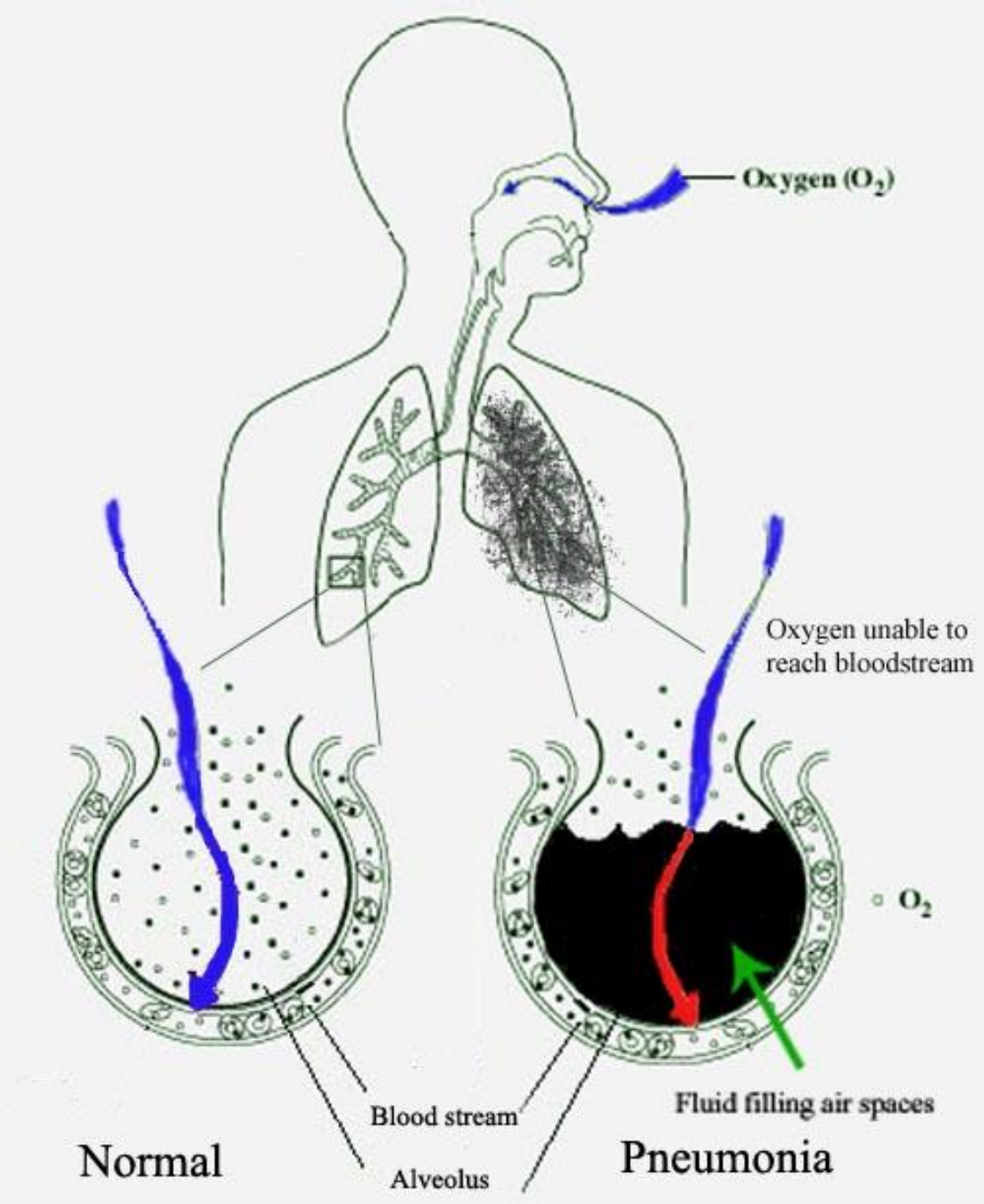
Eman MS Muhammad



The lungs contain millions of tiny alveoli

Oxygen (O_2) from air breathed in, goes into the red blood cells via alveoli. Carbon dioxide (CO_2) goes from the red blood cells into alveoli and breathed out

Lung showing alveoli


Oxygen (O_2)
Oxygen unable to reach bloodstream
O_2
Blood stream
Fluid filling air spaces
Normal
Alveolus
Pneumonia

PULMONARY INFECTIONS

- Respiratory tract infections are more frequent than infections of any other organ and account for the largest number of workdays lost in the general population.
- The vast majority are upper respiratory tract (URT) infections caused by viruses (common cold, pharyngitis), but bacterial, viral, mycoplasmal, and fungal infections of the lung; pneumonia still account for a large incidence of morbidity and mortality worldwide.

- Pneumonia can result whenever pulmonary anti-microbial local defense mechanisms are impaired or the systemic resistance of the host is lowered.
- ***Factors that affect the resistance in general include:***
- Chronic diseases, immunologic deficiency, treatment with immunosuppressive agents, and leukopenia.
- ***The local defense mechanisms of the lung can be compromised by many factors, including:***

1. *Loss or suppression of the cough reflex* as a result of coma, anesthesia, neuromuscular disorders, drugs, or chest pain.

2. *Injury to the mucociliary apparatus* by either impairment of ciliary function or destruction of ciliated epithelium, due to cigarette smoke, inhalation of hot or corrosive gases, viral diseases, or genetic defects of ciliary function (e.g., the immotile cilia syndrome).
3. *Accumulation of secretions* in conditions such as cystic fibrosis and bronchial obstruction.
4. *Interference with the phagocytic or bactericidal action of alveolar macrophages* by alcohol, tobacco smoke, anoxia, or oxygen intoxication.
5. *Pulmonary congestion and edema.*

- Defects in innate immunity including neutrophil and complement defects and humoral immunodeficiency typically lead to an increased incidence of infections with pyogenic bacteria.
- On the other hand, cell-mediated immune defects; congenital or acquired lead to increased infections with intracellular microbes such as mycobacteria and herpes viruses as well as with microorganisms of very low virulence.

- ***Several other points should be emphasized:***
- **First,** the most common cause of death in viral influenza epidemics is superimposed bacterial pneumonia.
- **Second,** although the portal of entry for most bacterial pneumonias is the respiratory tract, hematogenous seeding of the lungs from another organ may occur and may be difficult to distinguish from primary pneumonia.
- **Finally,** many patients with chronic diseases acquire terminal pneumonia while hospitalized (*nosocomial infection*).

PNEUMONIA & PNEUMONITIS

- **Definition:**
- **Pneumonia/pneumonitis** is defined as an inflammation of the lung parenchyma distal to the terminal bronchioles; consisting of the respiratory bronchiole, alveolar ducts, alveolar sacs and alveoli.
- Lung inflammation is more accurately referred to as **pneumonitis**; whereas **pneumonia** refers usually to pneumonitis due to infection but sometimes non-infectious conditions that has the additional feature of **pulmonary consolidation**.

RISK FACTORS

- Risk factors include other lung diseases such as cystic fibrosis, chronic obstructive pulmonary diseases (COPD), and asthma, smoking, poor ability to cough such as following a stroke.
- Conditions and risk factors that predispose to pneumonia include chronic diseases; congestive heart failure, diabetes, chronic kidney disease, and liver disease.
- Other predisposing conditions include extremes of age, congenital or acquired immune deficiencies, and alcoholism.

- The use of acid-suppressing medications such as proton-pump inhibitors or H2 blockers is associated with an increased risk of pneumonia.
- Decreased or absent splenic function (sickle cell disease or postsplenectomy, which puts the patient at risk for infection with encapsulated bacteria such as pneumococcus).

TYPES OF PNEUMONITIS/PNEUMONIA

- **Pneumoniitis are classified according to:**

I. ***Onset and duration:***

A. *Acute pneumonitis/pneumonia*

B. *Chronic pneumonitis/pneumonia*

II. ***Morphological types:***

A. *Lobar pneumonitis/pneumonia*

B. *Lobular/Broncho-pneumonitis/pneumonia*

C. *Interstitial pneumonitis/pneumonia*

III. ***Etiological types:***

A. *Infective pneumonitis/pneumonia*

- Bacterial pneumonia
- Viral pneumonia
- Fungal pneumonia
- Protozoal pneumonia
- ***Tuberculous pneumonia***

B. *Non-infective pneumonitis*

- Toxins-induced *pneumonitis*
- Chemical *pneumonitis*
- Aspiration *pneumonitis*
- Auto-immune disease associated *pneumonitis*

IV. ***Clinical types:***

A. *Primary/ secondary pneumonitis/pneumonia* depending on the mode of infection

B. *Typical/ atypical pneumonitis/pneumonia* depending on the presenting symptoms

C. *Community acquired/ hospital acquired pneumonitis/pneumonia* depending on where it was acquired

D. *Pneumonitis/pneumonia in immuno-compromised patients*

- Currently, combined clinical classification put pneumonia in seven distinct clinical settings **“pneumonia syndromes”.**
- The implicated pathogens are reasonably specific to each category.

PNEUMONIA SYNDROMES

1. **Community-Acquired Acute Pneumonia(CAP):**

- *Streptococcus pneumoniae*
- *Hemophilus influenzae*
- *Moraxella catarrhalis*
- *Staphylococcus aureus*
- *Legionella pneumophila*
- *Enterobacteriaceae (Klebsiella pneumoniae) & Pseudomonas spp.*
- *Mycoplasma pneumoniae*

- *Chlamydia spp. (C. pneumoniae, C. psittaci, C. trachomatis)*
- *Coxiella burnetii (Q fever)*
- Viruses: Respiratory syncytial virus (RSV), para-influenza virus and human metapneumovirus (children); influenza A & B (adults); adenovirus (military recruits)

II. Health Care-Associated Pneumonia

- *Staphylococcus aureus, methicillin-sensitive*
- *Staphylococcus aureus, methicillin-resistant*
- *Pseudomonas aeruginosa*
- *Streptococcus pneumoniae*

III. Hospital-Acquired Pneumonia

- Gram-negative rods, Enterobacteria (*Klebsiella spp., Serratia marcescens, Escherichia coli) and Pseudomonas spp.*
- *Staphylococcus aureus (usually methicillin-resistant)*

IV. Aspiration Pneumonia:

- Anaerobic oral flora (*Bacteroides*, *Prevotella*, *Fusobacterium*,
- *Peptostreptococcus), admixed with aerobic bacteria (Streptococcus pneumoniae, Staphylococcus aureus, Hemophilus influenzae, Pseudomonas aeruginosa).*

V. Chronic Pneumonia

- *Nocardia*
- *Actinomyces*
- Granulomatous: *Mycobacterium tuberculosis & atypical mycobacteria, Histoplasma capsulatum, Coccidioides immitis, Blastomyces dermatitidis*

VI. Necrotizing Pneumonia and Lung Abscess

- Anaerobic bacteria (extremely common), with or without mixed aerobic infection
- *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Streptococcus pyogenes* & type 3 pneumococcus (uncommon)

VII. Pneumonia in Immunocompromised Host

- Cytomegalovirus
- *Pneumocystis jiroveci*
- *Mycobacterium avium-intracellulare*
- Invasive aspergillosis
- Invasive candidiasis
- “Usual” bacterial, viral, and fungal infection

COMMUNITY-ACQUIRED BACTERIAL PNEUMONIAS (CAP)

- Community-acquired acute pneumonia refers to lung infection in otherwise healthy individuals that is acquired from the normal environment.
- It is an infectious pneumonia in a person who has not recently been hospitalized.
- The most common causes of CAP vary depending on a person's age, but they include *Streptococcus pneumonia*, viruses, the atypical bacteria, and *Hemophilus influenza*.

- Overall, ***Streptococcus pneumoniae*** is the most common cause of CAP worldwide.
- Gram-negative bacteria cause CAP in certain at risk populations.
- CAP is the fourth most common cause of death in the United Kingdom and the sixth in the United States.
- The term ***"walking pneumonia"*** has been used to describe a type of CAP of less severity because the sufferer can continue to **"walk"** rather than require hospitalization.
- Walking pneumonia is usually caused by the atypical bacterium, *Mycoplasma pneumoniae*.

PNEUMOCOCCAL PNEUMONIA

- *Streptococcus pneumoniae, or pneumococcus is the most common* cause of community-acquired acute pneumonia.
- Examination of Gram-stained sputum is an important step in its diagnosis.
- The presence of numerous neutrophils containing the typical gram-positive, lancet-shaped diplococci supports the diagnosis of pneumococcal pneumonia, but it must be remembered that *S. pneumoniae is a part of the endogenous flora in 20% of* adults, and therefore false-positive results may be obtained.

- C-reactive protein and procalcitonin, both acute-phase reactants produced primarily in the liver, are significantly elevated in bacterial more than in viral infections.
- Isolation of pneumococci from blood cultures is more specific but less sensitive (in the early phase of illness, only 20% to 30% of patients have positive blood cultures).

HEMOPHILUS INFLUENZAE PNEUMONIA

- *Hemophilus influenza* is a pleomorphic, gram-negative organism that occurs in encapsulated and non-encapsulated forms.
- There are six serotypes of the encapsulated form (types a to f), of which type b is the most virulent.
- Antibodies against the capsule protect the host from *H. influenza infection*.
- With routine use of *H. influenza* vaccines, the incidence of disease caused by the b serotype has declined significantly.

- By contrast, infections with non-encapsulated forms, also called *non-typeable forms, are* increasing.
- They are less virulent, spread along the surface of the URT, and produce otitis media, sinusitis, and bronchopneumonia.
- *H. influenzae pneumonia, which may follow a viral* respiratory infection, is a pediatric emergency and has high mortality rate.
- Descending laryngo-tracheo-bronchitis results in airway obstruction as the smaller bronchi are plugged by dense, fibrin-rich exudates containing neutrophils, similar to that seen in pneumococcal pneumonias.

- Pulmonary consolidation is usually lobular and patchy and may be confluent and involve the entire lung lobe.
- *H. influenza was a* common cause of suppurative meningitis in children up to 5 years of age.
- *H. influenza also causes an acute purulent* conjunctivitis in children and, in predisposed older patients, and may cause septicemia, endocarditis, pyelonephritis, cholecystitis, and suppurative arthritis.
- *H. influenza* is the most common bacterial cause of acute exacerbation of COPD.

MORAXELLA CATARRHALIS PNEUMONIA

- *Moraxella catarrhalis* is being increasingly recognized as a cause of bacterial pneumonia, especially in the elderly.
- It is the second most common bacterial cause of acute exacerbation of COPD.
- Along with *S. pneumonia and H. influenza*, *M. catarrhalis* constitutes one of the three most common causes of otitis media in children.

STAPHYLOCOCCUS AUREUS PNEUMONIA

- *Staphylococcus aureus* is an important cause of ***secondary*** bacterial pneumonia in children and healthy adults following viral respiratory illnesses (e.g., measles in children and influenza in both children and adults).
- Staphylococcal pneumonia is associated with a high incidence of complications, such as lung abscess and empyema.
- *Intravenous drug users* are at high risk for development of staphylococcal pneumonia in association with endocarditis.
- It is also an important cause of hospital-acquired pneumonia.

KLEBSIELLA PNEUMONIAE PNEUMONIA

- *Klebsiella pneumoniae* is the most frequent cause of gram negative bacterial pneumonia.
- It commonly affects debilitated and malnourished people, particularly chronic alcoholics.
- Thick, mucoid, often blood-tinged sputum is characteristic, because the organism produces an abundant viscid capsular polysaccharide, which the patient may have difficulty expectorating.

PSEUDOMONAS AERUGINOSA PNEUMONIA

- *Pseudomonas aeruginosa* most commonly causes hospital-acquired infections, but it may occur in cystic fibrosis and in immunocompromised patients as a community acquired pneumonia.
- It is common in patients who are neutropenic and it has a propensity to invade blood vessels with consequent extrapulmonary spread.
- *Pseudomonas* septicemia is a very fulminant disease.

LEGIONELLA PNEUMOPHILA PNEUMONIA

- *Legionella pneumophila* is the agent of *Legionnaires' disease*, an epidemic and sporadic forms of pneumonia caused by this organism.
- This organism flourishes in artificial aquatic environments, such as water-cooling towers and the tubing systems of domestic water supplies.
- The mode of transmission is either inhalation of aerosolized organisms or aspiration of contaminated drinking water.

- *Legionella* pneumonia is common in individuals with predisposing conditions such as cardiac, renal, immunologic, or hematologic disease.
- Organ transplant recipients are particularly susceptible.
- It can be severe, requiring hospitalization, and immunosuppressed patients may have fatality rates of up to 50%.
- Rapid diagnosis is made by demonstration of *Legionella antigens in urine* or by positive fluorescent antibody test on sputum samples; culture remains the diagnostic gold standard.

MORPHOLOGY OF BACTERIAL PNEUMONIA

- Bacterial invasion of the lung parenchyma causes the alveoli to be filled with an inflammatory exudate, thus causing solidification "*consolidation*" of the pulmonary tissue.
- *Consolidation:* The term used for gross and radiologic appearance of the lungs in pneumonia.
- **Bacterial pneumonia has two patterns of anatomic distribution:**
- Lobular bronchopneumonia and lobar pneumonia.
- Patchy consolidation of the lung is the dominant characteristic of ***bronchopneumonia***, while consolidation of a large portion of a lobe or of an entire lobe defines ***lobar pneumonia***.

- These anatomic categorizations may be difficult to apply in individual cases because patterns overlap.
- The patchy involvement may become confluent, producing total lobar consolidation; however, effective antibiotic therapy may limit involvement to a subtotal consolidation.
- Moreover, the same organisms may produce either pattern depending on the patient susceptibility.
- **Most important from the clinical standpoint are identification of the causative agent and determination of the extent of disease.**

CLINICAL COURSE OF BACTERIAL PNEUMONIA

- The major symptoms of community acquired acute bacterial pneumonia are abrupt onset of high fever, shaking chills, and cough producing muco-purulent sputum; occasional patients may have hemoptysis.
- When pleuritis is present it is accompanied by pleuritic pain and pleural friction rub.
- The whole lobe is radiopaque in lobar pneumonia, whereas there are focal opacities in bronchopneumonia.
- The clinical picture is markedly modified by the administration of antibiotics.

- Treated patients may be relatively afebrile with few clinical signs 48 to 72 hours after initiation of antibiotics.
- The identification of the organism and the determination of its antibiotic sensitivity are the keystones to appropriate therapy.
- Fewer than 10% of patients with pneumonia is severe enough to need hospitalization.
- Death results from complications, such as empyema, meningitis, endocarditis, or pericarditis, or to some predisposing influence, such as debility or chronic alcoholism.

COMPLICATIONS OF BACTERIAL PNEUMONIA

- **Complications include:**

1. **Tissue destruction and necrosis**, causing **abscess** formation (particularly common with type 3 pneumococci or *Klebsiella* infections);
2. **Spread of infection** to the pleural cavity, causing the intrapleural fibrinosuppurative reaction known as **empyema**; and
3. **Bacteremic dissemination** to the heart valves, pericardium, brain, kidneys, spleen, or joints, causing metastatic abscesses, endocarditis, meningitis, or suppurative arthritis.

LOBAR PNEUMONIA

- **Definition:**
- Lobar pneumonia is an acute bacterial infection of a part of a lobe, the entire lobe, or even two lobes of one or both the lungs.

ETIOLOGY OF LOBAR PNEUMONIA

- Based on the etiological microbial agent types of lobar pneumonia are:

1. **Pneumococcal pneumonia:**

- More than 90% of all lobar pneumonias are caused by *Streptococcus pneumoniae*, a lancet-shaped diplococcus.
- Out of various types, type *3-S. pneumoniae causes particularly virulent form of lobar* pneumonia.
- Pneumococcal pneumonia in the majority of cases is community-acquired infection.

2. **Staphylococcal pneumonia:**

- Staphylococcus aureus causes pneumonia by hematogenous spread of infection from another focus or after viral infections.

3. **Streptococcal pneumonia:**

- Β-hemolytic streptococci may rarely cause pneumonia such as in children after measles or influenza, in severely debilitated elderly patients and in diabetics.

4. **Pneumonia by gram-negative aerobic bacteria.**

- Less common causes of lobar pneumonia are gram-negative bacteria like *Hemophilus influenzae*, *Klebsiella pneumoniae (Friedlander's bacillus)*, *Pseudomonas*, *Proteus and Escherichia coli*, *H. influenzae commonly causes pneumonia in children* below 3 years of age after a preceding viral infection.

MORPHOLOGY OF LOBAR PNEUMONIA

- In **lobar pneumonia**, ***four stages of the inflammatory response*** have classically been described:

1. **Stage of congestion (initial phase):**

- The initial phase corresponds to the onset of the disease.
- It represents the early acute inflammatory response to bacterial infection and lasts for 1 to 2 days.
- **Clinically:**
- It is characterized by high grade fever, coughing rusty sputum.

- **Grossly:**
- The affected lobe is enlarged, heavy, and dark red.
- The consistency is wet sponge, and the cut surface exudes frothy blood-stained serous exudate.

- **Microscopic:**
- The typical features of acute inflammatory response to the organisms are seen.
- **It is characterized by:**

1. Dilatation and congestion of the capillaries in the alveolar walls.
2. Pale eosinophilic edema fluid in the air spaces.
3. A few red cells and neutrophils in the intra-alveolar fluid.
4. Numerous bacteria demonstrated in the alveolar fluid by Gram's staining.

2. **Stage of red hepatization (early consolidation):**

- This phase lasts for 2 to 4 days.
- The term hepatisation in pneumonia refers to liver-like consistency of the affected lobe on cut section.
- ***Gross examination:***
- The lobe is red, firm, and airless (consolidated),with liver-like consistency.
- The cut surface is dark red, dry, and granular.
- The bronchi contain purulent exudate.
- The pleura show serofibrinous pleurisy.
- The hilar lymph nodes are enlarged.

- **Microscopic picture:**

1. The edema fluid of the preceding stage is replaced by strands of fibrin.
2. There is marked cellular exudate of neutrophils and extravasation of red cells.
3. Many neutrophils show ingested bacteria.
4. The alveolar septa are less prominent than in the first stage due to cellular exudation.
5. The increased consolidation is due to continuous exudation and neutrophils emigration.

3. **Stage of gray hepatization (late consolidation):**

- This phase lasts for 4 to 8 days.
- It is marked by progressive disintegration of red cells and the persistence of a fibrino-suppurative exudate, resulting in a color change to grayish-brown.

- ***Grossly:***
- The affected lobe is firm and heavy.
- The ***cut surface*** is dry, granular and grey in appearance with liver-like consistency.
- The change in color from red to grayish brown begins at the hilum and spreads towards the periphery.
- Fibrinous pleurisy is prominent.

- ***Histologically:***

1. There is less marked thickening of the alveolar wall and less capillary congestion.
2. The fibrin strands are dense, more numerous and clumped in the center of the alveoli.
3. The cellular exudate of neutrophils is reduced due to disintegration of many inflammatory cells as evidenced by their pyknotic nuclei.
4. The red cells are also fewer.
5. The macrophages begin to appear in the exudate.

5. The cellular exudate is often separated from the septal walls by a thin clear space.
6. The organisms are less numerous and appear as degenerated forms.

4. **Stage of resolution (final stage):**

- This stage begins by 8th to 9th day if no treatment is administered and is completed in 1 to 3 weeks.
- However, antibiotic therapy induces resolution on about 3rd day.
- Resolution proceeds in a progressive manner.

- ***Grossly:***
- The exudate within the alveolar spaces is broken down by enzymatic digestion to produce granular, semifluid debris that is resorbed, ingested by macrophages, expectorated, or organized by fibroblasts growing into it, eventually restoring the normal aeration in the affected lobe.
- The process of softening begins centrally and spreads to the periphery.
- The ***cut surface*** is grey-red or dirty brown and frothy, yellow, creamy fluid can be expressed on pressing.
- The pleural fibrinous reaction **(pleuritis)** may also show resolution but may undergo organization leading to fibrous thickening or permanent adhesions.

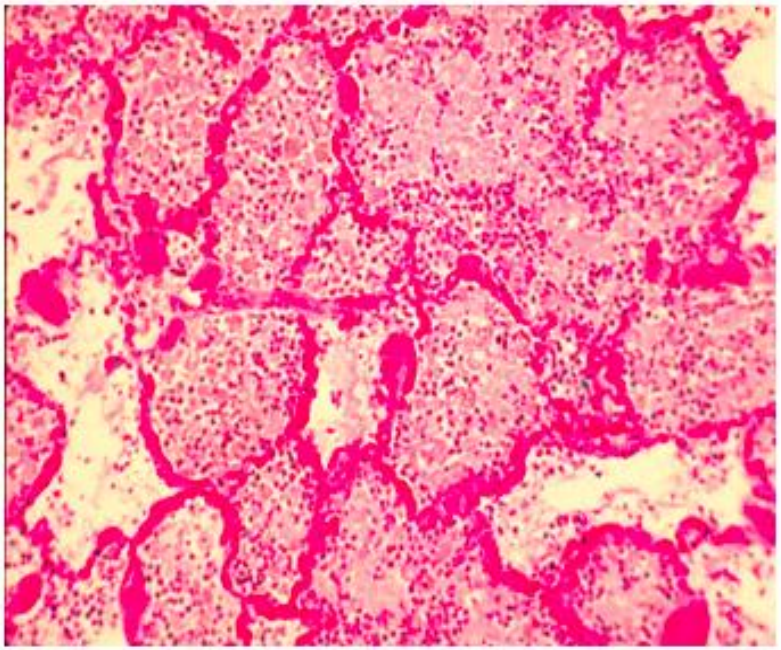
- ***Histologically:***

1. Macrophages are the predominant cells in the alveolar spaces, while neutrophils diminish in number.
2. Many of the macrophages contain engulfed neutrophils and debris.
3. Granular and fragmented strands of fibrin in the alveolar spaces are seen due to progressive enzymatic digestion.
4. Alveolar capillaries are engorged.
5. There is progressive removal of fluid content as well as cellular exudate from the air spaces, partly by expectoration but mainly by lymphatics, resulting in restoration of normal lung parenchyma with aeration.

THE FOUR STAGES OF LOBAR PNEUMONIA, SHOWING CORRELATION OF GROSS APPEARANCE OF THE LUNG WITH MICROSCOPIC APPEARANCE IN EACH STAGE.

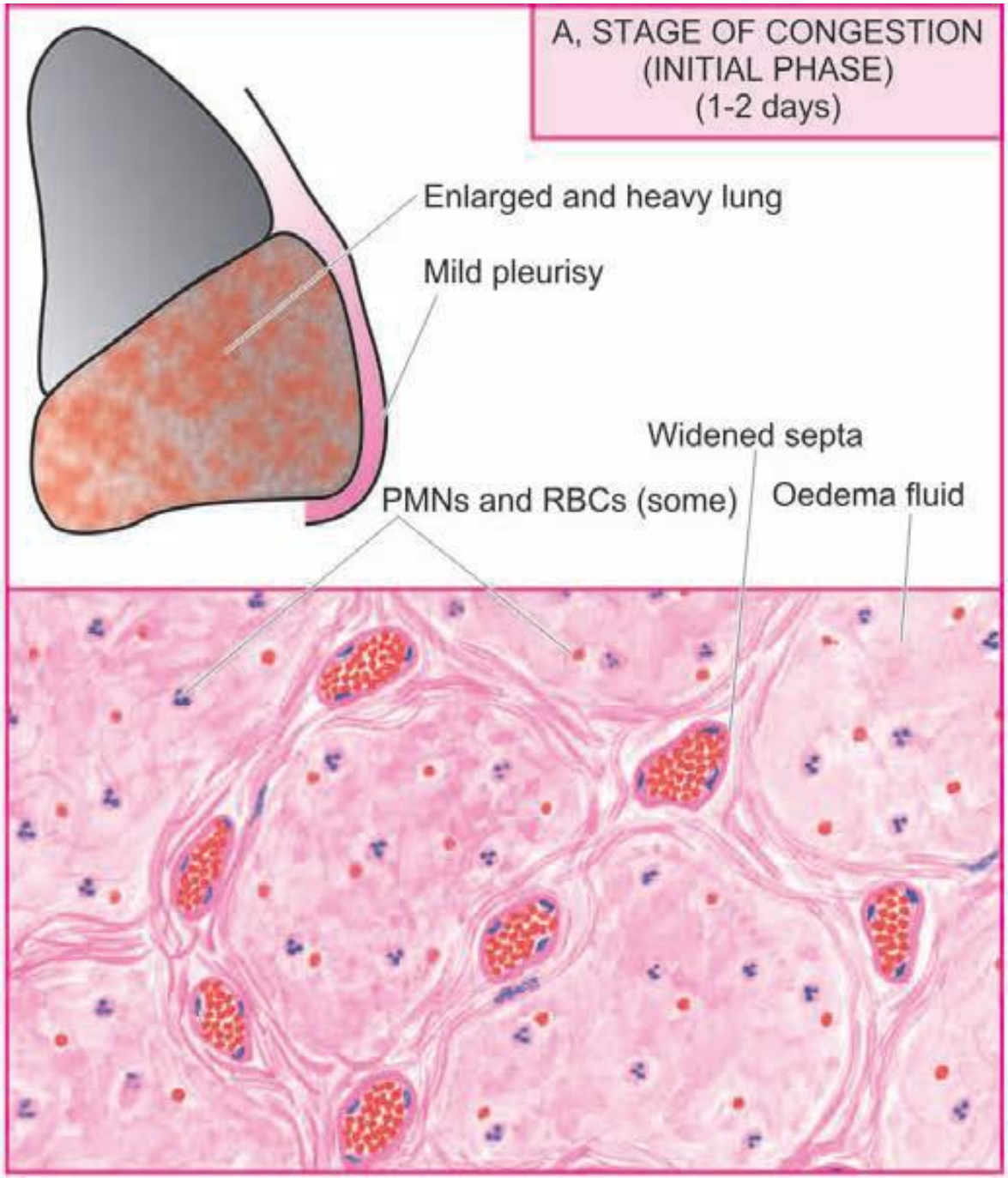
Lobar Pneumonia- Microscopic

Congestion

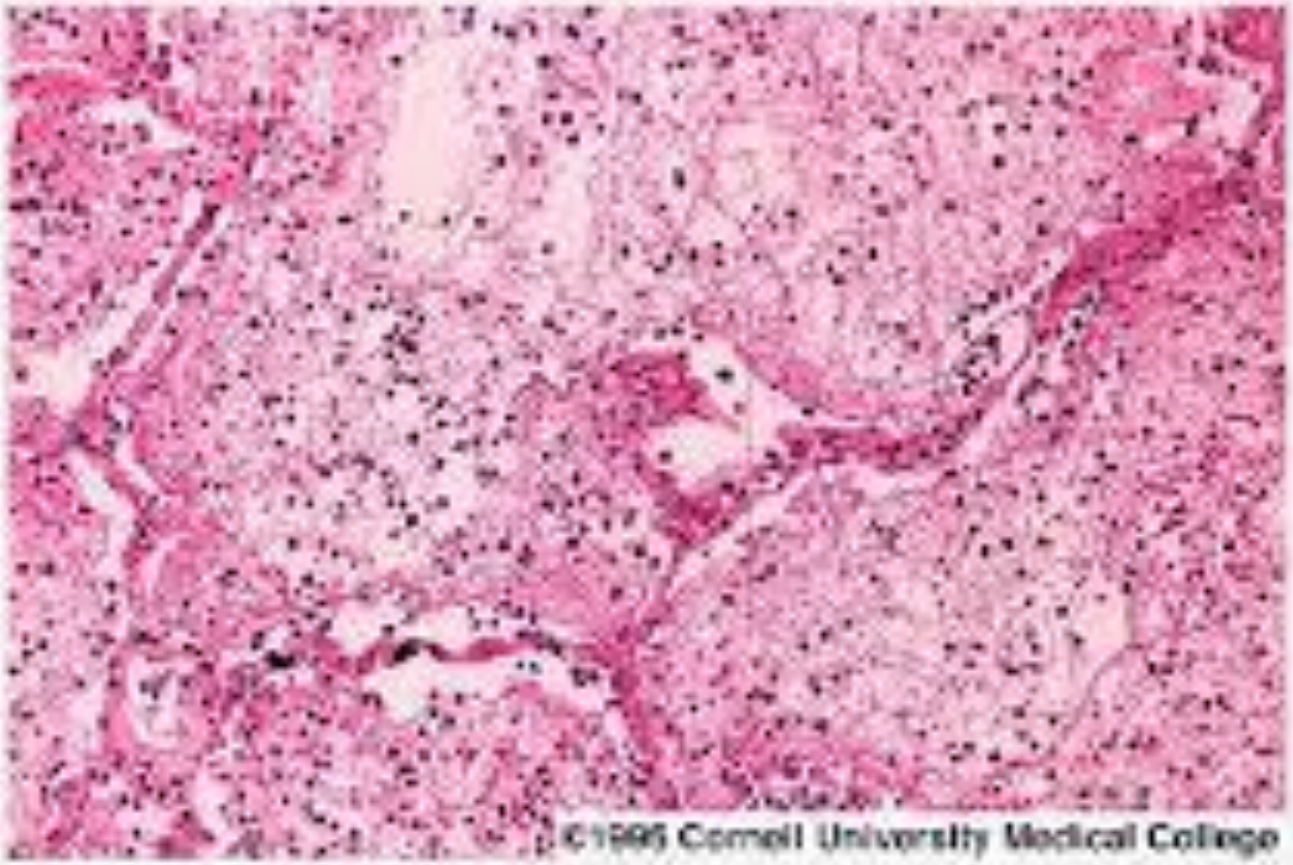


Red Hepatization

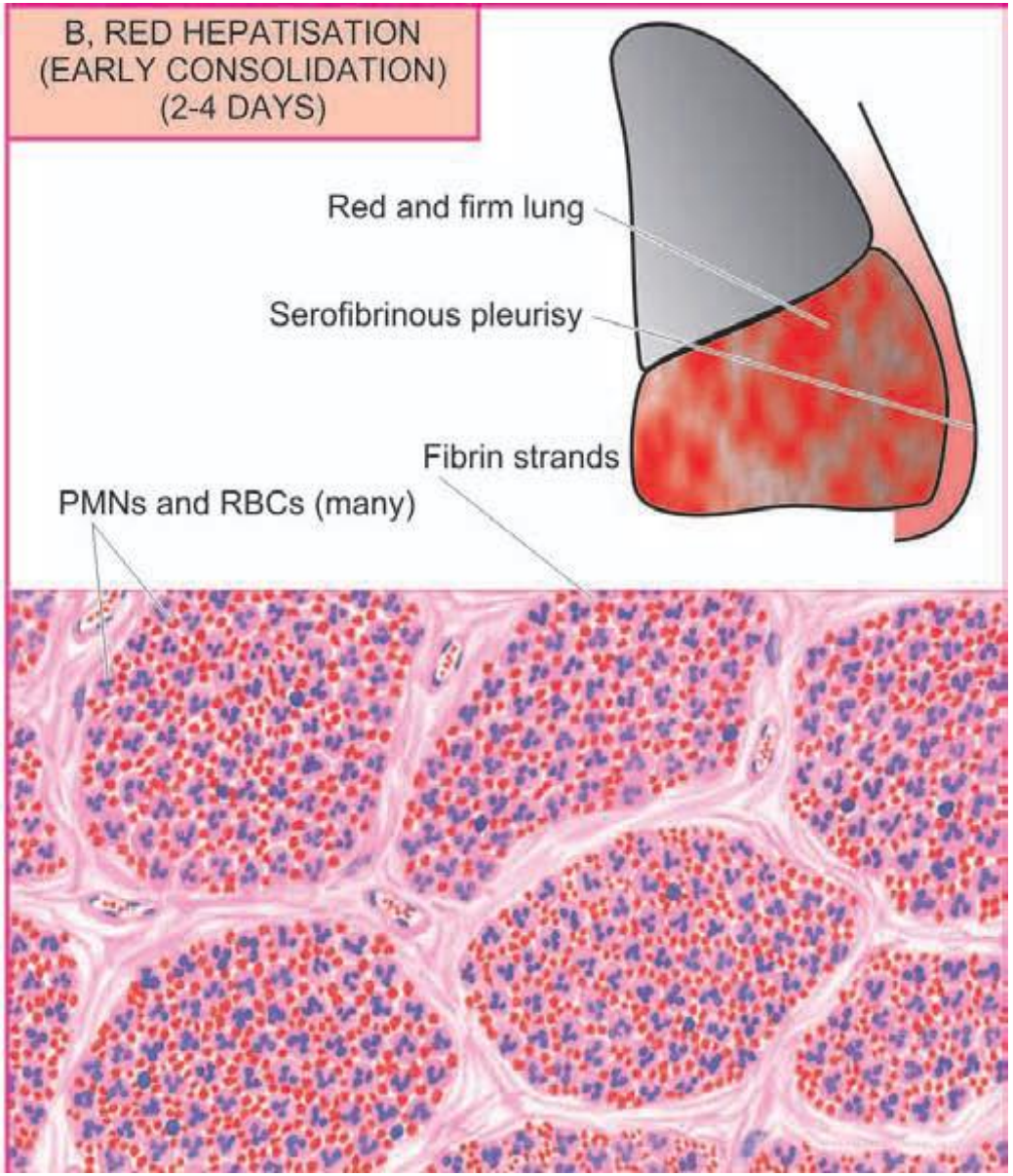
STAGE OF CONGESTION



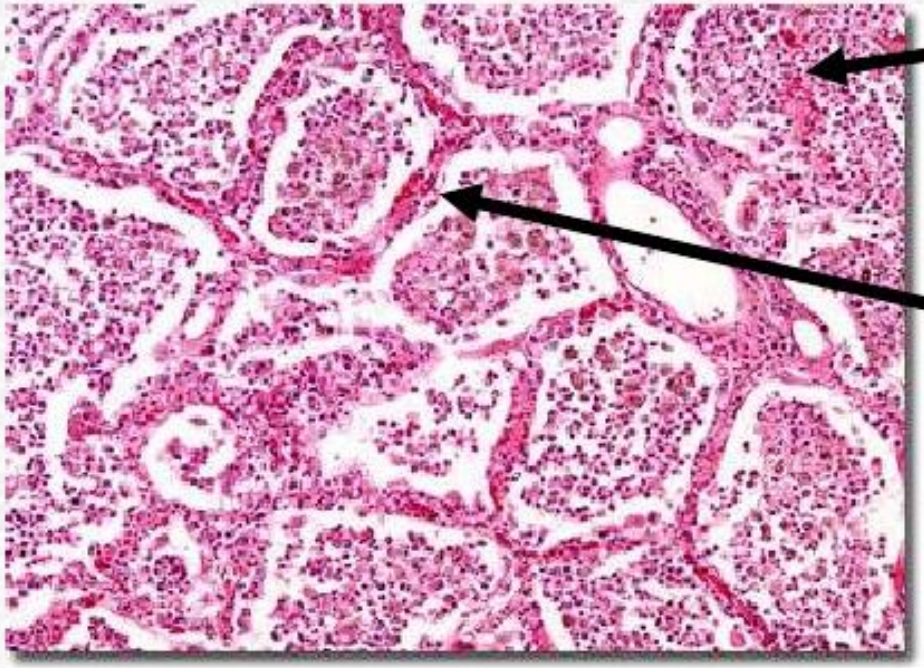
Lobar Pneumonia: Microscopy: Congestion → red hepatization



STAGE OF RED HEPATISATION

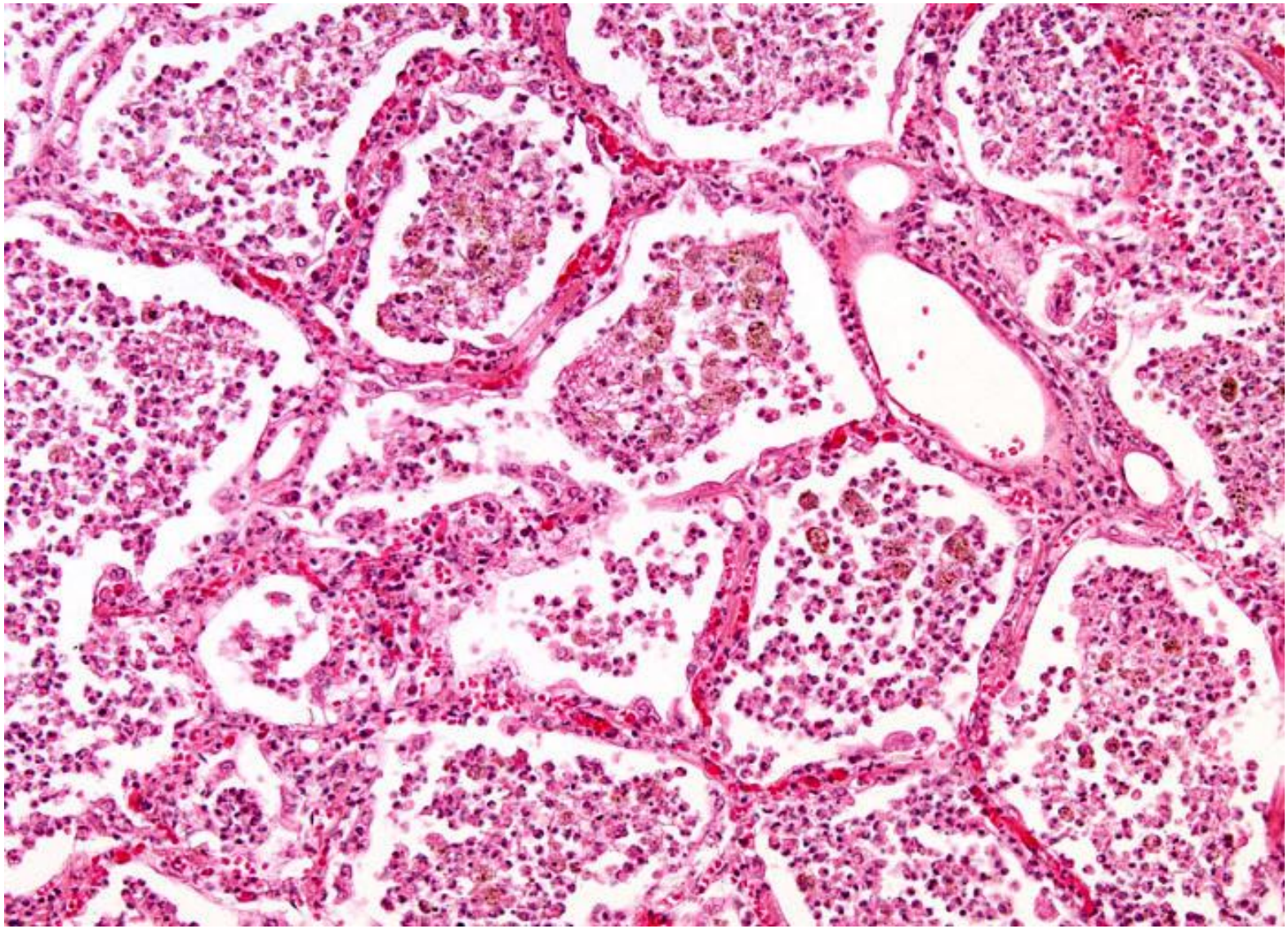


Lobar Pneumonia: Red hepatization.

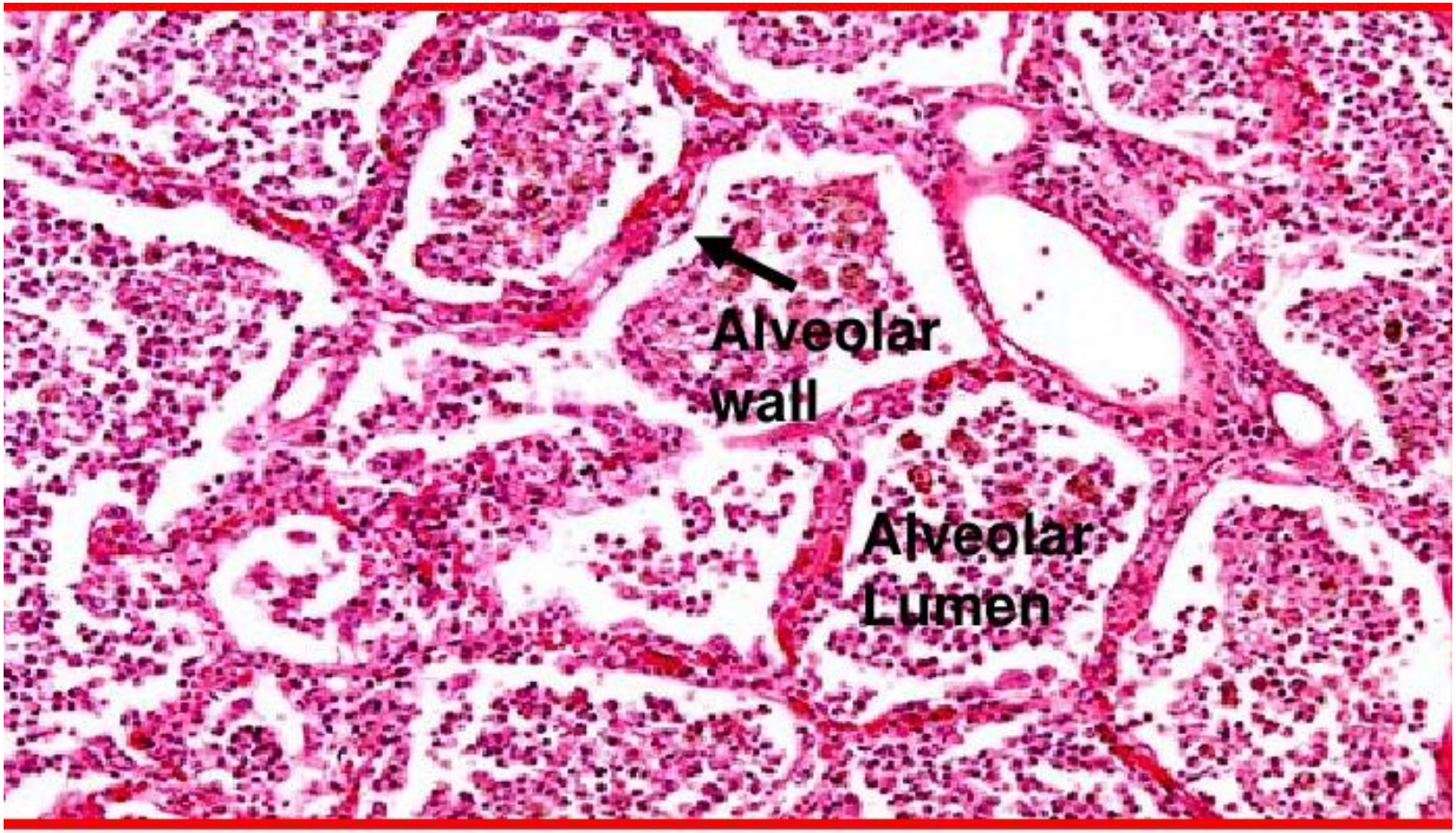


- Acute inflammatory cells & RBC Filling alveolar spaces Uniformly.
- Congested capillaries in the alveolar septa

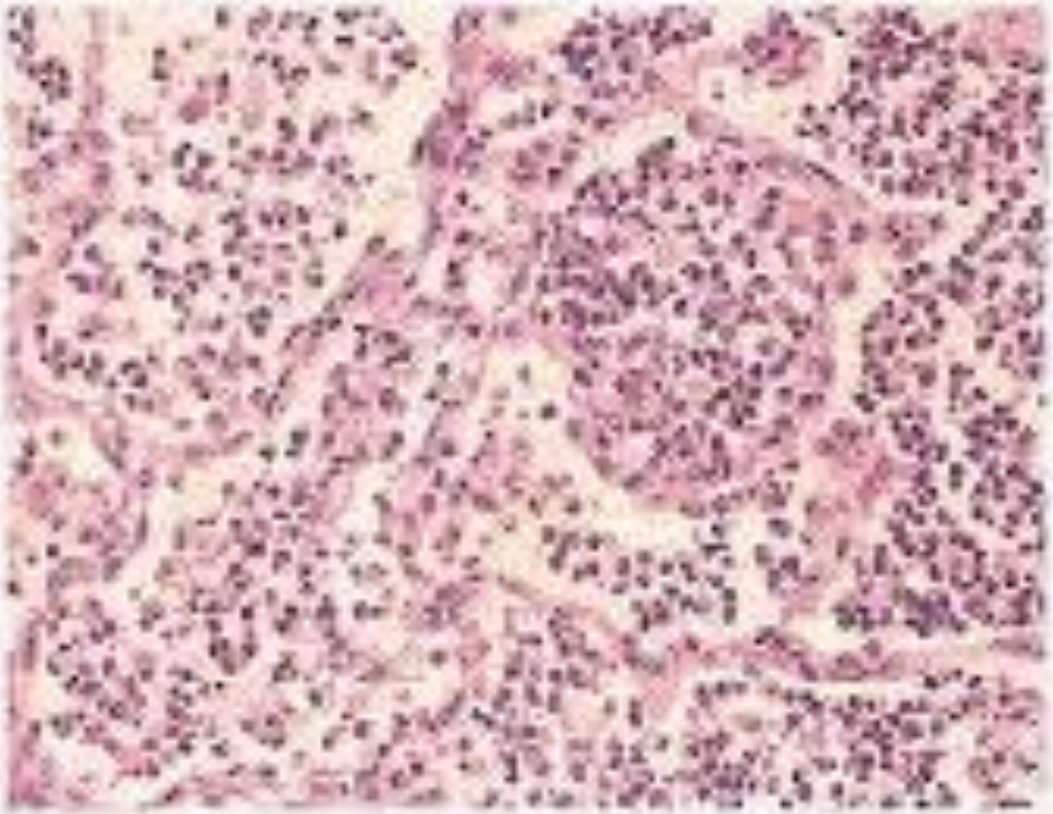
Red hepatisation



Red hepatisation

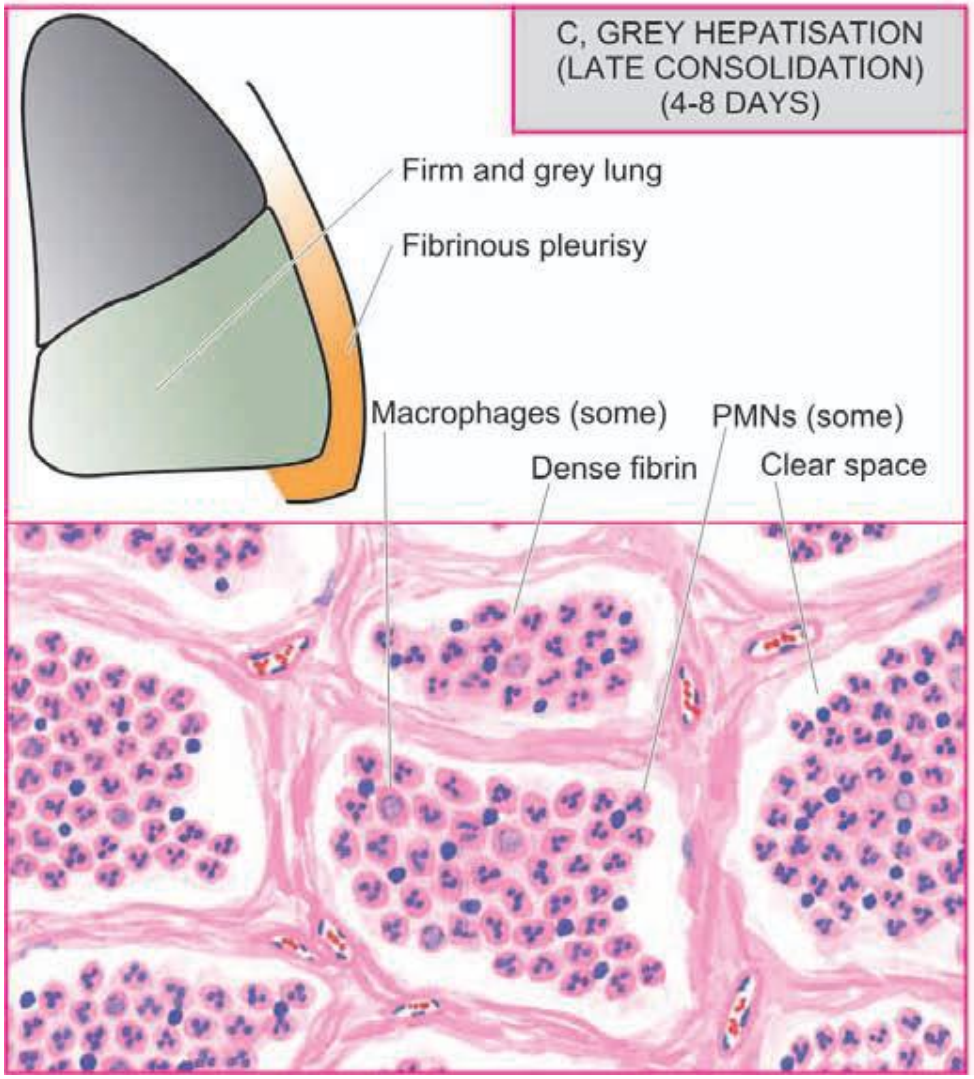


Lobar Pneumonia: Red → Grey hepatization.

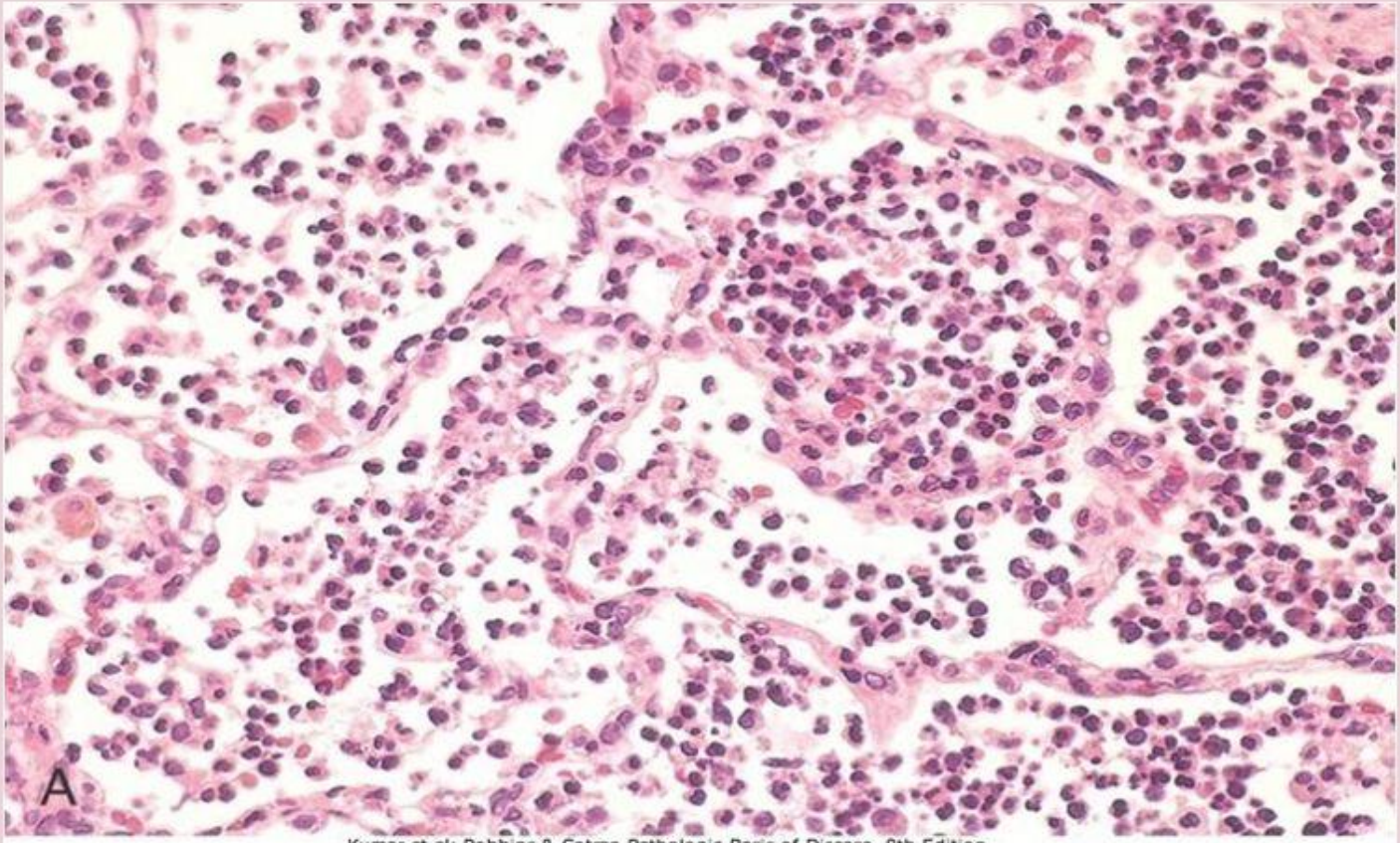


Lobar pneumonia: Gray hepatization.
The lower lobe is uniformly consolidated.

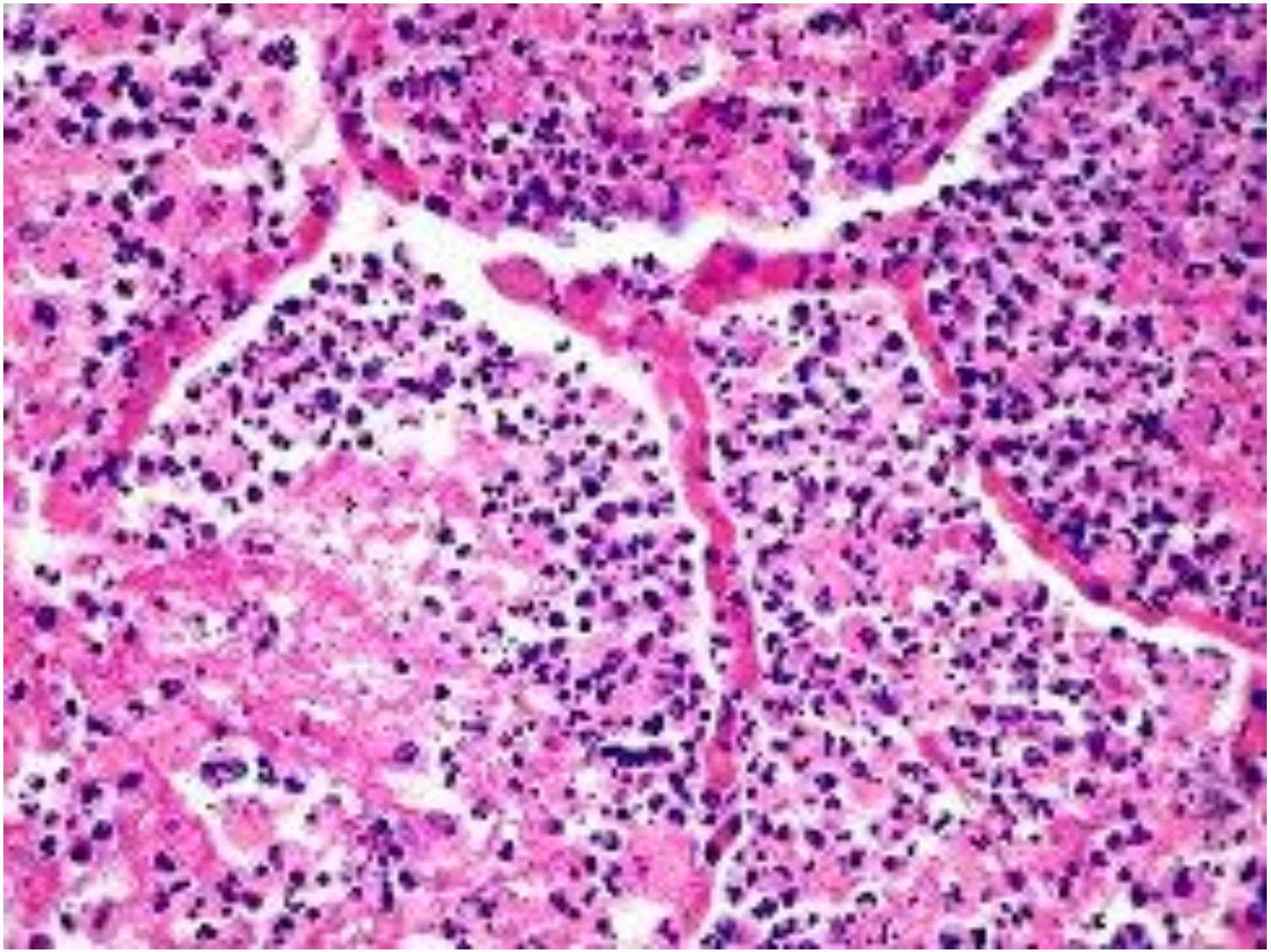
STAGE OF GREY HEPATISATION



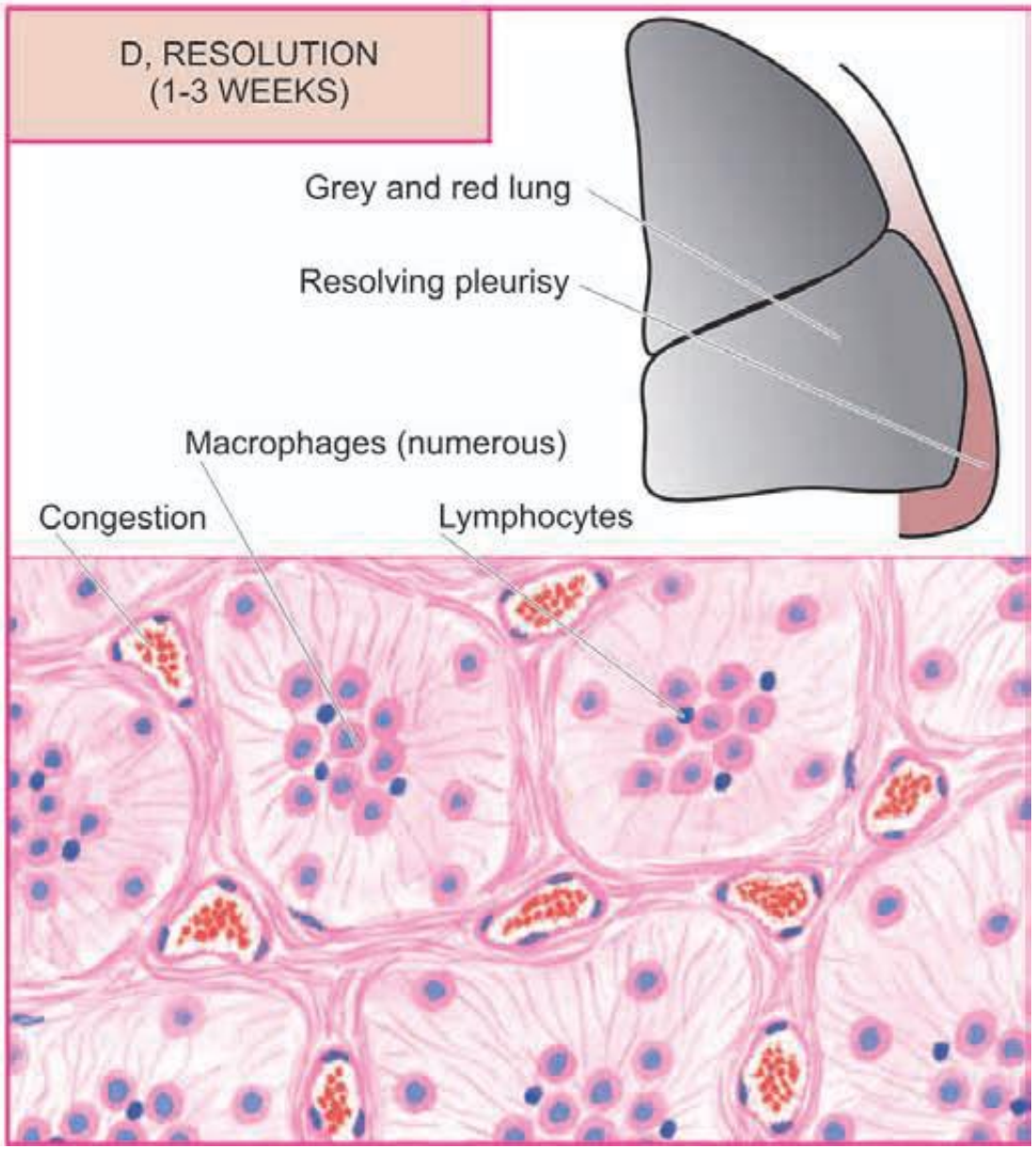
Lobar Pneumonia: Gray Hepatization



Grey hepatisation



STAGE OF RESOLUTION



CLINICAL FEATURES OF LOBAR PNEUMONIA

- Classically, the onset of lobar pneumonia is sudden.
- The major symptoms are: shaking chills, fever, malaise with pleuritic chest pain, dyspnea and cough with expectoration of mucoid, purulent or even bloody sputum.
- The common physical findings are fever, tachycardia, and tachypnea, and sometimes cyanosis if the patient is severely hypoxemic.

- There is generally marked neutrophil leucocytosis.
- Blood cultures are positive in about 30% of cases.
- Chest radiograph may reveal consolidation.
- Culture of the organisms in the sputum and antibiotic sensitivity are the most significant investigations for institution of specific antibiotics.
- The response to antibiotics is usually rapid with clinical improvement in 48 to 72 hours after the initiation of antibiotics.

COMPLICATIONS OF LOBAR PNEUMONIA

- Since the advent of antibiotics, serious complications of lobar pneumonia are uncommon.
- However, they may develop in neglected cases and in patients with impaired immunologic defenses.

- **These are:**

1. **Organization:**

- In about 3% of cases, resolution of the exudate does not occur but instead it undergoes organization.
- There is ingrowth of fibroblasts from the alveolar septa resulting in fibrosed, tough, airless leathery lung tissue.
- This type of post-pneumonic fibrosis is called ***carnification***.

2. **Pleural effusion:**

- About 5% of treated cases of lobar pneumonia develop inflammation of the pleura with effusion.
- The pleural effusion usually resolves but sometimes may undergo organization with fibrous adhesions between visceral and parietal pleura.

3. **Empyema:**

- Less than 1% of treated cases of lobar pneumonia develop encysted pus in the pleural cavity termed empyema.

4. **Lung abscess:**

- A rare complication of lobar pneumonia is formation of lung abscess, especially when there is secondary infection by other organisms.

5. **Bacteremic dissemination:**

- Occasionally, infection in the lungs and pleural cavity in lobar pneumonia may extend into the pericardium and the heart causing purulent pericarditis, bacterial endocarditis and myocarditis.
- Other forms of metastatic infection encountered rarely in lobar pneumonias are otitis media, mastoiditis, meningitis, brain abscess and purulent arthritis.

BRONCHOPNEUMONIA (LOBULAR PNEUMONIA)

- **Definition:**
- Bronchopneumonia or lobular pneumonia is infection of the terminal bronchioles that extends into the surrounding alveoli resulting in ***patchy consolidation of the lung***.
- The condition is particularly frequent at the *extremes of life* as a terminal event in chronic debilitating diseases and as a **secondary** infection following viral respiratory infections such as influenza, measles etc…..

ETIOLOGY OF LOBULAR PNEUMONIA

- The most common organisms responsible for bronchopneumonia are staphylococci, streptococci, pneumococci, *Klebsiella pneumoniae*, *Hemophilus influenzae*, and gram-negative bacilli like *Pseudomonas and coliform* bacteria.

MORPHOLOGY OF LOBULAR PNEUMONIA

- ***Grossly:***
- Bronchopneumonia is identified by *patchy areas* of red or grey consolidation affecting *one or more lobes*, frequently found *bilaterally* and more often involving the *lower zones* of the lungs due to *gravitation of the secretions*.

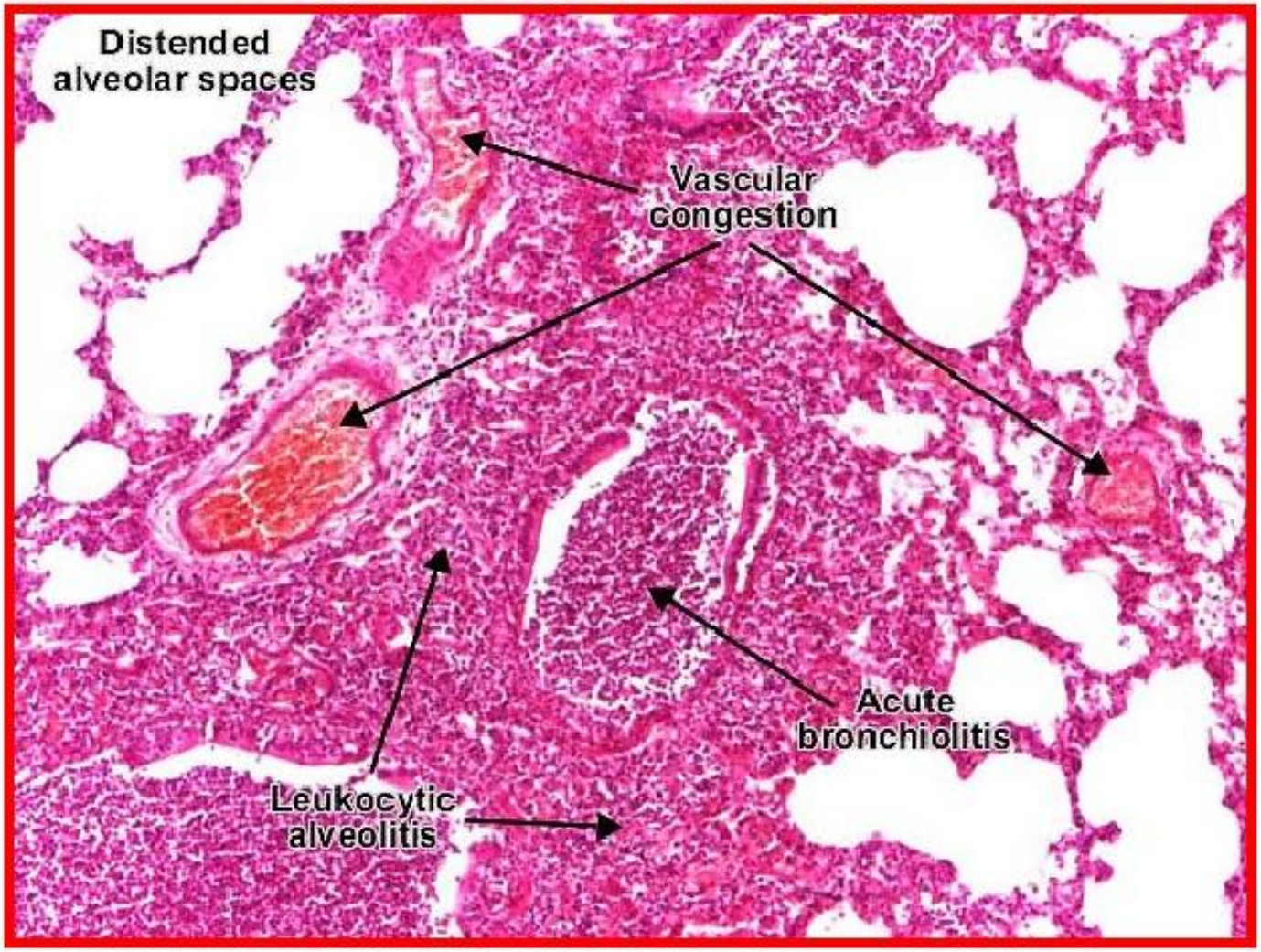
- On cut surface, these patchy consolidated lesions are dry, granular, firm, red or grey in color, 3 to 4 cm in diameter, slightly elevated over the surface and are often centered around a bronchiole.
- These patchy areas are best picked up by passing the fingertips on the cut surface.

- ***Histologically:***

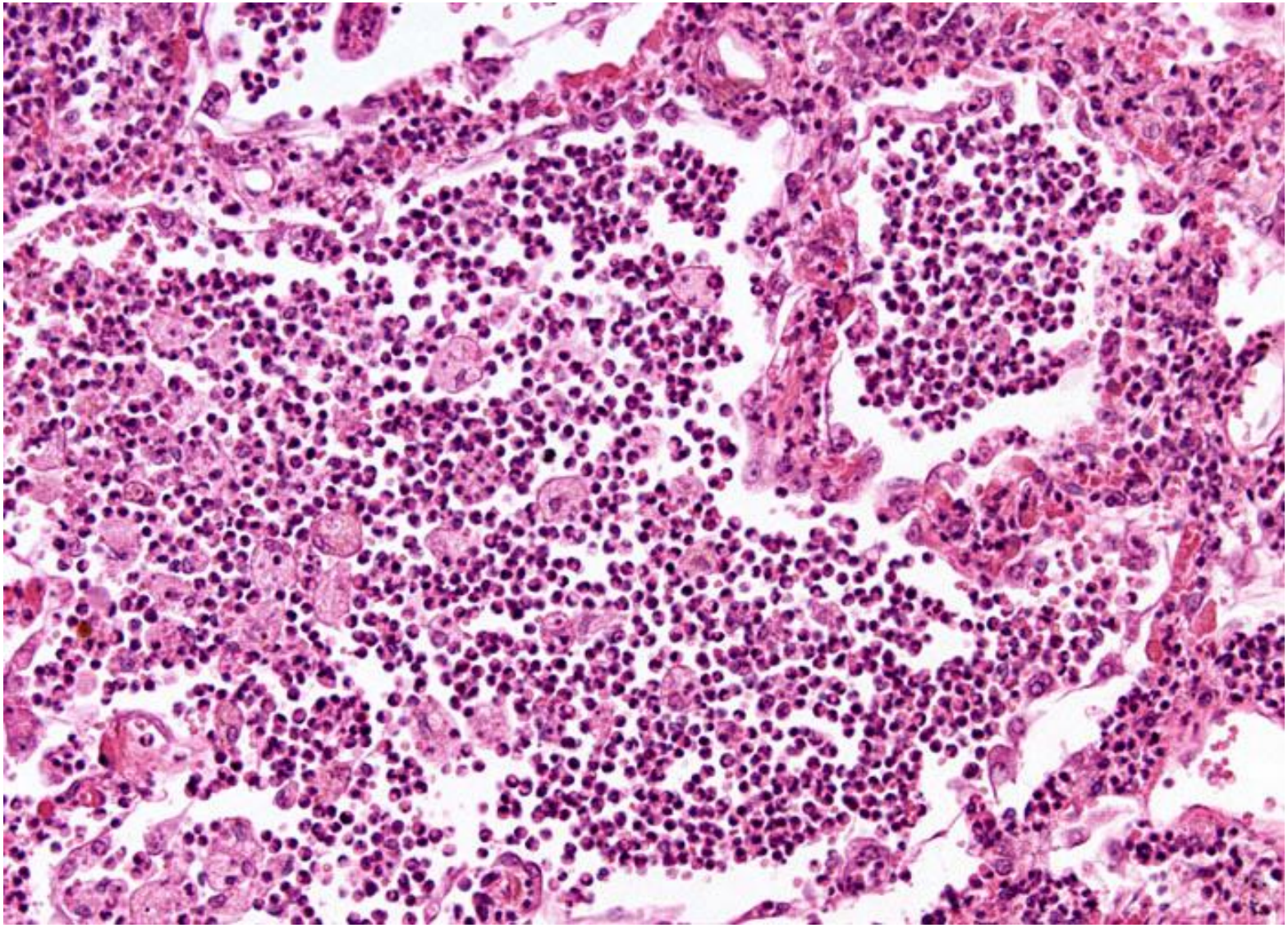
1. Acute bronchiolitis.
2. Suppurative exudate, consisting chiefly of neutrophils, in the peri-bronchiolar alveoli.
3. The reaction usually elicits a neutrophil-rich exudate that fills the bronchi, bronchioles, and adjacent alveolar spaces.
4. Thickening of the alveolar septa by congested capillaries and leucocytic infiltration.
5. Less involved alveoli contain edema fluid.

Bronchopneumonia: Section of lung showing patches of consolidation.

Bronchopneumonia



Bronchopneumonia



CLINICAL FEATURES OF LOBULAR PNEUMONIA

- The patients of bronchopneumonia are generally *infants or elderly* individuals.
- There may be history of preceding bed-ridden illness, chronic debility, aspiration of gastric contents or URT infection.
- For initial 2 to 3 days, there are features *of acute bronchitis* but subsequently signs and symptoms similar to those of lobar pneumonia appear.
- Blood examination usually shows a neutrophilic leucocytosis.
- Chest radiograph shows *mottled*, *focal opacities* in *both lungs*, chiefly in the *lower zones*.

COMPLICATIONS *OF LOBULAR PNEUMONIA*

- The complications of lobar pneumonia may occur in bronchopneumonia as well.
- However, *complete resolution of bronchopneumonia is uncommon*.
- There is generally some degree of *destruction of the bronchioles* resulting in foci of *bronchiolar fibrosis* that may eventually cause bronchiectasis.

1. **Tissue destruction** and necrosis, causing **abscess formation** (particularly common with *Klebsiella infections);*
2. **Spread of infection** to the pleural cavity, causing the intrapleural fibrino-suppurative reaction known as **empyema;** and
3. **Bacteremic dissemination** to the heart valves, pericardium, brain, kidneys, spleen, or joints, causing metastatic abscesses, endocarditis, meningitis, or suppurative arthritis.

COMPARISON OF LOBAR PNEUMONIA AND LOBULAR PNEUMONIA

Features	Lobar Pneumonia	Lobular Pneumonia
Definition	Acute bacterial infection of a part of a lobe of one or both lungs, or the entire lobe/s	Acute bacterial infection of the terminal bronchioles extending into adjoining alveoli
Age group	More common in adults	Commoner at extremes of age–infants and old age
Predisposing factors	More often affects healthy individuals	Preexisting diseases e.g. chronic debility, terminal illness, flu, measles

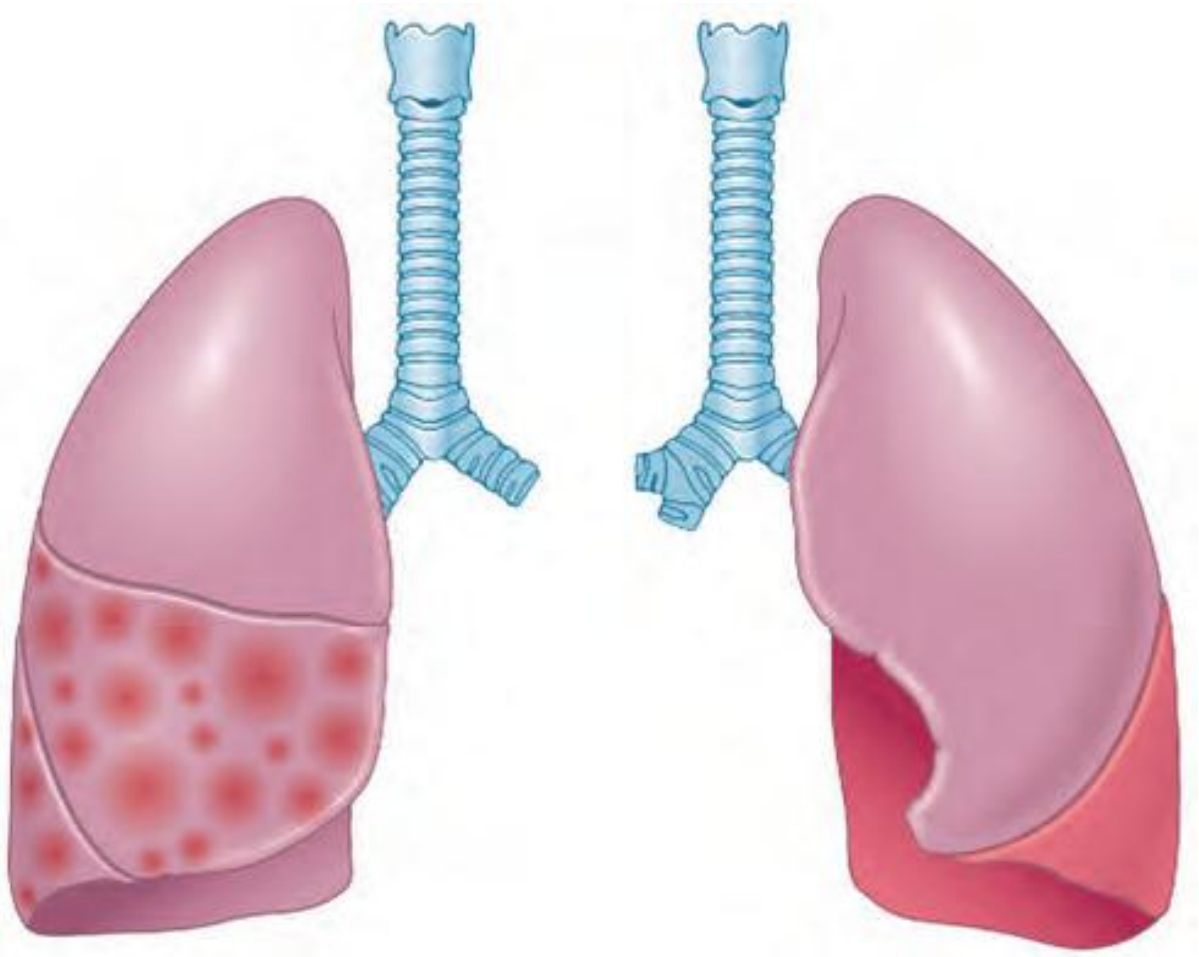
Features	Lobar Pneumonia	Lobular Pneumonia
Common etiological agents	*Pneumococci, Klebsiella pneumoniae, staphylococci, streptococci*	Staphylococci, streptococci, Pseudomonas, *Hemophilus influenze*
Pathologic features	Typical case passes through stages of congestion (1-2 days), early consolidation (2-4 days) and late consolidation (4-8 days), followed by resolution (1-3 weeks)	Patchy consolidation with central granularity, alveolar exudation, thickened septa
Investigations	Neutrophilic leucocytosis, positive blood culture, culture, X-ray shows consolidation	Neutrophilic leucocytosis, positive blood culture, X-ray shows mottled focal opacities

Features	Lobar Pneumonia	Lobular Pneumonia
Prognosis	Better response to treatment, resolution, prognosis good	Response to treatment variable, organization may occur, prognosis poor
Complications	Less common; pleural effusion, empyema lung abscess, organization	Bronchiectasis may occur; other complications the same as for lobar pneumonia

Item	Lobar pneumonia	Lobular pneumonia
Age	Young and adults	Extremes of ages
Organism	Mostly pneumococci	Mixed organisms
Grossly	Affects one or more than one lobe (Diffuse)	Patchy, bilateral basal affection of both lungs
Type of inflam-mations	Fibrinous inflammation	Suppurative inflammation
Mode of healing	Resolution occurs in the most cases	Lysis & complications are most common

Differences between lobar and lobular pneumonia

Comparison of lobar pneumonia and lobular pneumonia



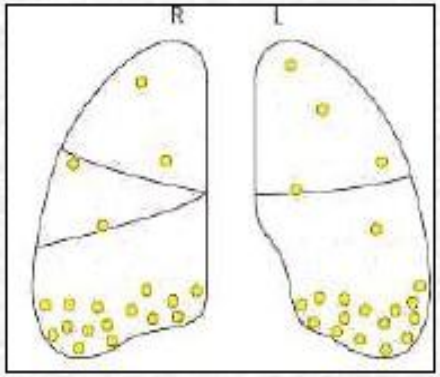
Bronchopneumonia

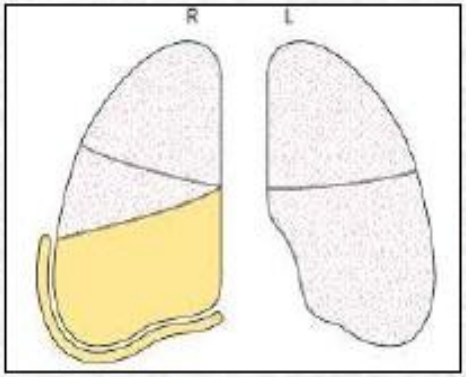
Lobar pneumonia

Broncho – **Pneumonia** - Lobar

- Extremes of age.
- Secondary, in sick.
- Both genders.
- Staph, Strep, H.infl.
- Patchy consolidation
- Around Small Bronchi
- Not limited by anatomic boundaries.
- Usually bilateral.

- Middle age – 20-50
- Primary in a healthy adult.
- males common.
- 95% pneumococcus (Klebs.)
- Entire lobe consolidation
- Diffuse
- Limited by anatomic boundaries.
- Usually unilateral





COMMUNITY-ACQUIRED VIRAL PNEUMONIA

- In adults, viruses account for about a third and in children for 15% of pneumonia cases.
- Common viral infections include influenza virus types A and B, and para-influenza virus, rubella, &varicella viruses, respiratory syncytial viruses (RSV), corona viruses, adenovirus & rhinoviruses.
- ***Herpes simplex virus*** rarely causes pneumonia, except in **newborns**, persons with cancer, transplant recipients, and people with significant burns.

- People following organ transplantation or those otherwise-immunocompromised present high rates of cytomegalovirus (CMV) pneumonia.
- Any of these agents can cause an URT infection; common cold, or a more severe lower respiratory tract (LRT) infection.
- **Factors favoring extension of infection include:** Extremes of age, malnutrition, alcoholism, and underlying debilitating illnesses.
- All of the viruses that cause pneumonia produce disease through similar general mechanisms.
- These viruses have **tropisms** that allow them to ***attach to*** and ***enter*** respiratory lining cells.

- Viral replication and gene expression leads to ***cytopathic*** changes, inducing cell death and secondary inflammation.
- The resulting damage and impairment of local pulmonary defenses, such as muco-ciliary clearance, may *predispose to bacterial super-infections*, which are often more serious than the viral infection itself.
- Secondary bacterial infection occurs by *Strept. pneumoniae*, *Staph. aureus*, or *Hemophilus influenzae*, particularly when other health problems are present.

PATHOGENESIS OF VIRAL PNEUMONIA

- **Viruses may reach the lung by different routes:**
- **RSV** is typically contracted when people *touch contaminated objects* and then they touch their eyes or nose.
- Other viral infections occur when contaminated airborne droplets are *inhaled* through the mouth or the nose.
- Once in the URT, the viruses may make their way in the lungs, where they *invade the cells* lining the airways, alveoli, or lung parenchyma.

- **Measles** and **herpes simplex** may reach the lungs *via the blood*.
- Invasion of the lungs may lead to **cell death**.
- When the immune system responds to infection, more **lung damage** may occur.
- Primarily white blood cells, mainly mononuclear cells, *generate the inflammation*.
- As well as damaging the lungs, many viruses *simultaneously affect other organs* and thus *disrupt other body functions*.
- Viruses make the body *more* ***susceptible to bacterial infections***.

SEVERE ACUTE RESPIRATORY SYNDROME (SARS)

- ***Severe acute respiratory syndrome (SARS)*** first appeared in ***Nov 2002*** in Guangdong Province of China and spread to Hong Kong, Taiwan, Singapore, Vietnam, and Toronto, where large outbreaks also occurred.
- The ease of travel between continents clearly contributed to this initial rapid spread.
- The epidemic went no further, however, perhaps in part because of public health measures, and the last cases of SARS were laboratory-associated infections reported in ***April 2004***.

- The cause of SARS was ***new corona virus***.
- SARS virus differed from other corona viruses in that it infect the LRT and spread throughout the body.
- SARS is a cardinal example of sudden emergence of a new infectious agent, but since 2004 the virus has completely disappeared as mysteriously as its original debut.
- It is unknown if or when it will appear again.

MORPHOLOGY OF VIRAL PNEUMONIA

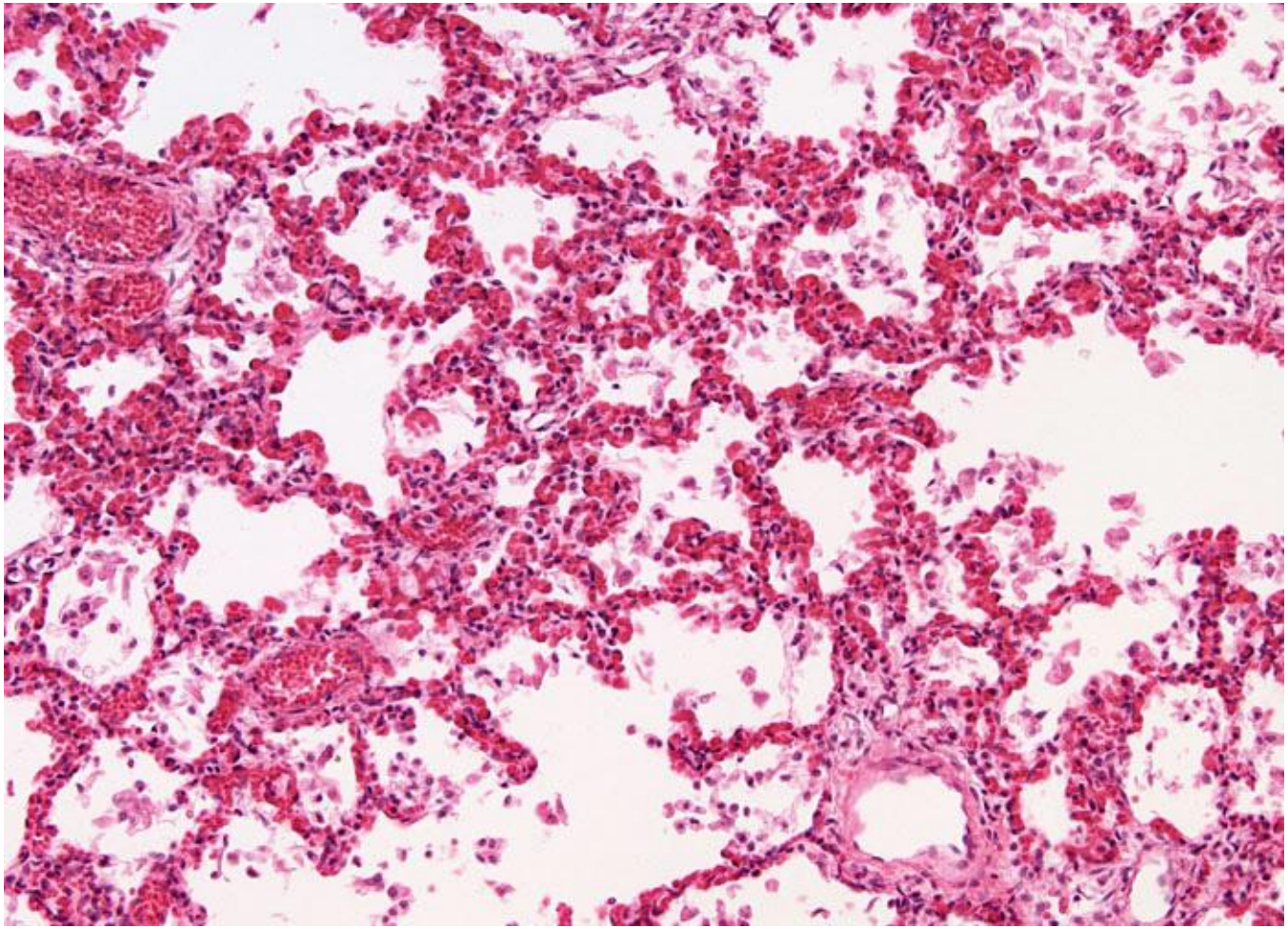
- **All viral infections produce similar morphologic changes.**
- URT infections are marked by *mucosal hyperemia* and *swelling*, *lymphomonocytic and plasmacytic infiltration* of the *submucosa*, & *overproduction of mucus secretions*.
- The swollen mucosa and viscous exudate may *plug* the nasal channels, sinuses or Eustachian tubes, leading to *secondary suppurative bacterial infectio*n.
- *Virus-induced tonsillitis* causing hyperplasia of the lymphoid tissue within the Waldeyer ring is frequent in children.

- In **viral laryngo-tracheo-bronchitis** and **bronchiolitis** there is vocal cord swelling and abundant mucus production.
- ***Impairment of broncho-ciliary function*** invites bacterial super-infection with more marked suppuration.
- ***Plugging of small airways*** may give rise to focal **lung atelectasis**.
- With more severe bronchiolar involvement, widespread plugging of secondary and *terminal airways by cell debris, fibrin, and inflammatory exudate may, if prolonged, lead to organization and fibrosis*, resulting ***in obliterative bronchiolitis*** and ***permanent lung damage***.

- Lung involvement may be patchy or may involve the whole lobes bilaterally or unilaterally.
- The affected areas are *red-blue* and *congested*.
- *Pleuritis* or *pleural effusions* are *infrequent*.
- *The histological pattern depends on the severity of the disease*.
- **Predominant is an interstitial inflammatory reaction involving the walls of the alveoli.**
- **The alveolar septa** are ***widened*** and ***edematous*** and usually have a mononuclear inflammatory infiltrate of lymphocytes, macrophages, and occasionally plasma cells.
- **In acute cases neutrophils may also be present**.

- The alveoli may be *free of exudate*, but in many patients there is ***intra-alveolar proteinaceous material*** and ***cellular exudate***.
- If complicated by ARDS, pink hyaline membranes line the alveolar walls.
- Eradication of infection is followed by reconstitution of the normal lung architecture.
- Superimposed bacterial infection modifies this picture by causing ulcerative bronchitis, bronchiolitis, and bacterial pneumonia.
- Some viruses, such as herpes simplex, varicella, and adenovirus, may be associated with *necrosis* of *bronchial and alveolar epithelium and acute inflammation*.
- ***Characteristic viral cytopathic changes are seen.***

VIRAL PNEUMONIA



CLINICAL COURSE OF VIRAL PNEUMONIA

- The clinical course of viral infections is extremely varied.
- Many cases manifest as severe URT infections.
- *Even individuals* with ***well-developed atypical pneumonia*** have few localizing symptoms.
- ***Cough may be absent***, and the major manifestations may consist ***only of fever, headache, muscle aches, and pains in the legs***.

- **Edema and exudation** cause *mismatching of ventilation and blood flow*, *thus evoke symptoms out of proportion to the scanty physical findings*.
- Viral infections are usually *mild* and *resolve spontaneously* without any lasting sequelae.
- The complications of interstitial viral pneumonias epidemic, can lead to significant morbidity and mortality, as is typically true of influenza epidemics.

INTERSTITIAL PNEUMONIA (PRIMARY ATYPICAL PNEUMONIA)

- **Community Acquired Primary Atypical Pneumonia** is characterized by patchy inflammatory changes, largely confined to the interstitial tissue of the lungs, without any alveolar exudate.
- Other terms used for this respiratory tract infections are ***interstitial pneumonia/pneumonitis, reflecting the interstitial location of the* inflammation**, and ***primary atypical pneumonia, atypicality being the absence of alveolar exudate*** commonly present in other pneumonias.
- Interstitial pneumonia may occur in *all ages*.
- Most of the cases are *mild* and *transient*; but it may be *severe* and *fulminant*.

ETIOLOGY OF INTERSTITIAL PNEUMONIA

- **Interstitial pneumonitis** is caused by a wide variety of agents, the most common being *RSV.*
- *Others are Mycoplasma pneumoniae and many* viruses such as influenza and parainfluenza, CMV adenoviruses, rhinoviruses, & coxsackie viruses.
- Occasionally, psittacosis *(Chlamydia)* and Q fever *(Coxiella) are associated with interstitial* pneumonitis.

MORPHOLOGIC FEATURES OF INTERSTITIAL PNEUMONIA

- ***MORPHOLOGIC FEATURES:***
- ***Irrespective of the etiologic*** agent, the pathological changes are similar in all cases.
- ***Grossly, depending upon the severity of infection, the involvement*** may be **patchy** to **massive** and widespread consolidation of one or both lungs.
- The lungs are **heavy**, **congested** and **subcrepitant**.
- Sectioned surface of the lung exudes small amount of **frothy** or **bloody fluid**.
- The **pleural reaction** is usually **infrequent** and **mild**.

- ***Histologically:***
- ***The hallmark of viral pneumonias is the*** interstitial nature of the inflammatory reaction.

1. **Interstitial inflammation:**

- There is thickening of alveolar walls due to congestion, edema and mononuclear inflammatory infiltrate comprised by lymphocytes, macrophages and some plasma cells.

2. **Necrotising bronchiolitis:**

- This is characterized by foci of necrosis of the bronchiolar epithelium, inspissated secretions in the lumina and mononuclear infiltrate in the walls and lumina.

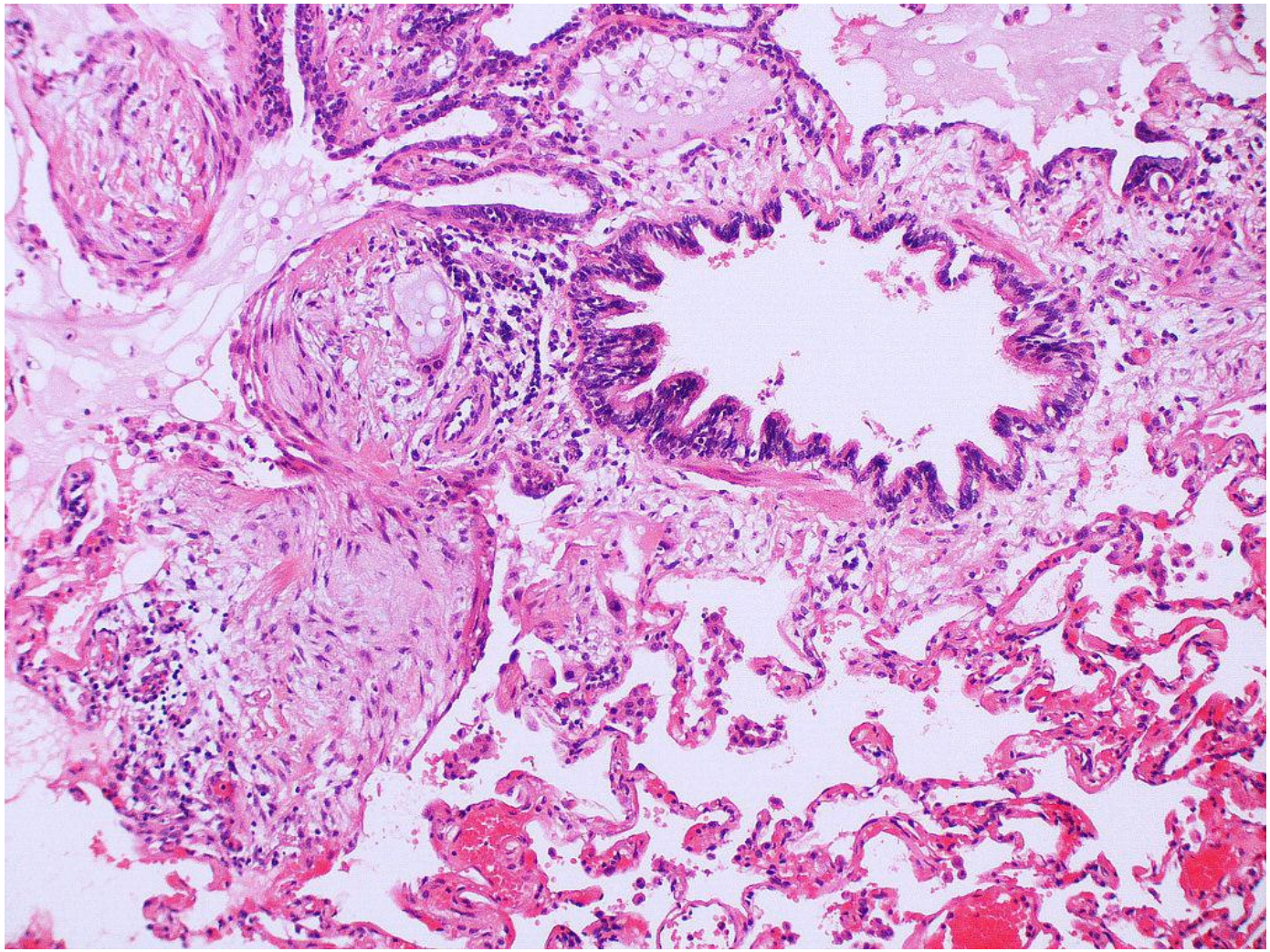
3. **Reactive changes:**

- The lining epithelial cells of the bronchioles and alveoli proliferate in the presence of virus and may form multinucleate giant cells and syncytia in the bronchiolar and alveolar walls.
- Occasionally, viral inclusions (intranuclear and/or intracytoplasmic) are found, especially in pneumonitis caused by CMV.

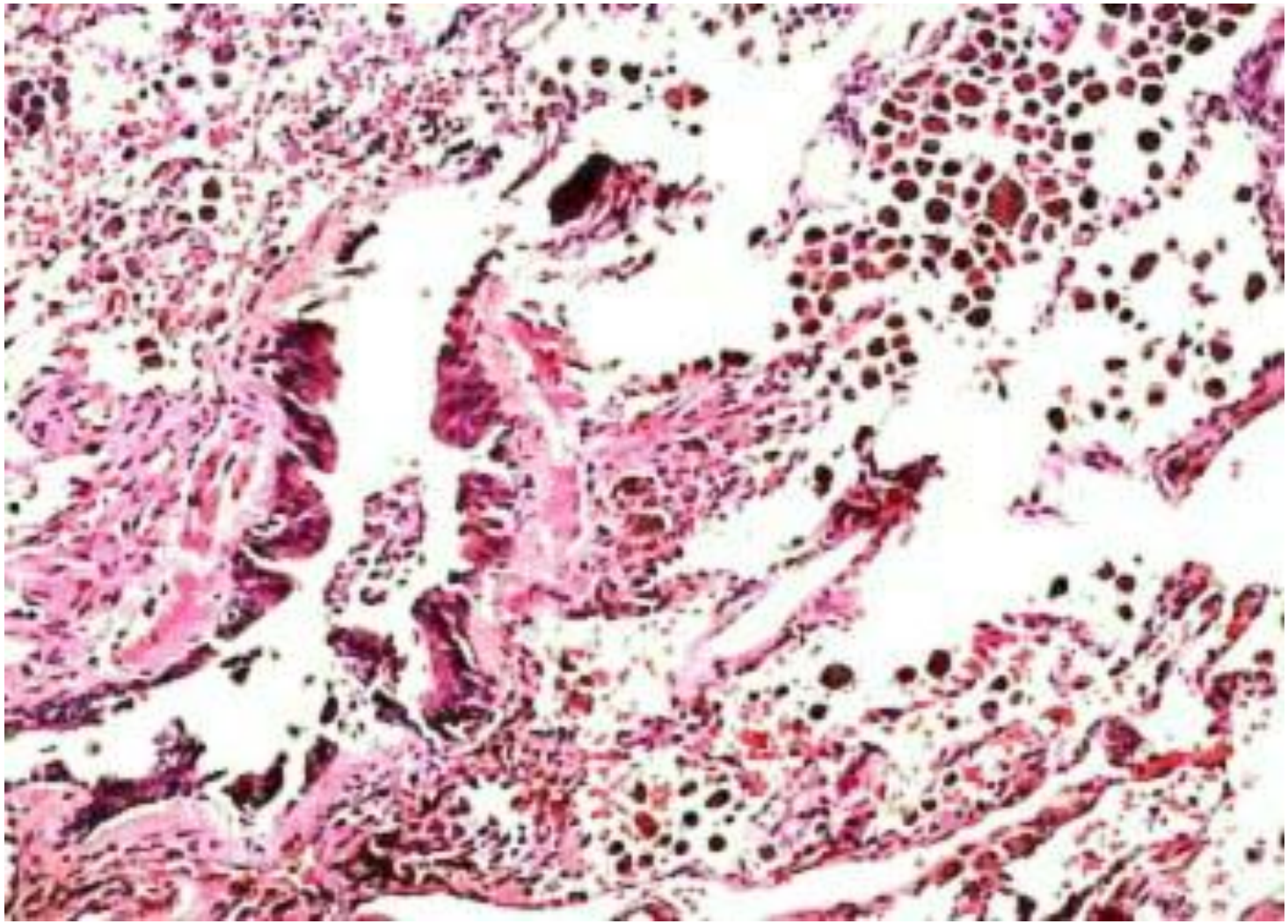
4. **Alveolar changes:**

- In severe cases, the alveolar lumina may contain edema fluid, fibrin, scanty inflammatory exudate and coating of alveolar walls by pink, hyaline membrane similar to the one seen in respiratory distress.
- Alveolar changes are prominent if bacterial infection supervenes.

INTERSTITIAL PNEUMONIA



INTERSTITIAL PNEUMONIA



CLINICAL FEATURES OF INTERSTITIAL PNEUMONIA

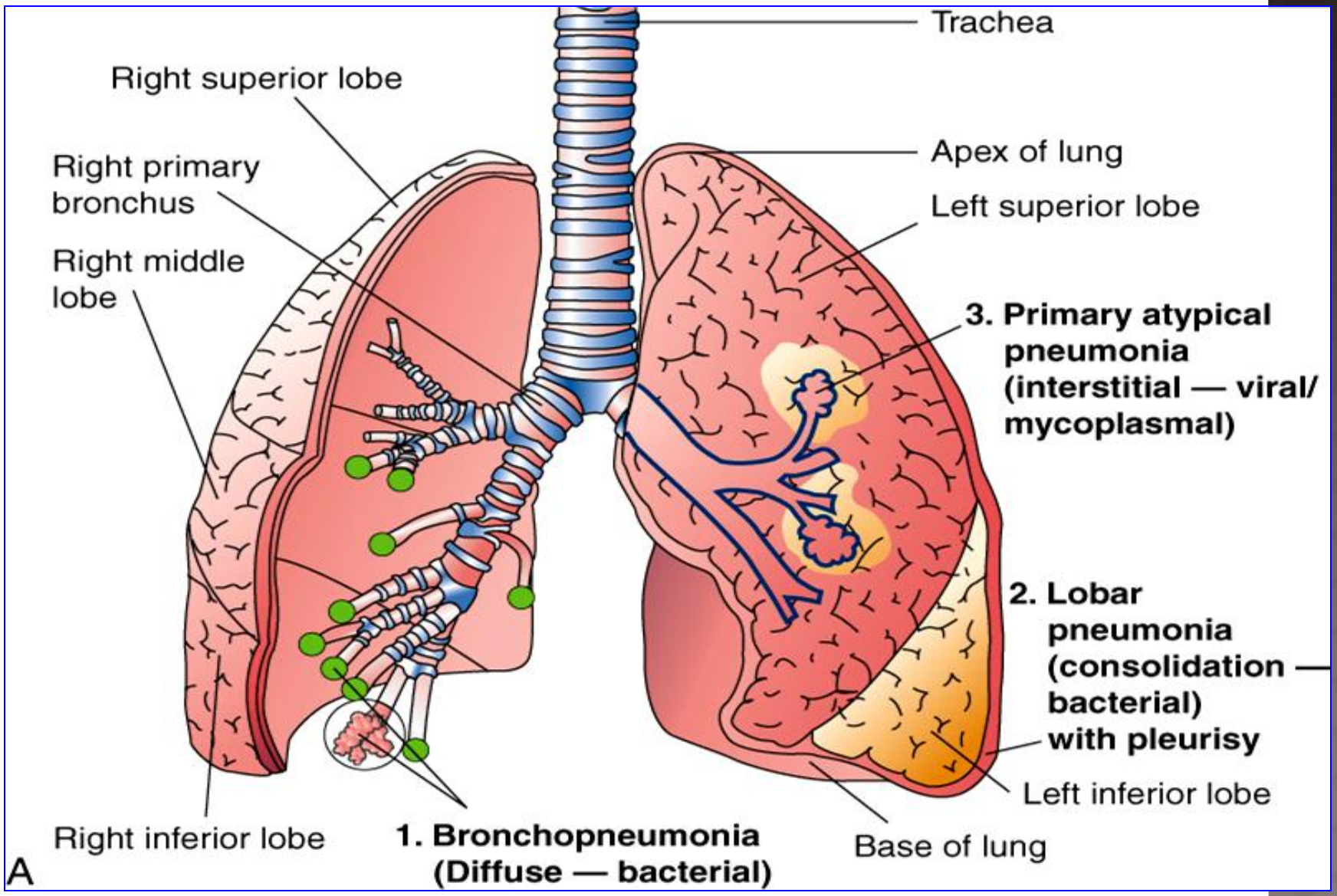
- The majority of interstitial pneumonia cases initially have URT infection symptoms with fever, headache and muscle-aches.
- A few days later appears dry, hacking, non-productive cough with retrosternal burning due to tracheitis and bronchitis.
- Blood film shows characteristic neutrophilic leucocytosis.
- Chest radiograph may show patchy or diffuse consolidation.

- **Cold agglutinin titers** in serum are elevated in almost half the cases of **mycoplasmal pneumonia**, 20% cases of adenovirus infection but absent in other forms of viral pneumonia.
- Isolation of the etiological agent, otherwise, is difficult.

COMPLICATIONS OF INTERSTITIAL PNEUMONIA

- The major complications of interstitial pneumonia is ***superimposed bacterial infection*** and its complications.
- Most cases of interstitial pneumonia ***recover completely***.
- In more severe cases, there may be ***interstitial fibrosis*** and ***permanent damage***.

COMPARING TYPES OF PNEUMONIA



HEALTH CARE-ASSOCIATED PNEUMONIA (HCAP)

- Health-care associated pneumonia was recently described an infection associated with recent exposure to the health care system, including hospitalization of at least 2 days within the recent past; presentation from a nursing home or long-term care facility; attending hemodialysis clinic; and recent intravenous antibiotic therapy, chemotherapy or wound care.
- HCAP is sometimes called ***medical care-associated pneumonia (MCAP)***.
- The most common organisms isolated are methicillin-resistant *Staphylococcus aureus and P. aeruginosa*.
- *These patients have a higher mortality* than those with community-acquired pneumonia.

HOSPITAL-ACQUIRED PNEUMONIA (HAP)

- Hospital-acquired pneumonias are defined as pulmonary infections acquired in the course of a hospital stay.
- They are common in patients with severe underlying disease, prolonged antibiotic therapy, immunosuppression, or invasive access devices such as intravascular catheters.
- Patients on mechanical ventilation are at particularly high risk.

- Superimposed on an underlying disease (that caused hospitalization), hospital-acquired infections are serious and often life-threatening complications.
- Gram-positive cocci (mainly *S. aureus and S. pneumonia) and gram-negative* rods (Entero-bacteria and *Pseudomonas species) are the* most common isolates.
- There are similar organisms isolated in ***ventilator associated pneumonia*** but gram-negative bacilli are more common.

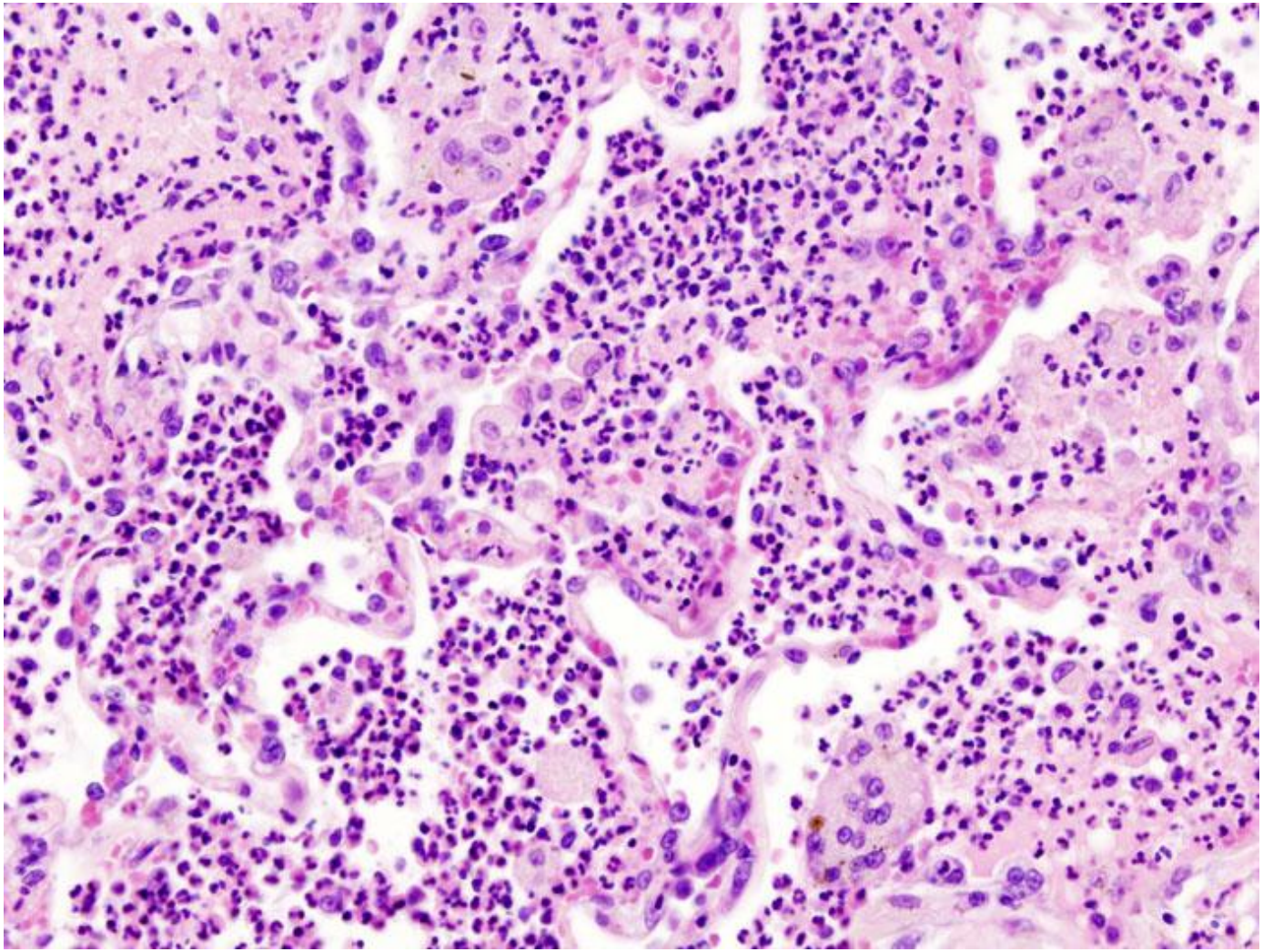
ASPIRATION PNEUMONITIS/PNEUMONIA

- Aspiration pneumonitis (or 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* or *gastric reflux*.
- Those patients have *abnormal gag* and *swallowing reflexes* that predispose to aspiration.
- The resultant bronchopneumonia is *partly chemical* due to the irritating effects of gastric acid, and *partly bacterial* (from the oral flora).

- Typically, more than one organism is recovered on culture, aerobes being more common than anaerobes.
- This type of pneumonia is often *necrotizing*, has *fulminant* clinical course, and is a frequent cause of death since susceptible patients often cannot adequately protect their airways and may have otherwise impaired defenses.
- In those who survive, *lung abscess* is a common complication.

- *Micro-aspiration*, in contrast, occurs frequently in *almost all people*, especially those with gastro-esophageal reflux disease; *GERD*.
- It usually results in *small*, *poorly formed non-necrotizing granulomas* with *multinucleated foreign body giant cell reaction*.
- It is usually inconsequential, but may exacerbate other preexisting lung diseases such as asthma, interstitial fibrosis, and lung rejection.

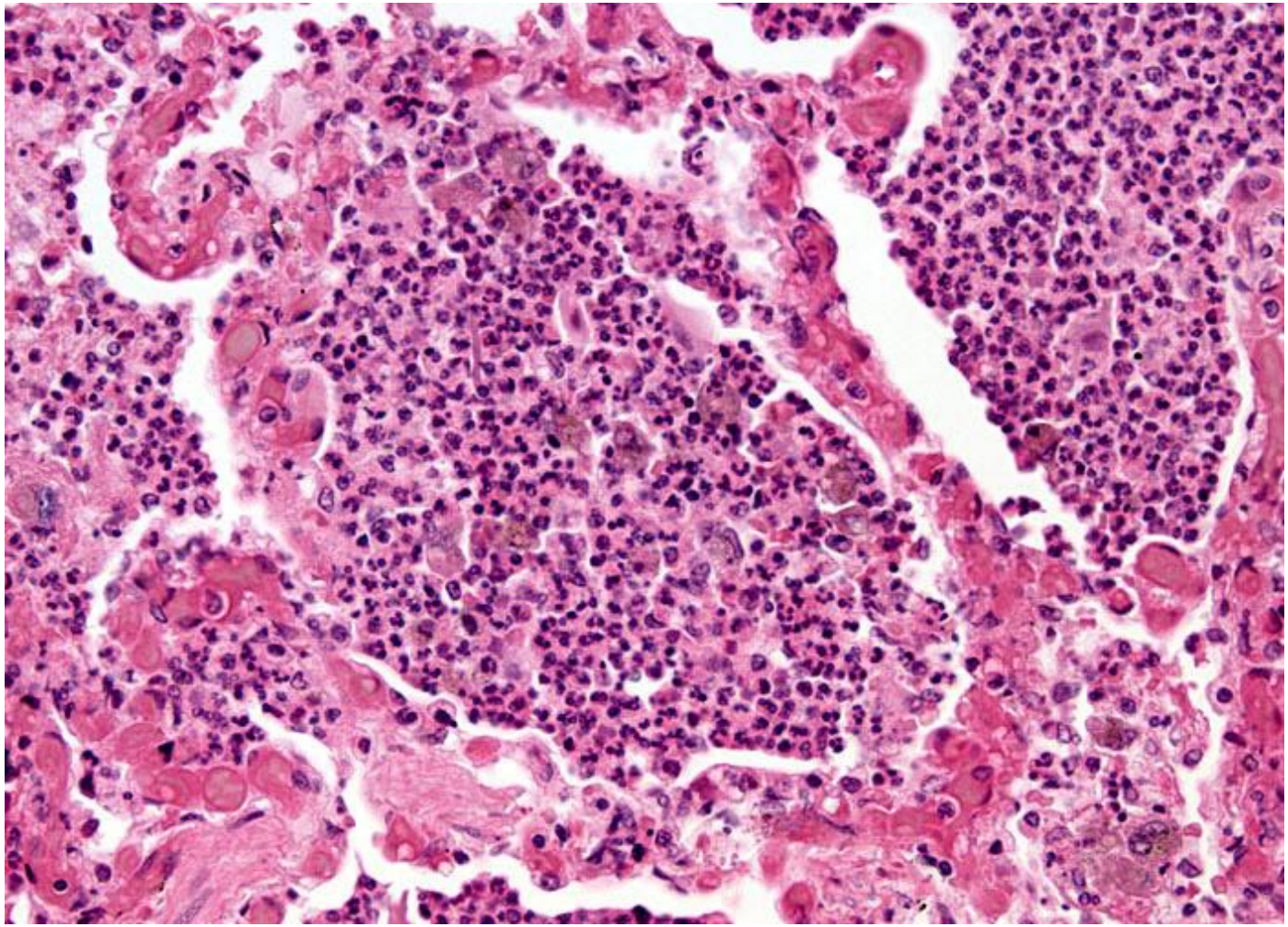
ASPIRATION PNEUMONITIS



CHRONIC PNEUMONIA

- Chronic pneumonia is most often a localized lesion in the immunocompetent patient, with or without regional lymph node involvement.
- Typically, the inflammatory reaction is *granulomatous*, and is caused by ***bacteria*** (e.g., *M. tuberculosis) or* ***fungi*** *(e.g., Histoplasma capsulatum).*

CHRONIC PNEUMONIA



PULMONARY TUBERCULOSIS

- The classical and most common example of chronic infection of the lungs is pulmonary tuberculosis.
- Pulmonary lesions caused by Mycobacterium tuberculosis and other mycobacteria have already been discussed along with general aspects of tuberculosis and other granulomatous inflammations.

HISTOPLASMOSIS

- *Histoplasma capsulatum infection is acquired by inhalation of dust particles from soil contaminated with bird or bat droppings that contain small spores (microconidia)*; the infectious form of the fungus.
- It is endemic along the Ohio and Mississippi rivers and the Caribbean.
- It is also found in Mexico, Central and South America, parts of Eastern and Southern Europe, Africa, Eastern Asia and Australia.
- Like *M. tuberculosis, H. capsulatum is an intracellular pathogen* that is found mainly in phagocytes.

- The **clinical presentations** and **morphological lesions** of histoplasmosis also strikingly resemble those of tuberculosis, including:

1. A self-limited and often latent primary pulmonary involvement, which may result in coin lesions on chest radiography.
2. Chronic, progressive, secondary lung disease, which is localized to the lung apices and causes cough, fever, and night sweats.
3. Spread to extrapulmonary sites, including mediastinum, adrenals, liver, or meninges.
4. Widely disseminated disease in immuno-compromised patients.

- Histoplasmosis can occur in immuno-competent individuals but as expected it is more severe in those with *depressed cell mediated immunity*.
- The pathogenesis of histoplasmosis is incompletely understood.
- It is known that *macrophages* are the major target of infection.
- *H. capsulatum may be internalized into macrophages after opsonization with antibody*.
- *Histoplasma* yeasts can multiply and lyse the host cells.

- *Histoplasma infections are controlled by helper T cells* that recognize fungal antigens and subsequently secrete IFN-γ, which *activates macrophages to kill intracellular yeasts*.
- *Histoplasma induces macrophages to secrete TNF*, which recruits and stimulates other macrophages to *kill Histoplasma*.

MORPHOLOGY OF HISTOPLASMOSIS

- In the lungs of otherwise healthy adults, *Histoplasma infections* produce ***granulomas, which usually undergo caseation necrosis*** and coalesce to produce large areas of consolidation, but may also liquefy to form cavities particularly in patients with COPD.
- With spontaneous resolution or effective treatment, these lesions undergo fibrosis and concentric calcification ***(Treebark Appearance)***.

- *Histological differentiation from* tuberculosis, sarcoidosis, and coccidioidomycosis requires identification of the 3- to 5-μm *thin-walled yeast forms*, which may persist in tissues for years.
- In ***fulminant disseminated histoplasmosis***, which occurs in immunosuppressed individuals, granulomas do not form; instead, there *are focal accumulations of mononuclear phagocytes filled with fungal yeasts throughout the body.*

DIAGNOSIS OF HISTOPLASMOSIS

- The diagnosis of histoplasmosis is established by culture or identification of the fungus in tissue lesions.
- In addition, *serologic tests* for antibodies and antigen are also available.
- **Antigen detection** in body fluids is most useful in the *early stages*, because **antibodies** are formed 2 to 6 weeks after infection.

BLASTOMYCOSIS

- *Blastomyces dermatitidis is a soil-inhabiting dimorphic fungus*.
- It causes disease in Central and South-Eastern United States; infection also occurs in Canada, Mexico, the Middle East, Africa, and India.
- **There are three clinical forms:** *pulmonary blastomycosis, disseminated blastomycosis,* and a rare *primary cutaneous form* that results from direct inoculation of organisms into the skin.

- **Pulmonary blastomycosis** most often presents as an abrupt illness with productive cough, chest pain, headache, weight loss, fever, abdominal pain, night sweats, chills, and anorexia.
- **Chest radiographs** reveal lobar consolidation, multilobar infiltrates, perihilar infiltrates, multiple nodules, or miliary infiltrates.
- The *upper lobes* are most frequently involved.
- The pneumonia most often *resolves spontaneously*, but it may *persist*, or *progress to a chronic lesion*.

MORPHOLOGY OF BLASTOMYCOSIS

- In the normal host, the lung lesions of blastomycosis are **suppurative granulomas**.
- Macrophages have a limited ability to ingest and kill *B. dermatitidis, and the persistence of the yeast* cells leads to continued recruitment of neutrophils.
- In tissue, *B. dermatitidis is a round, 5- to 15-μm yeast cell that divides* by broad-based budding.
- It has a *thick, double-contoured cell wall, and visible nuclei*.
- Involvement of the *skin and larynx* is associated with marked *epithelial hyperplasia*, which may be mistaken for squamous cell carcinoma.

COCCIDIOIDOMYCOSIS

- Almost everyone who inhales the *spores of Coccidioides immitis becomes infected and develops a delayed-type hypersensitivity reaction* to the fungus.
- More than 80% of people in *endemic areas* of the SouthWestern and Western United States and in Mexico have a *positive skin test reaction*.
- One reason for the infectivity of *C. immitis is* that infective arthroconidia, when *ingested by alveolar macrophages*, *resist intracellular killing*.

- As is the case with *Histoplasma*, *most primary infections with C. immitis are* asymptomatic, but 10% of infected people develop lung lesions, fever, cough, and pleuritic pains, accompanied by erythema nodosum or erythema multiforme (the San Joaquin Valley fever complex).
- Less than 1% of people develop disseminated *C. immitis infection, which frequently* involves the *skin and meninges*.
- **Certain ethnic groups** (e.g., Filipinos and African Americans) and the immunosuppressed are at particularly *high risk for disseminated disease*.

MORPHOLOGY OF COCCIDIOIDOMYCOSIS

- The primary and secondary lung lesions of *C. immitis are similar to* the granulomatous lesions of *Histoplasma*.
- *Within macrophages* or *giant cells*, *C. immitis is present as thick-walled*, *non-budding spherules* 20 to 60 μm in diameter, often *filled with small endospores*.
- A *pyogenic reaction* is superimposed when the spherules rupture to release the endospores.

- Rare progressive *C. immitis disease involves the lungs, meninges,* skin, bones, adrenals, lymph nodes, spleen, or liver.
- At all these sites, the **inflammatory response** may be purely *granulomatous*, *pyogenic*, or *mixed*.
- *Purulent lesions* dominate in patients with *diminished resistance* and with widespread dissemination.

NECROTIZING PNEUMONIA & LUNG ABSCESS

- Although overlapping with many other classifications, necrotizing pneumonia includes *pneumonias that cause substantial necrosis of lung cells*, and sometimes even *lung abscess*.
- *Implicated bacteria* are extremely commonly aerobic bacteria, like Staphylococcus aureus, Klebsiella pneumoniae and Streptococcus pyogenes with or without additional facultatively anaerobic ones.
- The bacteria Pneumococcus type III is sometimes implicated.

LUNG ABSCESS

- ***The term pulmonary abscess describes a local suppurative process that produces necrosis of lung tissue.***
- Oro-pharyngeal surgical or dental procedures, sino-bronchial infections, and bronchiectasis play important roles in their development.

ETIOLOGY & PATHOGENESIS OF LUNG ABSCESS:

- Although under appropriate circumstances any pathogen can produce an abscess, the commonly isolated organisms include *aerobic and anaerobic streptococci, S. aureus, and gram-negative organisms*.
- Mixed infections often occur because of the important causal role played by *inhalation of foreign material*.
- *Anaerobic organisms normally found in the oral cavity,* including members of the *Bacteroides, Fuso-bacterium, and Peptococcus species, are the exclusive isolates in about 60%* of cases.

- **The causative organisms are introduced by the following mechanisms:**

1. ***Aspiration of infective material (the most frequent cause):***

- This is particularly common in acute alcoholism, coma, anesthesia, sinusitis, gingivo-dental sepsis, and debilitation in which the cough reflexes are depressed.
- Aspiration first causes pneumonia which progresses to *tissue necrosis* and formation of *lung abscess*.

2. ***Antecedent primary lung infection:***

- *Post-pneumonic abscess* formations are usually associated with *S. aureus*, *K. pneumoniae*, *and type 3 pneumococcus*.
- ***Post-transplant*** or otherwise immuno-suppressed individuals are at special risk.

3. ***Septic embolism:***

- *Infected emboli from thrombophlebitis* in any portion of the systemic venous circulation or from the vegetations of infective bacterial endocarditis on the right side of the heart are trapped in the lung.

4. ***Neoplasia:***

- *Secondary infection is particularly common in* the bronchopulmonary segment obstructed by a primary or secondary malignancy *(post-obstructive pneumonia)*.

5. ***Miscellaneous:***

- *Direct traumatic penetrations of the lungs*; spread of infections from a neighboring organ, such as suppuration in the esophagus, spine, subphrenic space, or pleural cavity; and *hematogenous seeding* of the lung by pyogenic organisms all may lead to lung abscess formation.

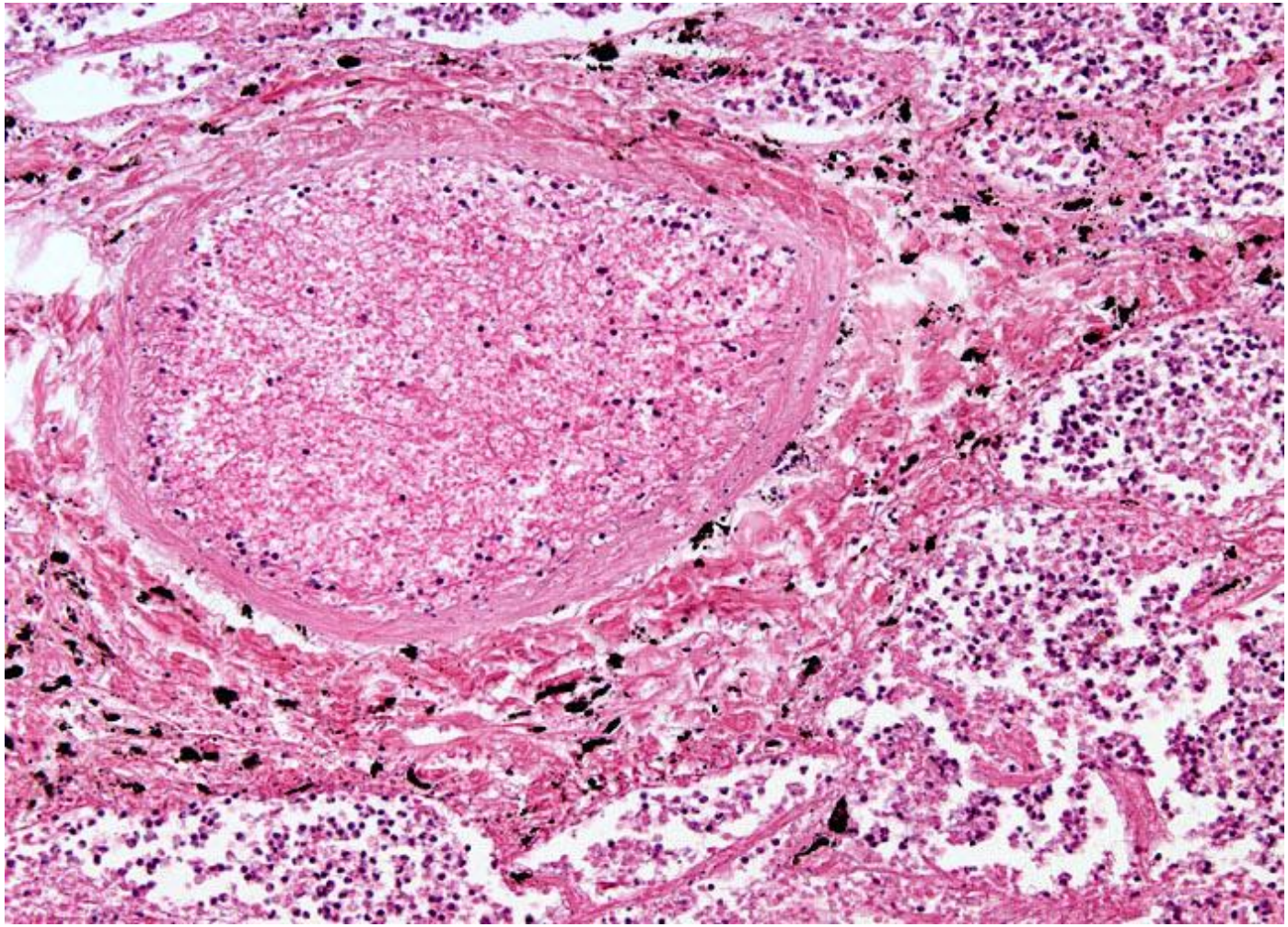
- When all these causes are excluded, there are still cases in which no discernible basis for the abscess formation can be identified.
- These are referred to as ***primary cryptogenic lung abscesses.***

MORPHOLOGY OF LUNG ABSCESS

- Abscesses vary in diameter from a few millimeters to large cavities of 5 to 6 cm.
- They may affect any part of the lung and may be single or multiple.
- Pulmonary abscesses due to aspiration are more common on the *right* (because of the more vertical right main bronchus) and are most often *single*.
- Abscesses that develop in the course of pneumonia or bronchiectasis are usually *multiple*, *basal*, and *diffusely scattered*.
- Septic emboli and pyemic abscesses are multiple and may affect *any region of the lungs*.

- The **cardinal histological change in all abscesses is suppurative destruction of the lung parenchyma within the central area of cavitation**.
- **The abscess cavity may be** filled with suppurative debris or, if there is communication with an air passage, may be partially drained to create an air-containing cavity.
- Superimposed *saprophytic infections* are prone to develop within the necrotic debris.
- Continued infection leads to ***large***, ***poorly demarcated***, ***fetid***, ***green-black***, ***multilocular cavities designated gangrene of the lung***.
- In **chronic** cases considerable *fibroblastic proliferation* produces a **fibrous wall**.

LUNG ABSCESS



CLINICAL COURSE OF ABSCESS

- The manifestations of pulmonary abscesses are much *like* those of *bronchiectasis* and are characterized principally by *cough*, *fever*, and copious amounts of *foul-smelling purulent* or *sanguineous sputum*.
- *Chest pain*, and *weight loss* are common.
- *Clubbing of the fingers* and *toes* may appear within few weeks after the onset of an abscess.
- **Diagnosis** can be only suspected from the clinical findings and must be confirmed radiologically.

- Whenever an abscess is discovered in older individuals, it is important to rule out an underlying carcinoma, which is present in 10% to 15% of cases.
- The course of abscesses is variable.
- With antimicrobial therapy, most *resolve leaving behind a scar*.
- **Complications:** include extension of infection into the pleural cavity, hemorrhage, development of *brain abscesses or meningitis* from septic emboli, and secondary amyloidosis (type AA).

PNEUMONIA IN THE IMMUNOCOMPROMISED HOST

- The appearance of a *pulmonary infiltrate*, with or without signs of infection (e.g., fever), is one of the most common and serious complications in patients whose immune defenses are suppressed by disease, immunosuppressive therapy for organ transplants, chemotherapy or irradiation.
- A wide variety of the so-called *opportunistic infectious agents*, many of which rarely cause infection in normal hosts, can cause these pneumonias, and often more than one agent is involved.

- **Mortality** from these opportunistic infections is high.
- The **differential diagnosis** of such infiltrates includes **drug reactions** and involvement of the lung by **tumor**.
- The specific infections that commonly involve the lung can be classified according to the etiologic agent:

1. Bacteria (*P. aeruginosa*, *Mycobacterium* species, *L. pneumophila, and Listeria monocytogenes)*,
2. Viruses (CMV and herpesvirus), and
3. Fungi (*Candida species*, *Aspergillus species*, *Phycomycetes, and Cryptococcus neoformans)*.

CAUSES OF PULMONARY INFILTRATES IN IMMUNOCOMPROMISED HOSTS

Diffuse Infiltrates	Focal Infiltrates
Common	
Cytomegalovirus *Pneumocystis jiroveci* Drug reaction	Gram-negative bacterial infections *Staphylococcus aureus* *Aspergillus* *Candida* Malignancy
Uncommon	
Bacterial pneumonia *Aspergillus* *Cryptococcus* Malignancy	*Cryptococcus* *Mucor* *Pneumocystis jiroveci* *Legionella pneumophila*

LIPOID PNEUMONIA/ PNEUMONITIS

- **Definition:**
- Lipoid pneumonia is non-infective pneumonia occurs often as complication of debilitating disease.
- It is found as an *incidental post mortem finding*.
- It may be confused with a malignant neoplasm and consequently may become a surgical problem.
- **Lipoid pneumonia** can be **divided into two types:** exogenous and endogenous.

1. ***The exogenous type:***

- This is caused by aspiration of a variety of oily materials, e.g., inhalation of oily nasal drops, regurgitation of oily medicines from stomach (e.g. liquid paraffin), administration of oily vitamin preparation to reluctant children or to debilitated old patients.

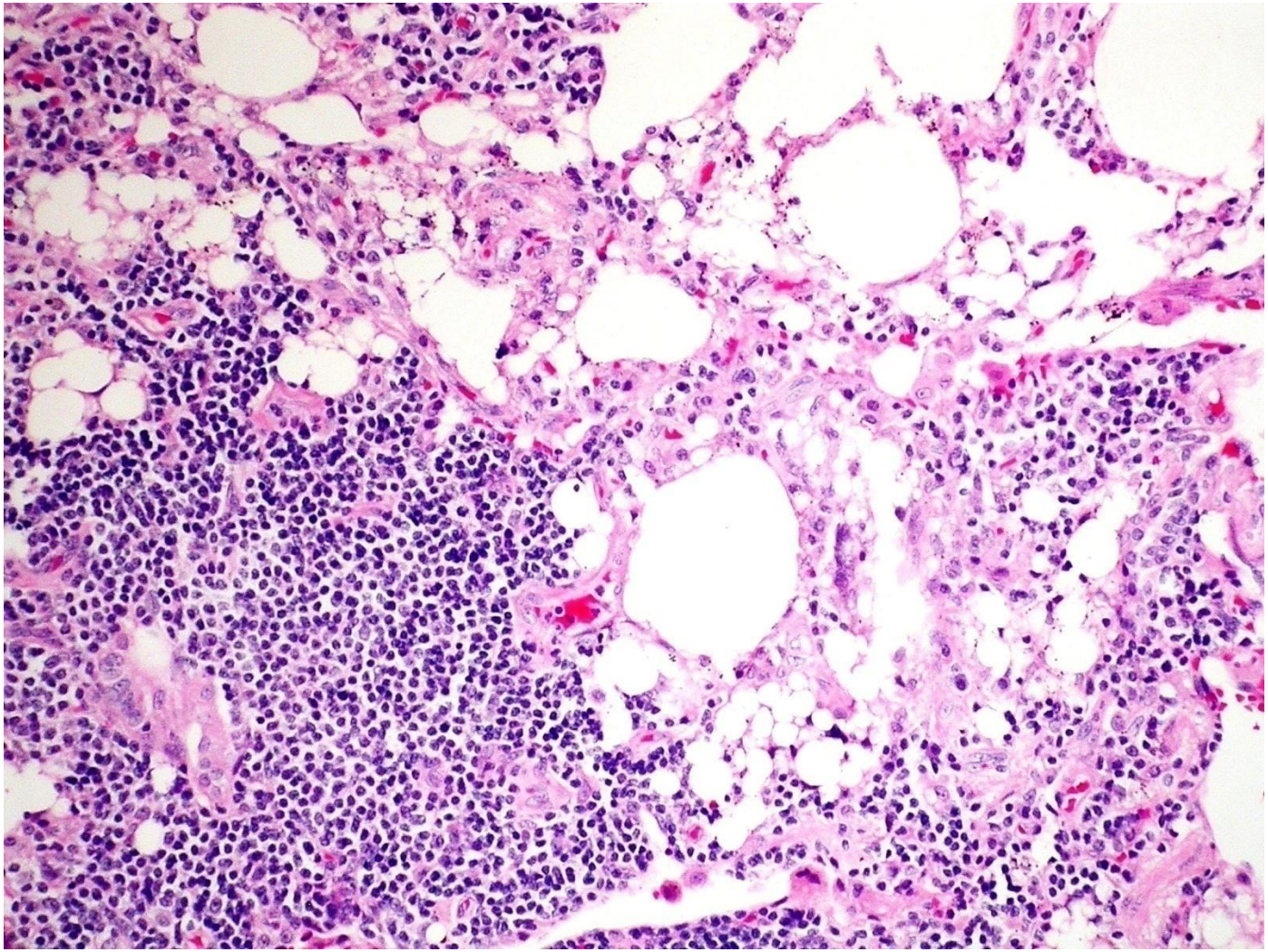
2. ***The endogenous type:***

- Endogenous origin of lipids causing pneumonic consolidation is more common.
- The sources of origin are tissue breakdown following obstruction to airways e.g. obstruction by bronchogenic carcinoma, tuberculosis and bronchiectasis.

- **Grossly:**
- **The exogenous lipid pneumonia** affects the right lung more frequently due to direct path from the main bronchus.
- Quite often, the lesions are bilateral.
- The affected part of the lungs is consolidated.
- Cut surface is characteristically ***'golden yellow'***.
- The lesion is well circumscribed and firm.
- In **endogenous lipid pneumonia** the lymph vessels over the surface of the lung are often prominent, suggesting lymphatic permeation by carcinoma, and fat droplets may be seen flowing from the fresh cut surface.

- **Microscopically:**
- Lipid is finely dispersed in the cytoplasm of macrophages forming foamy macrophages within the alveolar spaces.
- There may be formation of cholesterol clefts due to liberation of cholesterol and other lipids.
- Formation of granulomas with foreign body giant cells may be seen around the large lipid droplets.
- Both forms exhibit *sudanophilic lipoid material*, inflammatory cells, proliferating alveolar cells, and fibroblasts.
- There may also be reactive endarteritis.
- The marked **hyperplasia of alveolar cells** and histiocytes may cause confusion in cytology or frozen section interpretation.

LIPOID PNEUMONIA/ PNEUMONITIS



EOSINOPHILIC PNEUMONIA

- **Definition:**
- **Eosinophilic pneumonia** is a generic term that embraces all pulmonary infiltrations associated with peripheral eosinophilia, as well as infiltrations of the lung by eosinophils with or without peripheral eosinophilia.
- Langerhans cell histiocytosis is excluded.
- The acute form of eosinophilic pneumonia, characterized by *fleeting pulmonary infiltrates* accompanied by eosinophilia and lasting no more than a month, is commonly referred to as ***Löffler syndrome***.

- Most cases of eosinophilic pneumonia are of a chronic nature, although the onset can be quite sudden.
- This is a disease of women, usually between the ages of 20 and 50 years.
- **Clinically,** it is characterized by fever, weight loss, dyspnea, frequent peripheral eosinophilia, and pulmonary infiltrates.
- The **radiographic appearance** is very characteristic because of the distinctly peripheral distribution of the infiltrate.
- The most notable microscopic change is alveolar and interstitial infiltration by eosinophils, but there are also plasma cells and histiocytes.

- **Charcot–Leyden crystals** may be found.
- Additional features seen include mild angiitis, granulomatosis with giant cell formation, some fibrosis with organization, mucous plugging, and bronchiolitis with necrosis.
- Eosinophilic pneumonia has been described *in association with rheumatoid arthritis*, *polyarteritis nodosa*, *malignant lymphoma*, *breast carcinoma*, *nephrotic syndrome*, *scleroderma*, *ulcerative colitis*, and hypersensitivity to nitrofurantoin (a drug used in the treatment of urinary tract infection).

- Helminths, drugs, *Filaria*, *Aspergillus* have been identified as the etiologic agents in some cases.
- When **chronic eosinophilic pneumonia** develops in a patient who has long-standing asthma, it is usually due to **allergic aspergillosis**.
- If the changes of eosinophilic pneumonia are accompanied by *necrotizing vasculitis*, there is a good probability of *extrapulmonary involvement*.
- In *filariasis*, presentation may be in the form of *single or multiple subpleural infarcts* with central thrombosed artery containing the parasite.


THANK YOU