***Dr. Nadia Hameed***

***Respiretory System-2***

***Pulmonary infection***: it includes upper and lower respiratory tract infection.

1. Upper respiratory tract infection URTI include common cold, flu, sinusitis, laryngitis,…..
2. Lower respiratory tract infection LRTI:
3. include infection of airways from trachea and below
4. infection of lung parenchyma (pneumonia)

Acute LRT infectious diseases include *pneumonias, lung abscess* and *fungal infections.* *pulmonary tuberculosis* is regarded as chronic lung infections.

Defect in humoral immunity leads to an increase incidence of infections with pyogenic bacteria, whereas cell mediated immune defects lead to increase infection with intracellular microbes (mycobacteria and viruses eg herpes.

**Pneumonia** Pneumonia is one of the common causes of death. is defined as acute inflammation of LRT (the lung parenchyma) distal to the terminal bronchioles. Consolidation (meaning solidification) is the term used for gross and radiologic appearance of the lungs in pneumonia.

***Pathogenesis and predisposing factors:***

The microorganisms or the injurious materials gain entry into the lungs by one of the following four routes: *Inhalation, aspiration, haematogenous spread* and *direct spread*.

Predisposing factors. These conditions are:

1. Loss or **suppression of cough reflex** (coma, anesthesia), this May 🡪 gastric aspiration.
2. Injury to the **mucociliary apparatus** e.g. cigarette, inhaled hot or corrosive gases, viral diseases or genetic diseases (immotile cilia syndrome).
3. Interference with **phagocytosis.**
4. Pulmonary congestion and **pulmonary edema**.
5. **Accumulation of secretion** e.g. cystic fibrosis& bronchial obstruction.

CLASSIFICATION.

Etiologic classification which divides pneumonias into following 3 main groups:

A. Bacterial pneumonia (community acquired pneumonia)

B. Viral and atypical pneumonia

C. Pneumonias from other etiologies eg aspiration pneumonia

**BACTERIAL PNEUMONIA (community acquired pneumonia)**

Two types of acute bacterial pneumonias are distinguished

1. lobar pneumonia (community acquired pneumonia)
2. bronchopneumonia, (lobular pneumonia)

Each is with distinct etiologic agent and morphologic changes.

**1- Lobar bacterial Pneumonia (community acquired pneumonia)**

Lobar pneumonia is an acute bacterial infection of a part of a lobe or the entire lobe. The consolidation involves a **portion of a lobe or the whole lobe.**

**ETIOLOGY.**

**1. Pneumococcal pneumonia.** More than 90% of all lobar pneumonias are caused by *alpha hemolytic Streptococcus pneumoniae,( pneumococcus pneumonae)* a lancet-shaped diplococcus bacteria. Pneumococcal pneumonia in majority of cases is community-acquired infection.

**2. Staphylococcal 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 abusers* are at high risk of developing staphylococcal pneumonia by haematogenous spread.
* It is also an important cause of nosocomial pneumonia.

**3. Streptococcal pneumonia.** β-haemolytic 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 *Haemophilus influenzae,* *Klebsiella pneumoniae, Pseudomonas, Proteus* and *Escherichia coli, H. influenza*. They cause life-threatening pneumonia infections in children. In adults it is a very common cause of community-acquired acute pneumonia.

***Morphology of lobar pneumonia:***

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

1. Stage of congestion
2. Stage of red hepatization
3. Stage of grey hepatization
4. Stage of resolution.

**A-Congestion stage:**

**Grossly:** the lung is heavy and red in color.

**Mic.:** Alveolar vascular congestion & Intra-alveolar fluid with **neutrophil + bacteria.**

**B-Red hepatization:**

**Grossly:** red, firm, airless, look like a liver.

**Mic.:** The inflammatory exudate composed of **RBC+ neutrophil+ fibrin.**

**C-Grey hepatization:**

**Grossly:** grey –brownish, dry surface.

**Mic.:** The exudate is composed of **fibrin+ WBC** which is called (fibrino-suppurative) exudate.

**D-Resolution stage:**

The exudate undergoes **enzymatic digestion**🡪 formation of granular debris that is either resorbed & ingested by the macrophages or expectorated and coughed up.

**2- Bronchopneumonia (Lobular Pneumonia)**

Bronchopneumonia or lobular pneumonia is infection of the terminal bronchioles that extends into the surrounding alveoli

* Caused by staphylococcus, streptococcus, pneumococcus, hemophilus influenzae.
* The consolidation is ***patchy*** due to patchy distribution of inflammation that generally involves more than one lobe.
* Occur in ***infancy and old*** age groups caused by low immune resistance resistance (eg steroid therapy, diabetes, starvation,,,).
* As complication of pulmonary diseases
* Can complicate long term heart failure

***Morphology of Bronchopneumonia***

**Grossly:** the well-developed lesion is 3-4 cm, red /yellow, slightly elevated.

**Mic.:** The consolidating area shows acute ***suppurative*** inflammation rich in neutrophils, filling the bronchi, bronchioles, & adjacent alveolar spaces.

***Clinical presentation of pneumonia* it is life threatening infection**

* Fever
* Malaise
* Pleuritic chest pain
* Productive mucopurulent cough and hemoptysis in half of cases.
* Clubbing of the fingers and toes appears in about 20% of patients.
* All these will **improve** within 48-72 hours of antibiotic administration.

***Complications pneumonia:***

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

* **Abscess** formation, especially if the m.o is Klebsiella and pneumococcal infection.
* Spread of infection to the pleural cavity🡪 **empyema** (pus inside the pleural cavity).
* **Organization** of the exudate 🡪 part of the lobe will turn solid.
* **Bacteremic** dissemination to: heart valves, brain, pericardium, kidney.
* **RDS**

**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. It was called *atypical pneumonia,* due to the absence of alveolar exudate commonly present in other pneumonias.

**ETIOLOGY.** Interstitial pneumonitis is caused by a wide variety of agents,

1. *respiratory syncytial virus* (RSV) the most common one
2. *Mycoplasma pneumoniae*
3. influenza and parainfluenza viruses, adenoviruses, rhinoviruses, coxsackieviruses and cytomegaloviruses (CMV). Occasionally,
4. psittacosis *(Chlamydia)* and Q fever *(Coxiella)* are associated with interstitial pneumonitis.

**Pneumonia from other causes: Aspiration (Inhalation) Pneumonia**

Aspiration or inhalation pneumonia results from inhalation of 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.

***LUNG ABSCESS***

Lung abscess is a localised area of necrosis of lung tissue with suppuration.

It is of 2 types:

1. *Primary lung abscess* that develops in an otherwise normal lung. The commonest cause is aspiration of infected material.
2. *Secondary lung abscess* that develops as a complication of some other disease of the lung eg preceding bacterial infection.

The microorganisms commonly isolated from the lungs in lung abscess are streptococci, staphylococci and various gram negative organisms.

***FUNGAL INFECTIONS OF LUNG***

Fungal infections of the lung are more common than tuberculosis in the US.

These infections in healthy individuals are rarely serious but in immunosuppressed individuals may prove fatal.

**1. Aspergillosis.** Aspergillosis is the most common fungal infection of the lung caused by *Aspergillus fumigatus* that grows best in cool, wet climate.

Immunocompromised persons develop more serious manifestations of aspergillus infection, especially in leukaemic patients on cytotoxic drug therapy and HIV/AIDS.

**2. Candidiasis.** Candidiasis or moniliasis caused by *Candida albicans* is a normal commensal (normal flora) in oral cavity, gut and vagina but attains pathologic form

in immunocompromised host.

**3. Histoplasmosis.** It is caused by oval organism, *Histoplasma capsulatum,* by inhalation of infected dust or bird droppings.

**4. Cryptococcosis.** It is caused by *Cryptococcus neoformans* which is round yeast heavily encapsulated. The infection occurs by inhalation of pigeon droppings.

**Tuberculosis** **TB**

Tuberculosis is chronic infectious disease caused by *Mycobacterium tuberculosis*, arod-shaped aerobic bacteria. Tuberculosis is the world’s foremost cause of death from a single infectious agent, causing 25% of avoidable deaths in developing countries.

■ The organism is spread by inhaling the mycobacterium-containing droplet that circulate in the air. Mycobacterium bovis can be obtained by ingestion of contaminated mik.

■ The cell-mediated response plays a dominant protective role preventing the development of active tuberculosis. People with impaired cell-mediated immunity are more likely to experience active tuberculosis when infected.

■ A positive tuberculin skin test results from previous infection or vaccine.

**We have two types of TB:**

**I//Primary Tuberculosis**

* Primary tuberculosis occurs in a person lacking previous contact with the tubercle bacillus.
* Acquired by inhaling droplet that contains the tubercle bacillus.
* Affect the lower lobes.
* Soon after entering the lung, the bacilli are surrounded and engulfed by macrophages and gray-white, circumscribed granuloma formed. This granulomatous lesion, called a *Ghon’s focus*, that contains the tubercle bacilli, modified macrophages (epitheloid and multinucleated giant cells, and other immune cells.
* tubercle bacilli, free or inside macrophages, drain along the lymph channels to the hilar lymph nodes of the affected lung and there evoke the formation of lymph nodes caseous granulomas.
* The combination of the primary lung lesion and lymph node granulomas is called *Ghon’s complex* .

**Outcome of primary TB:**

1. Primary tuberculosis usually is asymptomatic, with the only evidence of the disease being a positive tuberculin skin test result and calcified lesions seen on the chest radiograph.
2. In immune compromised patients, primary tuberculosis may progress, causing more extensive destruction of lung tissue and spread within the lung or to the other body organs (military TB).

**II//Secondary Tuberculosis**

* Secondary tuberculosis represents either reinfection from inhaled droplet as in endemic areas or reactivation of a previously healed primary lesion (only 5% of primary TB develops secondary TB in developed countries).
* It often occurs in situations of impaired body defense mechanisms.
* It is classically localized to the apex of one or both upper lobes.
* The regional Lymph nodes involvement is less prominently than they are in primary TB
* Cavitation occurs which leads to erosion of airways and this converts the patient into source of infection to others.

**PULMONARY HYPERTENSION**

Normally, the pulmonary arterial circulation is high-flow and low-pressure system with much lower blood pressure than the systemic blood pressure; it does not exceed 30/15 mmHg even during exercise (normally, blood pressure in the pulmonary veins is between 3 and 8 mmHg). Pulmonary hypertension is defined as a systolic blood pressure in the pulmonary arterial circulation above 30 mmHg. Pulmonary hypertension is broadly classified into 2 groups:

**I/Primary (Idiopathic) Pulmonary Hypertension**

**II/Secondary Pulmonary Hypertension**

When pulmonary hypertension occurs secondary to a recognized lesion in the heart or lungs

**ETIOPATHOGENESIS.** It includes following:

**A. Passive pulmonary hypertension.** This is the commonest and is produced by diseases raising pressure in the pulmonary veins e.g.

1. Mitral stenosis.

2. Chronic left ventricular failure (e.g. in severe systemic hypertension, aortic stenosis, myocardial fibrosis).

**B. Hyperkinetic (Reactive) pulmonary hypertension.** In this group are included causes in which the blood enters the pulmonary arteries in greater volume or at a higher pressure, e.g. Atrial or ventricular septal defects.

**C. Vaso-occlusive pulmonary hypertension.** All such conditions which produce progressive diminution of the vascular bed in the lungs are included in this group. Vaso-occlusive causes may be further sub-divided into 3

types:

1. *Obstructive type* e.g. i) Multiple emboli or thrombi, ii) Sickle cell disease

and iii) Schistosomiasis.

2. *Obliterative type,* e.g. i) Chronic emphysema, ii) Chronic bronchitis, iii)

Bronchiectasis, iv) Pulmonary tuberculosis and v) Pneumoconiosis.