PRIMARY TUBERCULOSIS

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Tuberculosis is a common disease prevalent throughout the world.

It is a chronic specific inflammatory infectious disease caused by Mycobacterium tuberculosis in humans.

Tuberculosis usually attacks the lungs but it can also affect any parts of the body.
ETIOLOGY OF TB

- **Myco. tuberculosis** – most common cause
- **Other than tuberculosis**- includes
  - M. avium intracellularare
  - M. kansasi
  - M. scrofulaceum
  - M. ulcerans
  - M. marinum
  - M. fortuitum
  - M. chelonei
SITES INVOLVED

- **PULMONARY TB** - 85% of all TB cases
- **EXTRAPULMONARY SITES.**
  - LYMPH NODE
  - GENITO-URINARY TRACT
  - BONES & JOINTS
  - MENINGES
  - INTESTINE
  - SKIN
CHARACTERISTICS OF M. TUBERCULOSIS

- Rod shaped, 0.2-0.5 µ in D, 2-4 µ in L.
- Mycolic acid present in its cell wall, makes it acid fast,
- So it resists decolourization with acid & alcohol.
- Aerobic and non motile.
- Multiplies slowly.
- Can remain dormant for decades.
How is TB Transmitted?

- Person-to-person through the air by a person with active TB disease of the lungs.

- Less frequently transmitted by:
  - Ingestion of *Mycobacterium bovis* found in unpasteurized milk products or autoingestion.
  - Inoculation (in skin tuberculosis).
  - Transplacental route (rare route).
PATHOGENESIS OF TUBERCULOSIS

- *M. tuberculosis* starts a IV hypersensitivity immune reaction inside the lung which damages the lung tissue while killing the foreign microorganism.

- Pathologic manifestation of tuberculosis like **caseating granuloma** and **cavitation** are result of hypersensitivity that develops in concert with the protective host immune response.

- **Macrophages** are the primary cells infected by *M. tuberculosis*. 
B. PRIMARY PULMONARY TUBERCULOSIS (>3 weeks)

- **Alveolar macrophage**
  - IL-12
  - T-cell
  - Class II MHC
  - MTb antigen
- **Activated macrophage**
  - TNF, chemokines
  - Monocyte recruitment
  - Caseous necrosis
  - Sensitized T-cell
- **Epithelioid granuloma** ("hypersensitivity")

**T-cell**
- T-cell receptor
- T\(_H\)1
- Production of nitric oxide and reactive oxygen species
- Bactericidal activity ("immunity")
- Tuberculin positivity ("hypersensitivity")
MORPHOLOGY OF TB

- PRIMARY TUBERCULOSIS:
  - Form of disease that develops in a previously unexposed person.
  - Almost always begins in lungs.
  - Inhaled bacilli implant in the distal airspaces of lower part of upper lobe or upper part of lower lobe.
  - It forms a small sub pleural parenchymal lesion in the mid zone of the lung (ghon focus inflammation +caseous necrosis)
  - Tubercle bacilli drains to the regional lymph node which also often undergo caseous necrosis.
  - Parenchymal lung lesion + Nodal involvement= Ghon’s complex.
Histologically: Granulomatous inflammation forms both caseating and non caseating tubercles.

Tuberculous Granuloma has the following characteristics:

1. Central caseous necrosis
2. Transformed macrophages called epithelioid cells
3. Lymphocytes, plasma cells, and fibroblasts
4. Langhans giant cells
Alveolar lumena

Giant multinucleated cell (immature - Muller type)

Central caseous necrosis

Collar of lymphocytes

Aggregation of epithelioid cells

Giant multinucleated cell (mature - Langhans type)
This is a caseating granuloma. Epithelioid cells surround a central area of necrosis that appears irregular, amorphous, and pink.
Fate of Primary tuberculosis

- Limited to primary focus: No progression. Healing by fibrosis and calcification.
- Progressive primary tuberculosis.
- Primary miliary tuberculosis: Dissemination to organs like liver, spleen, kidney, and other organs.
Primary Complex
(localized caseation)

Progressive Primary TB
(Massive hematogenous dissemination)

Miliary TB

Progressive Secondary TB
(Massive hematogenous dissemination)

Liver
Spleen
Miliary TB

Latent Lesions
(organisms dormant; pulmonary or extrapulmonary)

Reactivation
Reinfecction

Localized Caseating Destructive Lesions
(pulmonary or extrapulmonary)

Scar

Caseation
Caseation in lymph node

Healed Lesions
(organisms not viable)

Scar

Organisms in lymph node

Primary infection

TIME AFTER INFECTION
Weeks

Years