

## **PNEUMONIAS**

Pneumonia is defined as acute inflammation of the lung parenchyma distal to the terminal bronchioles which consist of the respiratory bronchiole, alveolar ducts, alveolar sacs and alveoli. The terms 'pneumonia' and 'pneumonitis' are often used synonymously for inflammation of the lungs, while 'consolidation' (meaning solidification) is the term used for macroscopic and radiologic appearance of the lungs in pneumonia.

**PATHOGENESIS.** The microorganisms gain entry into the lungs by one of the following four routes:

1. Inhalation of the microbes. 2. Aspiration of organisms. 3. Haematogenous spread from a distant focus. 4. Direct spread from an adjoining site of infection. Failure of defense mechanisms and presence of certain predisposing factors result in pneumonias. These conditions are as under: 1. Altered consciousness. 2. Depressed cough and glottic reflexes. 3. Impaired mucociliary transport. 4. Impaired alveolar macrophage function. 5. Endobronchial obstruction. 6. Leucocyte dysfunctions.

**CLASSIFICATION.** On the basis of the anatomic part of the lung parenchyma involved, pneumonias are traditionally classified into 3 main types: 1. **Lobar pneumonia.** 2. **Bronchopneumonia** (or Lobular pneumonia). 3. **Interstitial pneumonia.**

### **A. BACTERIAL PNEUMONIA**

Bacterial infection of the lung parenchyma is the most common cause of pneumonia or consolidation of one or both the lungs. Two types of acute bacterial pneumonias are distinguished—lobar pneumonia and broncho-lobular pneumonia, each with distinct etiologic agent and morphologic changes.

#### **1. Lobar Pneumonia**

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.** Following types are described:

**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.

**2. Staphylococcal pneumonia.** *Staphylococcus aureus* causes pneumonia by haematogenous spread of infection.

**3. Streptococcal pneumonia,**  $\beta$ -haemolytic streptococci may rarely cause pneumonia such as in children after measles or influenza.

**4. Pneumonia by gram-negative aerobic bacteria.** Less common causes of lobar pneumonia are gram-negative bacteria like *Haemophilus influenzae*, *Klebsiella pneumoniae* (Friedlander's bacillus), *Pseudomonas*, *Proteus* and *Escherichia coli*.

**MORPHOLOGY.** Laennec's original description divides lobar pneumonia into 4 sequential pathologic phases:

**1. STAGE OF CONGESTION: INITIAL PHASE** The initial phase represents the early acute inflammatory response to bacterial infection and lasts for 1 to 2 days.

**G/A** The affected lobe is enlarged, heavy, dark red and congested. Cut surface exudes blood-stained frothy fluid.

**M/E** i) Dilatation and congestion of the capillaries in the alveolar walls.

ii) Pale eosinophilic oedema fluid in the air spaces.

iii) A few red cells and neutrophils in the intra-alveolar fluid.

iv) Numerous bacteria demonstrated in the alveolar fluid by Gram's staining.

**2. RED HEPATISATION: 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.

**G/A** The affected lobe is red, firm and consolidated. The cut surface of the involved lobe is airless, red-pink, dry, granular and has liver-like consistency.

**M/E** i) The oedema fluid of the preceding stage is replaced by strands of fibrin.

ii) There is marked cellular exudate of neutrophils and extravasation of red cells.

iii) Many neutrophils show ingested bacteria.

iv) The alveolar septa are less prominent than in the first stage due to cellular exudation.

**3. GREY HEPATISATION: LATE CONSOLIDATION** This phase lasts for 4 to 8 days.

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

**M/E** i) The fibrin strands are dense and more numerous.

ii) The cellular exudate of neutrophils is reduced due to disintegration of many inflammatory cells. The red cells are also fewer. The macrophages begin to appear in the exudate.

iii) The cellular exudate is often separated from the septal walls by a thin clear space.

iv) The organisms are less numerous and appear as degenerated forms.

**4. RESOLUTION:** This stage begins by 8th to 9th day if no chemotherapy is administered and is completed in 1 to 3 weeks. However, antibiotic therapy induces resolution on about 3rd day.

**G/A** The previously solid fibrinous constituent is liquefied by enzymatic action, 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.

**M/E** i) Macrophages are the predominant cells in the alveolar spaces, while neutrophils diminish in number. Many of the macrophages contain engulfed neutrophils and debris.

ii) Granular and fragmented strands of fibrin in the alveolar spaces are seen due to progressive enzymatic digestion.

iii) Alveolar capillaries are engorged.

iv) There is progressive removal of fluid content as well as cellular exudate from the air spaces.

**COMPLICATIONS.** 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 as under:

1. **Organisation.** In about 3% of cases, resolution of the exudate does not occur but instead it is organised. There is ingrowth of fibroblasts from the alveolar septa resulting in fibrosed, tough, airless leathery lung tissue.

2. **Pleural effusion.** About 5% of treated cases of lobar pneumonia develop inflammation of the pleura with effusion.

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.

5. **Metastatic infection.** 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.

**CLINICAL FEATURES.** The major symptoms are: shaking chills, fever, malaise with pleuritic chest pain, dyspnoea and cough with expectoration which may be mucoid, purulent or even bloody. The common physical findings are fever, tachycardia, and tachypnoea, and sometimes cyanosis if the patient is severely hypoxaemic. There is generally a marked neutrophilic leucocytosis. Blood cultures are positive in about 30% of cases. Chest radiograph may reveal consolidation.

## **II. Bronchopneumonia (Lobular Pneumonia)**

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 extremes of life (i.e. 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,

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

**G/A** 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 colour, 3 to 4 cm in diameter, slightly elevated over the surface and are often centred around a bronchiole. These patchy areas are best picked up by passing the fingertips on the cut surface.

**M/E** i) Acute bronchiolitis, ii) Suppurative exudate, consisting chiefly of neutrophils, in the peribronchiolar alveoli, iii) Thickening of the alveolar septa by congested capillaries and leucocytic infiltration, iv) Less involved alveoli contain oedema fluid.

**COMPLICATIONS.** 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.

**CLINICAL FEATURES.** 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 upper respiratory infection.

The salient features of the two main types of bacterial pneumonias are contrasted in Table:  
***Contrasting Features of Lobar Pneumonia and Bronchopneumonia.***

FEATURE	LOBAR PNEUMONIA	BRONCHOPNEUMONIA
<b>Definition</b>	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 in to adjoining alveoli
<b>Age group</b>	More common in adults	Commoner at extremes of age—infants and old age
<b>Predisposing factors</b>	More often affects healthy individuals	Preexisting diseases e.g. chronic debility, terminal illness, flu, measles
<b>Common etiologic agents</b>	Pneumococci, <i>Klebsiella pneumoniae</i> , staphylococci, streptococci	Staphylococci, streptococci, <i>Pseudomonas</i> , <i>Haemophilus influenzae</i>
<b>Pathologic features</b>	Typical case passes through stages of congestion (1-2 days), early (2-4 days) and late consolidation (4-8 days), followed by resolution (1-3 weeks)	Patchy consolidation with central granularity, alveolar exudation, thickened septa
<b>Investigations</b>	Neutrophilic leucocytosis, positive blood culture, X-ray shows consolidation	Neutrophilic leucocytosis, positive blood culture, X-ray shows mottled focal opacities
<b>Prognosis</b>	Better response to treatment, resolution common, prognosis good	Response to treatment variable, organisation may occur, prognosis poor
<b>Complications</b>	Less common; pleural effusion, empyema, lung abscess, organisation	Brochiectasis may occur; other complications same as for lobar pneumonia

## VIRAL AND MYCOPLASMAL PNEUMONIA (PRIMARY ATYPICAL PNEUMONIA)

Viral and mycoplasmal pneumonia is characterised by patchy inflammatory changes, largely confined to interstitial tissue of the lungs, without any alveolar exudate. Other terms used for these respiratory tract infections are interstitial 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 pneumonitis may occur in all ages.

**ETIOLOGY.** Interstitial pneumonitis is caused by a wide variety of agents, the most common being respiratory syncytial virus (RSV). Others are *Mycoplasma pneumoniae* and

many viruses such as influenza and parainfluenza viruses, adenoviruses, rhinoviruses, coxsackieviruses and cytomegaloviruses (CMV). **G/A** Depending upon the severity of infection, the involvement may be patchy to massive and widespread consolidation of one or both the lungs. The lungs are heavy, congested and subcrepitant. Sectioned surface of the lung exudes small amount of frothy or bloody fluid.

**M/E I) Interstitial Inflammation:** There is thickening of alveolar walls due to congestion, oedema and mononuclear inflammatory infiltrate comprised by lymphocytes, macrophages and some plasma cells.

illness, chronic debility, aspiration of gastric contents or upper respiratory infection.

ii) **Necrotising bronchiolitis:** This is characterised by foci of necrosis of the bronchiolar epithelium, inspissated secretions in the lumina and mononuclear infiltrate in the walls and lumina.

iii) **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.

vi) **Alveolar changes:** In severe cases, the alveolar lumina may contain oedema fluid, fibrin, scanty inflammatory exudate and coating of alveolar walls by pink, hyaline membrane similar to the one seen in respiratory distress syndrome.

**COMPLICATIONS.** The major complication of interstitial pneumonitis is superimposed bacterial infection and its complications. Most cases of interstitial pneumonitis recover completely.

**CLINICAL FEATURES.** Majority of cases of interstitial pneumonitis initially have upper respiratory 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. Chest radiograph may show patchy or diffuse consolidation.

## **C. OTHER TYPES OF PNEUMONIAS**

### **I. Pneumocystis carinii Pneumonia**

*Pneumocystis carinii*, a protozoon widespread in the environment, causes pneumonia by inhalation of the organisms as an opportunistic infection in neonates and immunosuppressed people. Almost 100% cases of AIDS develop opportunistic infection, most commonly *Pneumocystis carinii* pneumonia.

### **II. Legionella Pneumonia**

*Legionella pneumonia* or legionnaire's disease is an epidemic illness caused by gram-negative bacilli, *Legionella pneumophila* that thrives in aquatic environment. It was first recognised following investigation into high mortality among those attending American Legion Convention in Philadelphia in July 1976. The epidemic occurs in summer months by spread of organisms through contaminated drinking water or in air-conditioning cooling towers. Impaired host defenses in the form of immunodeficiency, corticosteroid therapy, old age and cigarette smoking play important roles.

### **III. Aspiration (Inhalation) Pneumonia**

Aspiration or inhalation pneumonia results from inhaling different agents into the lungs. These substances include food, gastric contents, foreign body and infected material from oral cavity. A number of factors predispose to inhalation pneumonia which include: unconsciousness, drunkenness, neurological disorders affecting swallowing, drowning, necrotic oropharyngeal tumours, in premature infants and congenital tracheo-oesophageal

fistula.

1. Aspiration of small amount of sterile foreign matter such as acidic gastric contents produce chemical pneumonitis. It is characterised by haemorrhagic pulmonary oedema with presence of particles in the bronchioles.
2. Non-sterile aspirate causes widespread bronchopneumonia with multiple areas of necrosis and suppuration.

#### **IV. Hypostatic Pneumonia**

Hypostatic pneumonia is the term used for collection of oedema fluid and secretions in the dependent parts of the lungs in severely debilitated, bedridden patients. The accumulated fluid in the basal zone and posterior part of lungs gets infected by bacteria from the upper respiratory tract and sets in bacterial pneumonia.

#### **V. Lipid Pneumonia**

Another variety of noninfective pneumonia is lipid pneumonia. It is of 2 types:

1. **Exogenous lipid pneumonia.** This is caused by aspiration of a variety of oily materials. These are: 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. **Endogenous lipid pneumonia.** 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 cancer, tuberculosis and bronchiectasis.

#### **LUNG ABSCESS**

Lung abscess is a localised area of necrosis of lung tissue with suppuration. It is of 2 types:

- Primary lung abscess that develops in an otherwise normal lung. The commonest cause is aspiration of infected material.
- Secondary lung abscess that develops as a complication of some other disease of the lung or from another site.

**ETIOPATHOGENESIS.** The microorganisms commonly isolated from the lungs in lung abscess are streptococci, staphylococci and various gram-negative organisms. These are introduced into the lungs from one of the following mechanisms: 1. Aspiration of infected foreign material. 2. Preceding bacterial infection. 3. Bronchial obstruction. 4. Septic embolism. 5. Miscellaneous (i) Infection in pulmonary infarcts, (ii) Amoebic abscesses, (iii) Trauma to the lungs. (iv) Direct extension from a suppurative focus.

**G/A** Abscesses may be of variable size from a few millimeters to large cavities, 5 to 6 cm in diameter. The cavity often contains exudate. An acute lung abscess is initially surrounded by acute pneumonia and has poorly-defined ragged wall. With passage of time, the abscess becomes chronic and develops fibrous wall.

**M/E** The characteristic feature is the destruction of lung parenchyma with suppurative exudate in the lung cavity. The cavity is initially surrounded by acute inflammation in the wall but later there is replacement by exudate of lymphocytes, plasma cells and macrophages. In more chronic cases, there is considerable fibroblastic proliferation forming a fibrocollagenic wall.