CHILDHOOD TUBERCULOSIS
OVERVIEW

• Introduction
• Pathophysiology
• Clinical presentation
• Diagnosis
• Treatment
• Prevention
ROBERT KOCH

- German physician and
- Microbiologist
- In 1882, he published his
- Findings on TB – Mycobacterium tuberculosis
- Nobel Prize in Physiology and
- Medicine in 1905
ROBERT KOCH’S CONT..

- World TB day 24 march
- Theme of the year –STOP TB IN MY LIFETIME
- India is the highest TB burden country accounting more than one fifth of the global incidence India (21%)
MYCOBACTERIUM TUBERCULOSIS
MYCOBACTERIUM TUBERCULOSIS

- Non–spore-forming, non motile, weakly gram-positive curved rods 2-4 microns long
- Obligate aerobe
- Grow best at 37-41°C
- Lipid rich cell wall
- Acid fastness – Stable mycolate complexes
PATHOPHYSIOLOGY

- Inhalation of viable microbe
- Lung most frequent portal of entry.
- Bacilli sets localized infection in the periphery of lung
- 4-6 wks later, tuberculin hypersensitivity with mild fever and malaise develop.
- Rupture of primary pulmonary focus into pleural cavity result in TB pleural effusion.
PATHOPHYSIOLOGY CONT..

• Site – lower segment of middle lobe or upper segment of lower lobe – mid zone – Maximum ventilation
• Initially polymorphonuclear response then
• macrophage/mononuclear response
• Multiply intracellularly in macrophages
• Cell mediated immune response in 2-12 weeks – caseous necrosis in centre of lesion & reduction of bacillary multiplication
• The primary focus, draining lymphatic and involved regional lymph node – PRIMARY COMPLEX
• GHON’S COMPLEX
PATHOPHYSIOLOGY

- Blood vessels
- Foamy macrophage
- Macrophage
- Infected macrophage
- Bacteria
- Necrotic tissue
- Fibrous cuff
- Epithelioid macrophage
- Lymphocyte
- Multinucleated giant cell
PATHOPHYSIOLOGY

• Area of necrosis surrounded by macrophages, lymphocytes, giant cells and collagen fibers – granuloma called TUBERCLE.

• 70% of Primary foci SUBPLEURAL - sluggish air current in lung periphery allow bacilli to stay longer.
• Hallmark of primary tuberculosis infection – relatively large size of adenitis, compared to relatively insignificant size of initial focus in the lung

• Right focus is common – greater volume & right bronchus is vertical, short & wide
PATHOPHYSIOLOGY

- Increased risk of TB in young children especially infants
- Tiniest aerosols 1-5 microns reach terminal airway establish pulmonary infection
- Larger droplets do not remain suspended & deposit in proximal airway – infection resisted
- The course of infection depends on the immune response of the host
CLINICAL PRESENTATIONS

Intrathoracic

• Pulmonary
• Latent tuberculosis infection
• Primary pulmonary complex
• Progressive primary disease
• Endobronchial tuberculosis
• Miliary tuberculosis
CLINICAL PRESENTATIONS Cont...

Extrathoracic

- Most common forms - peripheral lymphadenopathy & CNS
- Others are osteoarticular
- Abdominal/GIT
- Genitourinary
- Cutaneous and
- Congenital TB
LATENT TUBERCULOSIS INFECTION

• Reactive tuberculin skin test (TST) + absence of clinical and radiographic manifestations
• Infants with Latent TB Infection have up to 40% likelihood of developing disease
• But the risk for progression decreases gradually through childhood
• The greatest risk for progression occurs in the first 2 yr after infection
RISK FACTORS FOR PROGRESSION TO TUBERCULOSIS DISEASE

• Infants and children ≤4 yr of age, especially those <2 yr of age
• Co-infection with HIV
• Persons who are immunocompromised, especially in cases of malignancy and solid organ transplantation, immunosuppressive medical treatments including anti–tumor necrosis factor therapies, silicosis
• Diabetes mellitus, chronic renal failure and Malnutrition
PRIMARY PULMONARY COMPLEX

• Frequently encountered presentation in out patient
• Mild constitutional symptoms – mild fever, weight loss, anorexia, decreased activity, irritating dry cough due to enlarged node compressing bronchi and trachea
• Implantation site -> lymphatics -> regional lymph nodes.
• Primary complex- lesion at primary site of involvement, draining lymphatics and inflamed regional LN
PROGRESSIVE PRIMARY DISEASE

• Complication of primary pulmonary complex
• Reactivation due to old age, malnutrition, malignant disease, HIV infection & AIDS, use of immunosuppressive drugs, intercurrent infections.
• Progressive primary TB due to extension of inflammatory process i.e., consolidation of lung called galloping consumption or pneumonia alba.
• Caseation necrosis -> liquefaction -> cavity
PROGRESSIVE PRIMARY DISEASE Cont...

• Cavitating pulmonary TB uncommon in children
• Moderate to high grade fever, cough and hemoptysis suggest cavitation and ulceration of bronchus
• Findings of consolidation/cavitation – Dullness, decreased air entry and crepitations
• Extensive caseation necrosis, and cavitation.
• Cavities are better seen on CT
• Tuberculous cavities are site of growth of bacilli due to optimal temperature, more O2 content and nutrients from cell wall available

• Open TB

• Cavitary disease uncommon in children

• It primarily involves apical and posterior segment of upper lobes or superior segment of lower lobes in <95% cases
• Enlarged paratracheal nodes cause stridor or RD
• Subcarinal nodes on esophagus cause dysphagia
• Complete bronchial obstruction cause atelectasis
• Seeding of apex of lung leads to development of Simon's focus
• Endogenous reactivation of apex of lung is Pulh’s lesion
ENDOBRONCHIAL TUBERCULOSIS

- Fever & trouble some cough (with or without sputum)
- Dyspnea, wheezing and cyanosis
- Misdiagnosed as asthma
- Wheezing child < 2 yrs if there is poor response to anti-asthma medications should raise suspicion
- Partial airway compression – emphysema
- Complete airway compression - collapse
MILIARY TUBERCULOSIS

• Heavy hematogenous spread causing disease in 2 or more organs - Innumerable small foci
• Common in infants and malnourished or immunosuppressed patients
• Symptoms depend on bacillary load & organs involved
• High fever, rigors, altered sensorium, meningitis, lymphadenopathy & hepatosplenomegaly
• Smaller than 2-3 mm in diameter - coalesce to form larger lesions
Cont..

• TST is nonreactive in up to 40% of patients
• Rupture of subpleural focus result in pleural effusion due to hypersensitivity of tubercular proteins
• Tuberculous pleural effusion - uncommon in children below 5yrs, more common in boys and rarely associated with segmental lesion or miliary TB.
• Early stages - pleural rub, decreased chest wall movements, impairment of percussion note, decreased air entry on affected.
TB LYMPHADENITIS

• Most common form of extrapulmonary TB in children in endemic areas
• Incidence 8-10% of diagnosed with TB India
• Affects single node or localized group of nodes usually unilateral
• Prevalence 30-40% of TB
• In rural India 4.4/1000 children
TB OF PERIPHERAL LYMPH NODES

• From drinking unpasteurized cow milk or extension of primary lesion.
• Supraclavicular, tonsillar, submandibular lymph nodes (srofula)
• Untreated lymphadenitis, caseation necrosis, capsular rupture, spread to adjacent nodes and skin- > draining sinus tract.
STAGES OF TB LYMPHADENITIS

• Stage 1-enlarged, firm, MOBILE, DISCRETE
• Stage 2- large, rubbery, FIXED
• Stage 3-central softening due to abscess formation
• Stage 4-coller stud abscess
• Stage 5-sinus tract formation
Cont..

- Primary infection
  - Lung parenchyma
  - Lymph nodes involved are
  - Hilar
  - Mediastinal (MC in children)
  - Paratracheal
- Tonsils & adenoids
  - Lymph nodes involved are
  - Cervical
CNS TUBERCULOSIS

- MC form in TB meningitis in Indian children
- Incidence 1-4%
- MC route is lympho-heamatogenous
- From caseous lesion in cerebral cortex or meninges from early occult heamatogenous spread
- Rapid progression to hydrocephalus, seizures, cerebral edema, fever, headache, irritability, drowsiness
• Abruptly advances with lethargy, vomiting, nuchal rigidity, seizures, hypertonia, focal neurological signs
• Final stage-Coma, hypertension, decerebrate, decorticate posture
• Rapid confirmation difficult-wide variability in CSF characteristics, non reactive TST in 40%, normal CXR in 50%
CSF ANALYSIS IN TB MENINGITIS

• Drained under pressure manometer reading 30-40 cm H20
• Opaque or clear on gross examination
• Pellicle or cob web formation on standing
• Cell counts: increased with lymphocytic predominance, early stages polymorphonuclear response later lymphocyte predominance
• Proteins: moderately increased > 100 mg/dl
• Glucose: less than 2/3\textsuperscript{rd} of blood glucose level
CSF SMEAR

- Definitive diagnosis
- Staining clot that forms on standing
- Spinning down CSF sediment on to slide for microscopic examination 87% positivity
- Minimum of 6 ml of CSF examined microscopically for 30 mint.
ADA levels

- Produced by lymphocytes and monocytes
- Used in pleural, peritoneal and pericardial forms of TB
- Cut-off 8 units per lit. (sn-59%, SP-96%)
- ADA also increased in pyogenic meningitis can’t discriminate TBM from Pyogenic Meningitis
ABDOMINAL TUBERCULOSIS

• It is less common as compared to pulmonary, neuro & lymph node TB.
• Definition- TB infection of abdomen, including GIT, peritoneum, mesentery, abdominal LN, liver spleen & pancreas
• 0.8-3.6% of total admissions
• Intestinal TB-main cause of 15% of all intestinal obstruction & 5-7% of all GI perforation
• Primary involvement is rare, mostly secondary
<table>
<thead>
<tr>
<th>SITE</th>
<th>TYPE</th>
<th>FEATURES</th>
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<tbody>
<tr>
<td>intestines</td>
<td>Ulcerative</td>
<td>Chronic diarrhea with malabsorption</td>
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<tr>
<td></td>
<td>Hypertrophic ulcerohypertrophic</td>
<td>Subacute intestinal obstruction</td>
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<tr>
<td>Lymph nodes</td>
<td>Mesenteric Reteroperitoneal</td>
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<tr>
<td>peritoneum</td>
<td>Exudative or ascitic Plastic or adhesive</td>
<td>MC in female, present as abdominal distension, ascites fever, night sweats, diffuse abd tenderness, doughy feel</td>
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<td>Visceral organs</td>
<td>Liver Spleen pancreas</td>
<td>As a part of disseminated miliary TB</td>
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<td>esophagus</td>
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<td>Rare due to extension of disease from adjacent LN in mediastinum</td>
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<tr>
<td>gastric</td>
<td></td>
<td>Rare due to acidity and paucity of LN Symptoms nonspecific</td>
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ULTRASOUND ABDOMEN

1. Mesentric thickness >= 15mm
2. Increased echogenicity
3. Lymphadenitis
4. Intra-abdominal fluid-loculated & clear or complex with debris & septae (it is superior to CT to detect fluid)
ASCITIC FLUID ANALYSIS

1. Color - straw or clear
2. Exudative
3. Proteins >3g/dl
4. Cells >1000/cumm (MC lymphocytes)
5. Glucose-ascitic/blood ratio <1.1gm/dl
6. ADA >33U/L (sensitivity-93%, specificity-96%, PPV-93%)
   {screening test}
Thank You