### SELECTED HUMAN INFECTIOUS DISEASES

#### Viral Respiratory infections

These are the most frequent of all infections but the least preventable. They range from common cold to life-threatening pneumonia. Virus infection may be complicated by secondary bacterial infections e.g. Pneumococcus, Staphylococcus & Hemophilus.

**Upper respiratory tract infections** include Rhinitis, Sinusitis, Otitis media, Pharyngitis and Tonsillitis.

**Lower respiratory tract infections** include Laryngeotracheobronchitis, Bronchiolitis, Interstitial pneumonia and Pleuritis.

Rhinoviruses & influenza viruses are the most important & the best studied.

### **Rhinoviruses**

They are responsible for common cold. They are RNA viruses. The virus attaches to respiratory epithelial cells especially upper respiratory tract. This is because they grow best at 33-35 C<sup>0</sup>. It induces acute inflammation with excessive mucus secretion characteristic of common cold. This virus has >100 serotypes. It also escapes antibody attacks.

### Influenza virus

This is RNA virus. It is of three types A, B & C. the spherical surface of the virus is a lipid bilayer containing hemagglutinin (H) and neuraminidase (N) which determine the subtype (serotype).

**The pandemic** that occurred in 1918 was because of both hemagglutinin and neuraminidase was replaced through recombination of RNA segment with those of animal virus making all people susceptible to the new influenza virus (antigenic shift).

**Epidemics,** which are less severe and more localized in distribution, occur through mutations of hemagglutinin and neuraminidase that allow the virus to escape most host antibodies (antigenic drift).

A particular subtype of **avian influenza** ("**bird flu**," **H5N1**) has caused massive outbreaks in domesticated poultry in parts of Southeast Asia in the last few years; this strain is particularly dangerous, since it has the potential to "jump" to humans and thereby cause an unprecedented, worldwide influenza pandemic.

**The swine flu** 2009 outbreak is thought to be a mutation of four known strains of influenza A virus subtype H1N1: one endemic in humans, one endemic in birds, and two endemics in pigs (swine). The signs of infection with swine flu are similar to influenza. **People at higher risk of serious complications include** 1-people age 65 years and older

2-Children younger than 5 years old

3- Pregnant women

4- People of any age with chronic medical conditions (such as asthma, diabetes, or heart disease)

5- People who are immunosuppressed.

Transmission is through Sneezes or coughs, and contaminated objects (touching something with flu viruses on it and then touching your mouth or nose). Influenza viruses are not known to be transmissible to people through eating processed pork or other food products derived from pigs."

**Morphologically** there is upper respiratory tract mucosal hyperemia, and swelling leading to rhinitis. The condition may be complicated by sinusitis, otitis media, pharyngitis, tonsillitis, laryngo-tracheo-bronchitis, lower respiratory tract infections (bronchiolitis) and Streptococcal pneumonia.

## **Bacterial Respiratory Infections**

#### **Bacterial pneumonia**

An important infectious disease, which is an immediate cause of death in hospitalized patient

Common microorganisms include: *Streptococcus pneumonia*, *Haemophilus influenzae*, and *Staphylococcus aureus*.

### Hemophilus influenzae infection

*Hemophilus influenzae* is a gram-negative organism. It is a major cause of life threatening acute lower respiratory tract infections as well as meningitis in young children.

## **Tuberculosis**

This is caused by **mycobacterium tuberculosis** and **Mycobacterium bovis**. These organisms infect about 1/3 of the world population and kill about 3 million patients each year.

Mycobacterium tuberculosis is transmitted by inhalation of infective droplets coughed into the air by the infected patients. Mycobacterium bovis is transmitted through milk of diseased cows.

Mycobacteria are aerobic bacilli with a waxy coat that causes them to retain the red dye when treated with acid. That is why they are called Acid Fast Bacilli (AFB). They grow very slowly in culture (4-6 weeks).

Mycobacterium tuberculosis has no known exotoxins, endotoxin or histolytic enzymes. It escapes killing by being inside macrophages. It induces a delayed type hypersensitivity reaction which explains destruction of tissues and emergence of resistance to the organism.

Mycobacterium tuberculosis stimulates macrophages to secrete TNF- $\dot{\alpha}$ , which causes fever & tissue damage.

On the initial exposure to the microorganisms there is a nonspecific inflammatory response that resembles inflammation to any form of bacteria, but 2-3 weeks later the reaction becomes granulomatous and the center becomes caseous, forming typical tubercle. The pattern of host response depends on whether infection represents a primary (1st exposure) or secondary reaction in an already sensitized host.

### Primary tuberculosis

Infection begins with inhalation of the mycobecteria to the periphery of the lung. *M. tuberculosis* enters alveolar macrophages, once inside the macrophage; *M. tuberculosis* replicates within the phagosome by blocking fusion of the phagosome and lysosome, and eventually lyse the host cells and infect other macrophages, which will transport the bacilli to the hilar lymph nodes. After few weeks, T- cell mediated immunity develops,  $CD_4$  + (helper) secrete interferon gamma, which activate macrophages to kill the bacilli. This occurs in association with granuloma formation.  $CD_{8+}$  T cells kill infected macrophages resulting in caseation. So control of 95% of the infection occurs. Later on, calcified scar in the lung parenchyma and hilar lymph nodes occur and this is called **Ghon complex** 

## Secondary and Dissminated tuberculosis

Some individuals either become re-infected or there is reactivation of dormant bacilli in them from the primary infection. Progression can occur directly from primary lesions into disseminated disease (due to high virulence of the T.B. bacilli, or because the patient is particularly susceptible).

Granulomas in secondary T.B. occur at the apex of lung. Disseminated granulomas occur in lungs, kidneys, meninges, bone marrow and other organs. Caseous necrosis may be followed by cavitation with rupture into blood vessels. This results in spreading of Mycobacteria throughout the body and into airways. Hematogenous dissemination of the bacilli throughout the body may lead to what is called miliary tuberculosis in various organs (from millet seeds –small yellow-white lesions).

### **Clinical features**

Fever, sweating, loss of weight, cough with sputum, hemoptysis, shortness of breath, etc

*Diagnosis* is through examination of the sputum for AFB or culture. Nowadays PCR (detection of the T.B. bacilli DNA) is proving to be a rapid, sensitive and accurate way of diagnosis.

# Actinomycosis:

it is a localized but gradually spreading chronic suppurative disease affecting particularly the lower jaw (the condition known as- lumpy jaw), the ileocaecal region in the bowel and occasionally the lung.

The causative organism is *Actinomyces israelii*, wide spread in nature gram positive, branching filamentous anaerobes found as normal commensals in mouth, gut and female genital tract. The disease most commonly affects young adult males.

#### Sites of infection

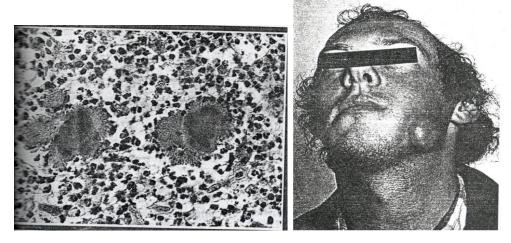
1- In the mouth---- the micro-organism gain an entrance following tooth extraction, carious tooth or after maxillofacial injury.

- 2- In abdominal actinomycosis---- in ileocaecal region.
- 3- Pulmonary actinomycosis

4- Pelvic actinomycosis---- involve fallopian tubes and ovaries and its strongly associated with presence of intra-uterine contraceptive devices

**Microscopically:** the lesion is usually characterized by chronic suppuration with multiple abscess formation, each containing one or more colonies of the organism the so called honeycomb abscess. Fibrous septa between abscesses are lined by granulation tissue which contains foamy cells (lipid-laden macrophages). In the centre is the pus containing actinomyces colonies, which are sometimes visible by nacked eye as small yellow or grey, gritty granules (sulphur granules)

**Cervicofacial manifestations:** lesions in the face and neck originating from the jaw produce much granulation tissue with many small foci of suppuration that persist and discharge through skin resulting in multiple sinuses.



Pus containing Actinomyces colonies

Actinomycosis abscess draining through the skin