Pulmonary infections

Pneumonia is an inflammatory process of infectious origin affecting the pulmonary parenchyma

**Classification of pneumonia :**

I- community – acquired acute pneumonia.

This type is caused by :

* *Streptococcus pneumoniae*
* *Haemophilus influenzae*
* *Legionella pneumophila*
* … etc.

II- community – acquired atypical pneumonia

This type is caused by :

* *Mycoplasma pneumoniae*
* *Chlamydia spp. (Chlamydia pneumonia )*
* *Viruses: ( like respiratory syncytial virus and parainfluenza virus )*

III- Nosocomial Pneumonia

* *Staphylococcus aureus .*
* *Gram – negative rods ( Klebsiella spp. E.coli ) and pseudomonas spp.*

IV- aspiration pneumonia:

This type is caused by :

*Anaerobic oral flora like Bacteroides admixed with aerobic bacteria like Streptococcus pneumonia , Staphylococcus aureus.*

*V- chronic pneumonia*

* *Nocardia*
* *Actinomyces*
* *Mycobacterium TB*
* *Fungal infection*

*VI- necrotizing pneumonia and lung abscess:*

*Staphylococcus aureus , Klebsiella pneumonia , streptococcus pypgen .*

*VII- pneumonia in immunocompromised host*

*This type is caused by*

* *Cytomegalovirus*
* *Pneumocystic carini*
* *… etc.*

***Community-Acquired Acute pneumonias. ( CAAP)***

*CAAP are bacterial in origin, the infection may follow viral upper respiratory tract infection.*

*Streptococcus pneumonia is the most common cause of CAAP*

*Pneumococcal pneumonia will be discussed as a prototype of this group*

*pneumococcal pneumonia occurs with increased frequency in three groups of individuals:*

*1- those with underlying chronic diseases such as congestive heart failure, COPD and DM .*

*2- Patients with congenital or acquired immunoglobulin deficiency like AIDS*

*3- Patients with decreased or absent splenic function (eg. Sickle cell disease).*

***Clinical Features :-***

*The onset is usually abrupt, with high fever , shaking chills , pleuritic chest pain, and productive mucopurulent cough . occasional patients may have heamoptysis.*

*V- chronic pneumonia*

* *Nocardia*
* *Actinomyces*
* *Mycobacterium TB*
* *Fungal infection*

*VI- necrotizing pneumonia and lung abscess:*

*Staphylococcus aureus , Klebsiella pneumonia , streptococcus pypgen .*

*VII- pneumonia in immunocompromised host*

*This type is caused by*

* *Cytomegalovirus*
* *Pneumocystic carini*
* *… etc.*

***Community-Acquired Acute pneumonias. ( CAAP)***

*CAAP are bacterial in origin, the infection may follow viral upper respiratory tract infection.*

*Streptococcus pneumonia is the most common cause of CAAP*

*Pneumococcal pneumonia will be discussed as a prototype of this group*

*pneumococcal pneumonia occurs with increased frequency in three groups of individuals:*

*1- those with underlying chronic diseases such as congestive heart failure, COPD and DM .*

*2- Patients with congenital or acquired immunoglobulin deficiency like AIDS*

*3- Patients with decreased or absent splenic function (eg. Sickle cell disease).*

***Clinical Features :-***

*The onset is usually abrupt, with high fever , shaking chills , pleuritic chest pain, and productive mucopurulent cough . occasional patients may have heamoptysis.*

**Morphology:-**

There are 2 morphological pattern:-

a- labor pneumonia .

b- Bronchopneumonia. (is much more prevalent at extremes age) .

Regardless the morphologic pattern , the lower lobes or the right middle lobe are most frequently involved.

**a- lobar pneumonia** which is most frequent is caused by streptococcus Pneumoniae, It involve the entire lobe. Consolidation can be seen by X-ray.

Microscopically + grossly

in preantibiotic era, it evolved through 4 stages:-

1-congestion: - The affected lobe is heavy, red and baggy.

histologically: - vascular congestion can be seen, with proteinaceous Fluid, scattered neutrophils and many bacteria in the alveoli

2-Red hepatization: - in which the lung has a liver like consistency, the alveolar spaces are packed with neutrophils, red cells and fibrin and the pleura shows fibrino or fibrinopurulent exudate

3-Grey hepatization; - the lung is dry, gray and firm because the red cells become lysed while fibrinous exudate persists within alveoli.

4- resolution: follows in uncomplicated cases, as exudates within the alveoli are enzymatically digested and either resorbed or expectorated leaving basic architecture intact. the pleural reaction, leaving fibrous thickening or permanent adhesions

**b- Bronchopneumonia:-**

foci of inflammation and consolidation are distributed in patches throughout one or several lobes, the lung area or substances immediately surrounding areas of consolidation is usually hyperemic and edematous, but the large intervening areas are generally normal

pleural involvement is less common than in lobar pneumonia

Histologically : the reaction consist of focal Suppurative exudate that fills the bronchi, bronchioles and adjacent alveolar spaces.

With appropriate therapy, complete restitution of lung is the rule for both forms of pneumonia.

**Complications:-**

1- tissue destruction and necrosis may lead to abscess formation

2- Suppurative material may accumulate in the pleural cavity, producing empyema.

3- Organization of the intra alveolar exudate may convert areas of the lung into solid fibrous tissue.

4- Bacteremic dissemination may lead to Meningitis, arthritis or infective endocarditis

Complication are much more likely with serotype 3 pneumococci.

**Community-Acquired Atypical pneumonia:-**

caused by Mycoplasma pneumonia (Interstitial pneumonia)

this is the most common form of interstitial pneumonia it usually occurs in children and young adults. The onset is more insidious compared to bacterial pneumonia. And usually follows a mild self-limited course.

Microscopically:

inflammatory reaction confined to the interstitium with no exudate in alveolar spaces, and intra-alveolar hyaline membrane.

Mycoplasma may be associated with cold agglutinin reaction to red cells which can provide early diagnosis.

**Tuberculosis (TB)**

TB is a communicable chronic granulomatous disease caused by Mycobacterium tuberculosis it usually involves the lungs but may affect any organ or tissue.

**((Immune Mechanisms in pathogenesis of TB.)):**

a- the organism are ingested by macrophages which process the bacterial antigens for presentation to CD4 TH1 T cells in the context of class II MHC molecules

b- The CD4+ T cells proliferate and secrete cytokines , attracting lymphocytes and macrophages.

IFN- γ released by CD4 + T cell is critical in activating macrophages

c- Activated Macrophages release a variety of mediators:

1-TNF is responsible for recruitment of monocytes which in turn undergo activation and differentiation into epithelioid histiocytes

2- IFN- γ in conjunction with TNF result in increased level of nitric oxide at site of infection , Nitric oxide is a powerful oxidizing agent and results in generation of free radicals capable of destruction of several Mycobacterial constituents.

d- CD4 T cells facilitate the development of CD8 cytotoxic T cells and kill TB infected macrophages.

e- Delayed hypersensitivity is marked by a positive tuberculin skin test result. The test result is positive in both primary and secondary infect on represents hypersensitivity and relative immunity and usually remains positive throughout life.

**Types of TB:**

**1° (Primary) TB**

is the form of disease that develops in a previously unexposed, Elderly and profoundly immunosuppressed persons may lose their sensitivity to tubercles and so may develop tuberculosis more than once.

With primary TB source of organism is exogenous, 5% only develop significant disease

**Morphology:-**

Gross:-

primary TB almost always begins in lungs. Typically the inhaled bacilli in distal airways of lower part of upper lobes or upper part of lower lobes. Gray white consolidation emerges which called Ghon focus, this focus undergoes caseous necrosis. The TB bacilli either free or within phagocytes drain to regional LN which is also caseate → Ghon focus + Regional LN complex = (Ghon complex). Which is a characteristic of primary TB.

Ghon complex undergoes fibrosis + calcification that can be detected by X-ray (Ranke complex)

Microscopically

granulomatous inflammatory reaction that forms both caseating and non caseating tubercles the granulomas are usually enclosed within a fibroblastic rim punctuated by lymphocytes

Multinucleated giant cells are present in granuloma.

**Fate of primary TB**

1- induce hypersensitivity and increased resistance

2- The foci of scarring may harbor viable bacilli for years perhaps for life.

3- Uncommonly the disease may progress without interruption into so called progressive TB because of well-defined disease such as AIDS.

Diagnosis of PPTB in adult is difficult. PPTB resembles pneumonia

lymphatohaematogenous dissemination may develop → TB meningitis and miliary TB

**2- Secondary TB (reactivation TB)**

Is the pattern of disease that arises in previously sensitized host, it may follow shortly after 1°TB, but more commonly it arises from reactivation of dormant 1° lesions many decades after initial infection when host resistance is weakened or it may results from exogenous reinfection.

2° TB is classically localized to the apex of one or both upper lobes , this is may be due to high oxygen tension

because of preexisting hypersensitivity, the bacilli excite a prompt and marked tissue response that tends to wall off the focus so there's no LN involvement

Cavitation occurs resulting in dissemination along airways in neglected 2° TB . So the erosion along the airway is an important source of infectivity because the patient raises sputum containing bacilli.

**Morphology:-**

a- localized lesions usually in the apical or posterior segments of upper lobes.

b- Tubercle formation:-The lesions frequently coalesce and rupture into the bronchi

The Caseous content may liquefy and be expelled resulting in cavitory lesions . Cavitations is a characteristic of 2° TB (not 1° TB).

c- Scarring and calcification

**Spread of disease of 2nd TB:-**

1-Miliary TB: - which is seeding of distal organs with innumerable small millet seed like lesions by lymphatic and haematogenous routes, this is may be pulmonary miliary TB or systemic.

2- Endobronchial, endotracheal and laryngeal TB develop either by spread of infective material through lymphatic channels or from expectorated infectious material.

3-Isolated organ TB:-

This is occur by haematogenous spread to any organ and may the presenting feature, most common sites meninges, kidneys, adrenals, bone, Para spinal abscess or Pott disease, and fallopian tube.

4- Lymphadenitis especially cervical LN

lymph adenopathy is unifocal and the patient don't have the ongoing disease.

5-Intestinal TB:-

due to ingestion of contaminated milk or secondary to swallowing of coughed up infective materials in 2° TB .Typically the organisms are trapped in mucosal lymphoid aggregations of the small and large bowel which undergo inflammatory enlargement and ulceration especially in the ileum

**Clinical Features**

Localized secondary TB may be asymptomatic when manifestation appear they are usually insidious. There are :

1- systemic symptoms related to (TNF and IL1)

Malaise, anorexia, weight loss and fever (remittent low grade) and night sweat.

2- localized symptoms (with progressive pulmonary TB)

- increase amount of sputum first mucoid then purulent.

- Haemoptysis (in 1/2 of patients).

- Pleuritic chest pain

in extrapulmonary TB the symptoms depend on the organ that is involved

E.g.: Fallopian tube → infertility

meninges → neurologic deficit.