


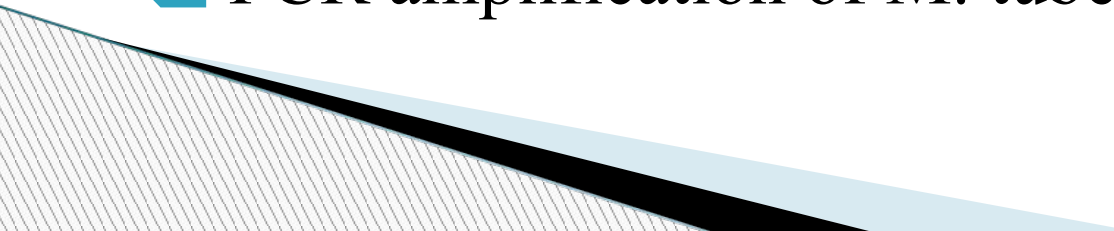
# Tuberculosis Disease

by  
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# Tuberculosis – Clinical Features

- Localized type may be asymptomatic
  - Low grade remittent fever, night sweats, malaise, anorexia, weight loss
  - Sputum at first mucoid and later purulent
  - 
  - Haemoptysis in half of the patients
  - Pleuretic pain
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# Tuberculosis – Diagnosis

- ❑ History, physical examination, radiological findings “*consolidation & cavitation*”
  - ❑ Identification of the acid-fast bacilli in smears and culture of sputum “10 weeks”
  - ❑ PCR amplification of *M. tuberculosis* DNA
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# Tuberculosis – Prognosis

- Depends on:
  - ✓ The extent of the disease and the patient immune status
  
- Secondary amyloidosis may occur in persistent cases

# Tuberculosis – Chronic Consequences

## 1. Pulmonary fibrosis

- ❖ The lung lesions may heal with fibrosis at any stage, particularly with treatment
- ❖ This ranges from minor apical scarring to extensive and severe widespread fibrosis producing localized to widespread honey-comb appearance of the lung tissue. It is particularly seen in relapsing and progressive untreated disease
- ❖ This is complicated by respiratory failure & cor pulmonale

## 2. Pleural fibrosis

- ❖ Fibrosis commonly obliterate the pleural space

## 3. Bronchiectasis

- ❖ Damage to the bronchial walls and scarring can cause distal pulmonary collapse, secondary infection and bronchiectasis

# Infection in Immunocompromised Individuals

- ❑ Mycobacterial infection of all types are increased in immunocompromised individuals and is in most cases due to reactivation of latent infection
- ❑ Features are similar to infection in immunocompetent individuals but disease usually progresses more rapidly due to decreased host response

# Atypical Mycobacterial Infection

- ❑ These infections are caused by a group of non-tuberculous mycobacteria of which the most important types are *M. avium-intracellulare* and *M. kansasii*
- ❑ The organisms are widely distributed in soil, water & domestic animals
- ❑ Infection is acquired directly from the environment and not by case to case contact
- ❑ Infection by these organisms is seen in immunocompromised patients particularly AIDS
- ❑ It can be seen also in immunocompetent individuals with chronic pulmonary disease

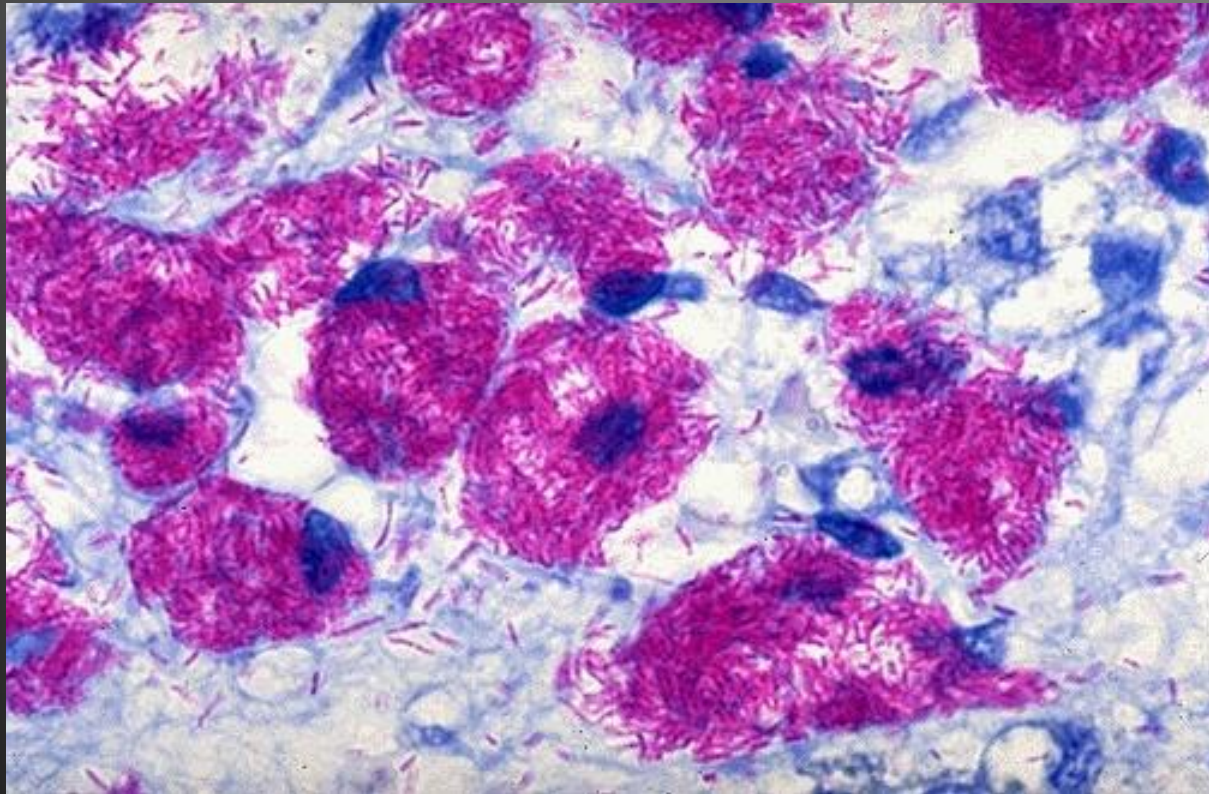
# Atypical Mycobacterial Infection – Gross Morphology



The lymph nodes in this mesentery, best seen at the left, are enlarged and have cut surfaces that appear yellow-tan. These nodes are filled with sheets of *Mycobacterium avium-complex* (MAC) organisms, and the immune response is so poor in this AIDS patient that there is **no focal granuloma formation**



# Atypical Mycobacterial Infection – Microscopic Morphology



Microscopically, *Mycobacterium avium-intracellulare* infection is marked by **numerous acid fast organisms growing within macrophages**. Lots of bright red rods are seen, particularly in macrophages, in this acid fast stain of lymph node

## PRIMARY

### Affect:

- \* Previously unexposed, unsensitized persons
- \* Non immune children
- \* Elderly & immunocompromised
- \* Very young

### Source:

- \* Exogenous
- \* 5% develop significant disease

### Morphology & Site:

- \* Primary T.B. mostly in lung rarely in intestine, pharynx, larynx & skin
- \* Involve lower part of upper lobe or upper part of lower lobe close to pleura
- \* Ghon-focus
  - A 1-1.5 cm gray white inflammatory consolidation with involvement of hilar lymph node
- \* In 95% of cases the development of cell-mediated immunity control the infection & no lesion develops

## SECONDARY

### Affect:

- \* Previously sensitized persons

### Sources:

- \* Develop from:
  - Reactivation of dormant lesion
  - Primary lesion if immunity of host is lowered “exogenous reinfection”
- 5% of primary T.B. develop secondary T.B

### Morphology & Site:

- \* Localized at the apex of both or one upper lobes because of better  $O_2$  tension
- \* Less lymph node involvement than the primary
- \* Cavitation is more common with dissemination along air ways
  - Cavitation is a source of infection by sputum
- \* Typically consolidating lesion 1-2 cm, firm gray-yellow at apical pleura with central caseation & peripheral fibrosis

### Histology:

\* Typically granulomatous inflammation, both caseating and non-caseating

### Outcome:

\* Hypersensitivity & increased resistance  
\* Healing & scarring of Ghon-focus but may be still a focus for reactivation  
\* May progress to progressive primary T.B. or disseminated T.B. especially in AIDS, malnourished, very old, very young & Eskimos

### Progressive primary T.B.:

1. Progressive enlargement of the primary focus with lung destruction & cavitation
2. Extension to the pleura □ pleural effusion or empyema
3. Enlarged lymph nodes □ obstruct bronchi □ bronchiectasis

### Histology:

\* Same

### Outcome:

\* The apical lesion may heal spontaneously Or with treatment and become a fibrocalcific Scar  
\* Depress and pucker the pleural surface and can cause pleural adhesion

### Progressive pulmonary tuberculosis:

1. Apical lesion enlarge with expansion of caseation.(cavitary fibrocaceous TB)
2. Erosion into bronchus with evacuation forming irregular cavity poorly delineated by fibrosis □ open lung lesion
3. Erosion into blood vessels □ hemoptysis
4. Invasion of pleural space □ pleural effusion, tuberculous empyema or obliterated fibrous pleuritis

### Dissemination:

- \* Erosion into bronchi with spread of the Infection □
  - Foci of infection in other parts of the lung
  - Tuberculous pneumonia
  - Laryngeal T.B. from coughed sputum
  - Intestinal T.B. from swallowing of infected material
  
- \* Erosion of vessels
  - Lymphatics □ foci of infection in the lung □ lung miliary T.B.
  - Blood vessels □ systemic miliary T.B.
  - Single-organ T.B. in bone, kidney, joint, brain...

### Fate:

- \* With adequate treatment:
  - The process is arrested by healing with fibrosis & destruction of the lung architecture
- If treatment is inadequate and host defence is impaired:
  - Spread of infection occurs

### Spread:

- \* Dissemination via airways, lymphatics & BV
- Miliary pulmonary disease when bacilli drain via lymphatics in lymphatic duct to the right heart back to the lung and give miliary T.B.
- Miliary systemic T.B. when infective foci seed the pulmonary venous return to the heart to the systemic circulation (liver, bone, spleen, adrenals, fallopian tubes)
- Endobronchial, endotracheal laryngeal miliary granuloma
- Isolated organ T.B.
- Intestinal T.B.

# Thanks

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