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Pneumonia

Peripneumonia was the term used by hippocrates in 4th century B.C., which was regarded as some inflammatory changes in lung. After the understanding of the anatomical and physiological concepts of respiratory system, Laennick in 1834 described three stages (stage of inflammatory congestion, Red hepatization and Grey hepatization) of lobar pneumonia. In the later part of 19th century Friedlander first found bacteria in the lung in a case of fatal pneumonia. Gramand Fraenkel, in 1984, isolated pneumococcus and subsequently various other organisms producing pneumonia were identified. Recently, in 1986, a new pathogen—chlamydia pneumonae—was isolated.

Etiology and Classification

Pneumonia or pneumonitis is an acute inflammatory reaction of lung parenchyma with outpouring of inflammatory exudate into the alveoli, resulting in respiratory and systemic symptoms. It is usually infective but may also be produced by inhalation of chemicals (gases, oily material, gastric aspiration), radiation, hypersensitivity reaction or as a result of parasitic infestation.

The types of pneumonia and the commonest etiological organisms involved are illustrated in table 10.1.

Radiological/anatomical classification as lobar pneumonia, segmental pneumonia and bronchopneumonia is mostly not useful clinically because none of them is specific for a particular organism. Another classification divides pneumonia into primary pneumonia (community acquired, normal host defence mechanism is disturbed by the invading micro organisms) and secondary pneumonia (when the defence mechanism is already disturbed because of pre-existing chronic lung diseases or systemic diseases). Classification according to etiological agent is also not useful because it does not specify the factor responsible for causation of pneumonia. Classification given in table 10.1 provides reference to the clinical circumstances under which the pneumonia developed. This will be more useful to guide for the investigations, management and therapy of the case. Specific pneumonias like tuberculosis, fungal infections, parasitic pneumonia and hypersensitivity pneumonitis will be described in the respective chapters.

Pathogenesis

Infective organisms can enter lung through inhaling airborne micro-organisms, aspiration of oropharyngeal secretions, bacteremia or direct extension into lung (amoebic liver abscess). It usually occurs when there is depletion of host defence mechanisms (physical defence of upper air passage, mucociliary apparatus, macrophages, surfactant and immunoglobulins). Larger particles (10 μm) are deposited in upper airway, medium size (5 - 10 μm) are deposited in tracheobronchial tree and small particles (0.3 - 5 μm) are deposited into the alveoli.

Table 10.3 Antimicrobial Therapy for Pneumonia.

Micro organisms	Drug of choice	Other drugs
• *Streptococcus pneumonae* • *Streptococcus pyogenes*	- Benzyl penicillin - Ampicillin - Sparfloxacin - Cefotaxine	- Erythromycin - Doxycycline - Clarithromycin - Azithromycin - Cephalexin - Ofloxacin - Cefaclor - Cefoperazone
• *Staphylococcus aureus*	- Benzyl Penicillin - Ampicillin - Sparfloxacin - Ticarcillin	- Erythromycin - Cefadroxil - Cefuroxime - Clarithromycin - Cefaperazone - Lomefloxacin - Azithromycin - Cephalexin - Clindamycin - Cefaclor
• *Klebsiella pneumonae*	- Cefotaxime with gentamycin	- Azithromycin - Ceftazidim - Cefoperazone - Cefaclor - Lomefloxacin - Cefadroxil
• *Haemophilus influenzae*	- Ampicillin	- Cotrimoxazol - Doxycycline - Chloramphenicol - Clarithromycin - Azithromycin - Sparfloxacin - Cephalexin - Cefaclor - Lomefloxacin
• *Branhamella catarrhalis*	- Cotrimoxazole - Amoxycillin + Clavulanic acid	- Erythromycin - Tetracycline - Azithromycin - Roxithromycin - Clarithromycin - Lomefloxacin
• *Legionella pneumophila*	- Erythromycin - Clarithromycin	- Rifampicin - Doxycycline - Cotrimoxazole - Tetracycline
• *Mycoplasma pneumonae*	- Oxy-Tetracycline	- Erythromycin - Azithromycin - Roxithromycin - Clarithromycin

Now-a-days this classical form of lobar pneumonia is rare because of frequent use of antibiotics and, therefore, sputum is usually not specific in colour which may be yellow or green with or without blood.

Clinically, there is diminished movement of the affected side, reduced breath sound, dullness on percussion and fine crackles. This is followed by findings of consolidation with bronchial breathing. Pleural rub may be heard initially which disappears after development of effusion.

Radiologically, there is a homogeneous opacity with air bronchogram. The opacity may be patchy or may present like broncho-pneumonia (Fig. 10.1), specially in children and in patients with chronic bronchitis and emphysema.

Appropriate antibiotic therapy (table 10.3 & 10.4), analgesics for pleural pain, oxygen in hypoxemic patients and general supportive therapy, as described in this chapter, should be initiated.

The recovery is usually complete with appropriate treatment in 7-10 days time. However, some times it may be complicated mainly because of delay in start of treatment. Bacteraemia is the commonest and dangerous complication. Other complications include pleural effusion, empyema, lung abscess, pericarditis, endocarditis, peritonitis, meningitis collapse, bronchiectasis and septic arthritis (Fig. 10.2).

There are chances of mortality specially in older persons, if following features are present :

1. Respiratory rate > 30/mt, Diastolic blood pressure < 60 mm of Hg.
2. Patient is confused or delirious.
3. Presence of signs of Uraemia.
4. Hypoxaemia (Pa O2 < 60 mm of Hg).
5. Leucocytosis (30,000 mm3) or leucopenia (< 4000 mm3).

These patients may require oxygen inhalation or even assisted mechanical ventilator for a short period.

Staphylococcal Pneumonia

It is produced by *staphylococcus aureus*. Commonly it is a secondary infection, but can even present as primary infection as a result of haematogenic spread from a staphylococcal infection elsewhere. It commonly follows influenza epidemics and the spread of infection is usually bronchial rather than lobar. The damage to bronchial and bronchiolar walls may lead to bronchiectasis.

The clinical presentation is more or less same as pneumococcal pneumonia, but the pleural involvement is common and there is usually no haemoptysis or rusty brown sputum. There may be presence of multiple pneumatocoeles (thin walled air containing cavities). The clinical examination shows multiple areas of consolidation and marked diminution of air entry over the pneumatocoeles. Signs of pleural effusion and signs of shock may also be present.

Disease is usually bilateral with patchy areas of consolidation, usually leading to cavitation in adults and pneumatocoeles or empyema in children. Pneumatocoele can be very big in size and can even occupy whole of hemithorax simulating tension pneumothorax. Pleural effusion is usually present.

Treatment should be initiated with appropriate antibiotics (Tables 10.3 & 10.4) with supportive therapy (as described in the previous chapter). The organism is usually resistant to a variety of antibiotics and therefore combination therapy is preferred. Complications are common in the form of lung abscess, empyema, bronchiectasis, pneumothorax and bacteraemia leading to endocarditis. Endotoxic shock is a term for generalised capillary leak due to bacteraemia in staphylococcal pneumonia.

Klebsiella Pneumonia (Friedlander's Pneumonia)

Klebsiella pneumonae (or Friedlander pneumonae) and Klebsiella oxytoca are the causative organisms which are non-motile gram negative bacilli of enterobacteriaceae family. It is a more important pathogen causing hospital acquired pneumonia and is less common in community. It is the commonest among all gram negative pneumonias.

Onset is acute with prostration, fever,

and is usually resistant to tri-methoxaprim and Ampicillin. Therefore, monotherapy with these antibiotics is not recommended.

Hospital Acquired (Nosocomial) pneumonia

Pneumonia devloped in a patient within 48 hours of admission in the hospital is labelled as hospital acquired pneumonia.

Impairment of mechanical defences, mucociliary apparatus and disturbance of surfactants lead to development of bacterial pneumonia (aspiration pneumonia). Immune supression due to chemotherapy for malignant disease, drugs used to prevent the rejection of transplanted organ and drugs for the treatment of fibrotic or collagen vascular lung disorders also lead to development of pneumonia with opportunistic pathogens (pneumonia in immunocompromised patients).

In healthy persons, inhalation of oro-pharyngeal secretions specially during sleep does not produce pneumonia except in persons belonging to the following categories :-

1. Failure of cough reflex : unconscious patient, alcoholics, narcotic overdose, head injury and in patients under general anaesthesia.
2. Patient with endotracheal tube while on mechanical ventilator.
3. Neuromuscular disorders : infective polyneuritis, myopathy, muscular dystrophy and in cases of chronic bronchitis and emphysema having expiratory muscle weakness.
4. Oesophageal obstruction : carcinoma, stricture, achlasia of oesophagus and achlorhydria due to drugs leading to vomiting and aspiration.
5. Bacteraemia : due to abdominal sepsis or infection during I.V. infusions.

Other factors which impair respiratory defence mechanisms include old age, obesity, diabetes, immunosuppression, granulocytopaenia, poor nutrition, etc. The aspirated material includes bacteria (from oropharynx), gastric acid (from regurgitation of gastric contents), inert liquid (drowning) and foreign body.

Pseudomonas Pneumonia

It is caused by *pseudomonas aeruginosa* previously known as *pseudomonas pyocyanea*, a gram -ve aerobe. It produces characteristic greenish pigment, 'pyoyanin', and produces various toxins and endotoxins which may even lead to bacteremia and shock. This organism is found normally in water, soil and vegetable materials. In hospital atmosphere, it is found in abundance in sinks, nebulizers and mechanical ventilators. The aspiration of large amount of organisms leads to development of pneumonia.

Clinical features include fever with rigor, cough with copious green coloured sputum and severe dyspnoea. Few cases present with bacteremia depending upon situation of underlying lung, specially those having neutropaenia, may even lead to pulmonary oedema. Radiological picture is that of nodular infiltration which afterwards leads to bilateral diffused bronchopneumonia with patchy areas of consolidation leading to small micro-abscesses. Nodular bronchopneumonia limited to lower lobe can also be seen. Pleural effusion may occur but empyema is uncommon. Mortality is high if not treated properly. Appropriate antibiotic, usually more than one, WBC transfusion in neutropenic patients with general supportive therapy is indicated.

Escherichia coli pneumonia

It is a gram -ve enteric bacteria of enterobacteraecea family. It is aerobic but also grows in anaerobic conditions. The spread of disease is usually haematogenous from gut and urinary tract. Clinical presentation is like other bacterial pneumonia but sputum may be absent. Clinical features of urinary tract or gastrointestinal infection may be present. Radiologically, there is bilateral patchy areas of consolidation. Occasionally, it may be complicated with empyema.

Proteus pneumonia

Proteus species produces pneumonia mainly due to aspiration in chronic alcoholics and persons having underlying chronic lung disease. The commonest organisms include *proteus mirabilis* and *proteus vulgaris*. The