Extra Pulmonary Tuberculosis:

TB Intensive Course
December, 2007
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Spectrum of EPTB:

- CNS
  - Meningitis
  - Tuberculoma
- Skeletal
  - Spinal
  - Joint
  - Osteomyelitis
  - Myositis
- Lymphadenitis
- Pleural
  - Effusion
  - Empyema
- Genitourinary
- Gastrointestinal
- Pericardial
- Cutaneous
- Other

Spread to extra-pulmonary sites:

1. Exit of infected phagocytes from the lung.
2. Transcytosis across alveoli to monocytes.
3. Necrosis facilitates spread to bloodstream.

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Schematic representation of central role of TNF-Alpha in cellular immune response to M.Tb infection

*The Lancet Infectious Diseases 2003;3(3):148-155*

Model of Granuloma formation. In presence of TNF-Alpha.

*The Lancet Infectious Diseases 2003;3(3):148-155*
Model of Granuloma formation. In absence of TNF-Alpha.

The Lancet Infectious Diseases 2003;3(3):148-155
CNS Tuberculosis:

- CNS TB includes:
  - meningitis,
  - and tuberculoma.
- CNS infection is encountered frequently in high incidence countries.

INTRODUCTION

- Approximately 300 to 400 cases of TB meningitis occur each year in the US.
  - 1% of all TB disease.
- The case fatality remains high at 15% to 40% despite effective treatment regimens.
INTRODUCTION

• Early recognition of TB meningitis is paramount.
• Immediate empiric Rx should be started in any Pt with
  – a meningitis syndrome and
  – low CSF glucose, elevated protein, and lymphocytic
    pleocytosis and
  – evidence of TB elsewhere in the body or
  – if prompt evaluation fails to establish an alternative diagnosis.

INTRODUCTION

• Serial examination of the CSF by AFB stain and culture is the best diagnostic approach.
• Smears and cultures may yield positive results days to weeks after therapy has been initiated.
Pathogenesis:

- **Post-primary infection:**
  - infants and young children
  - advanced HIV infection

- **Reactivation bacillemia:**
  - immune deficiency of aging,
  - TNF inhibitor Rx,
  - alcoholism,
  - malnutrition,
  - malignancy

Pathogenesis:

- Spillage of tuberculin into the subarachnoid space produces
  - intense hypersensitivity with
  - inflammatory changes,
  - most marked at the base of the brain.
Basilar Meningitis:
Thick grey shaggy exudate encasing cranial nerves & blood vessels:

Necrotizing granulomatous inflammation of meninges.

Pathogenesis:

• 3 features dominate the pathology and explain the clinical manifestations.
  – Proliferative arachnoiditis
  – Vasculitis
  – Communicating hydrocephalus
TB Meningitis - Vasculitis:

CLINICAL FEATURES:

1. **Prodromal** phase, 2 to 3 weeks, with
   a) insidious malaise, lassitude,
   b) headache,
   c) low-grade fever, and
   d) personality changes.
2. **Meningitic** phase with
   a) meningismus,
   b) protracted headache,
   c) vomiting,
   d) confusion, and
   e) varying cranial nerve and long-tract signs.

3. **Paralytic** phase is the stage during which the pace of illness may accelerate rapidly; confusion gives way to
   a) stupor and coma,
   b) seizures, and
   c) hemiparesis.
Atypical presentations:

- Acute, rapidly progressive, syndrome suggesting acute bacterial meningitis.

Atypical presentations:

- Slowly progressive dementia over months or even years characterized by
  - personality change,
  - social withdrawal,
  - loss of libido, and
  - memory deficits.
Atypical presentations:

• **Encephalitic** course manifested by
  – stupor,
  – coma, and
  – convulsions without overt meningitis.

Clinical stages:

• **Stage I** patients are **lucid** with no focal neurologic signs or evidence of hydrocephalus.
• **Stage II** patients are **confused** or have focal signs, such as cranial nerve palsies or hemiparesis.
• **Stage III** represents advanced illness with delirium, stupor, **coma**, or dense hemiplegia.
HIV infection:

- Co-infection with HIV has been observed in 20% of patients with EPTB in the US.
- In one study, cerebral tuberculomas were seen to be more common in the HIV-infected group (60% versus 14%).
- Otherwise, HIV co-infection did not alter the clinical manifestations, CSF findings, or response to therapy.

DIAGNOSIS CSF Examination:

- Diagnosis (Dx) can be difficult.
- Proper CSF specimens are critical for early Dx.
- Maintain a high degree of suspicion.
- Initiate empiric therapy promptly.
DIAGNOSIS CSF Examination:

- Typical CSF formula shows
  - elevated protein
  - lowered glucose and
  - a mononuclear pleocytosis.
- CSF protein ranges from 100 to 500 mg/dL.
- **Hydrocephalus** with subarachnoid block may have protein levels of 2 to 6 g/dL, xanthochromia and a poor prognosis.

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DIAGNOSIS CSF Examination:

- The CSF glucose is less than 45 mg/dL in 80%.
- The usual CSF cell count is between 100 and 500 cells/µL.

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DIAGNOSIS CSF Examination:

- Early in the course of illness, the cellular reaction may be atypical with
  - only a few cells or
  - with a PMN predominance.
- Usually changes rapidly to a lymphocytic predominance.

DIAGNOSIS AFB smears:

- Repeated, careful CSF examination and culture for *M. tuberculosis* cannot be overemphasized.
- In one series, only 37% of cases were diagnosed on the basis of an initial positive AFB smear.
- The yield increased to 87% when, despite therapy, up to 4 serial specimens were examined.
**DIAGNOSIS AFB smears:**

- A minimum of 3 lumbar punctures should be performed at daily intervals.
- Empiric therapy should **not** be delayed.

**DIAGNOSIS AFB smears:**

- The sensitivity of the AFB smear may be enhanced by:
  - Using the last fluid removed at lumbar puncture.
  - Removing a large volume (10 to 15 mL) of CSF.
  - Examining a smear of the clot or centrifuged sediment (cyto-centrifuge).
DIAGNOSIS AFB smears:

• 200 to 500 HPF should be examined methodically (approximately 30 minutes), preferably by more than one observer.
Polymerase chain reaction (PCR)

- There is considerable variability in sensitivity and specificity among labs.
- Comparing CSF PCR with AFB stain and culture, the sensitivity of PCR testing was only 60% in 15 patients with definite or probable TB meningitis.

Molecular Diagnosis of TB Meningitis
Thwaites, J Clin Micro, 2004; 42: 996

73 patients with TM vs. 79 without TM
AFB (Z/N) Staining C/W Gen-Probe Amplified Mycobacterium Direct Test (MTD)

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Z/N</td>
<td>52% (2% after Rx)</td>
<td>100%</td>
</tr>
<tr>
<td>MTD</td>
<td>38% (28% after Rx)</td>
<td>99%</td>
</tr>
<tr>
<td>Combined</td>
<td>68% (83% with repeated samples)</td>
<td></td>
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</tbody>
</table>

Before Treatment: Careful bacteriology is as good as, or better than, the commercial NAA assays.
PCR
• Nevertheless, submit CSF specimens for PCR testing whenever clinical suspicion is high and initial AFB stains are negative!
• A negative test result neither excludes the diagnosis nor obviates the need for continued empiric therapy.

Neuroradiology
• Contrast CT defines the presence and extent of
  – basilar meningitis,
  – cerebral edema and infarction, and
  – hydrocephalus.
Multiple tuberculomas along enhanced dural reflections:

Basilar Enhancement & Hydrocephalus:
Tuberculoma & Hydrocephalus:
Neuroradiology

• In two large community-based series
  – hydrocephalus was seen in ~ 75%,
  – basilar meningeal enhancement in 38%,
  – cerebral infarcts (stroke) in 15 to 30%, and
  – tuberculomas in 5 to 10%.

Neuroradiology

• A case series from Hong Kong documented
  – hydrocephalus was seen at diagnosis in 9 of 31 patients with TB meningitis;
  – hydrocephalus occurred after the start of therapy in only one of the remaining 22 patients.
Neuroradiology

• Compatible clinical features, CT evidence of basilar meningeal enhancement AND any degree of hydrocephalus is strongly suggestive of TB meningitis.

• Hydrocephalus combined with marked basilar enhancement is indicative of advanced disease and carries a poor prognosis.

Neuroradiology

• Marked basilar enhancement correlates well with vasculitis and, therefore, with a risk for basal ganglia infarction.

• MRI is superior to CT in defining lesions of the basal ganglia, midbrain, and brain stem and for evaluating all forms of suspected spinal TB.
Neuroradiology

- The CT scan is normal in approximately 30% of cases with Stage I disease.
- Patients with a normal CT scan nearly always recover completely on therapy.

Other Conditions Mimicking TB Meningitis Radiographically:

- Cryptococcal Meningitis
- Coccidioides Meningitis
- Viral Encephalitis
- Sarcoidosis
- Meningeal Metastases
- Lymphoma
THERAPY **Recommended regimens:**

- **Drug resistance** should be considered in individuals
  - from areas of the world where TB is prevalent,
  - in those with a history of prior TB treatment, and
  - in those with exposure to drug-resistant source.

**Duration of therapy**

- There are no randomized trials to establish the optimal duration of therapy.
- It is recommended that therapy be administered for **12 months** in the usual case of drug-sensitive infection.
Duration of therapy

- If PZA is omitted or cannot be tolerated, treatment should be extended to 18 months.
- There are no guidelines for the duration of therapy in patients with MDR-TB.
- The duration of therapy may be extended to 18 to 24 months, based on severity of illness, clinical response, and the pt’s immune status.

CSF Penetration of TB Drugs

<table>
<thead>
<tr>
<th>GOOD</th>
<th>FAIR</th>
<th>POOR</th>
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<tbody>
<tr>
<td>Isoniazid *</td>
<td>Rifampin *</td>
<td>Streptomycin *</td>
</tr>
<tr>
<td>Pyriziamide</td>
<td>Ethambutol</td>
<td>Capreomycin *</td>
</tr>
<tr>
<td>Ethionamide</td>
<td>Quinolones *</td>
<td>Amikacin *</td>
</tr>
<tr>
<td>Cycloserine</td>
<td></td>
<td></td>
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<tr>
<td>Linezolid *</td>
<td></td>
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* Can Be Given IV

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Adjunctive Corticosteroids:

- There is substantial data that adjunctive corticosteroid therapy is beneficial.
- A randomized, double-blind trial in Vietnam compared 6 to 8 weeks of dexamethasone with placebo in 545 patients over 14 years of age.

<table>
<thead>
<tr>
<th>Mortality (%)</th>
<th>Stage I:</th>
<th>Stage II:</th>
<th>Stage III:</th>
<th>Total:</th>
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<tbody>
<tr>
<td>Steroid Group:</td>
<td>17</td>
<td>31</td>
<td>55</td>
<td>32</td>
</tr>
<tr>
<td>Non-Steroid:</td>
<td>20</td>
<td>40</td>
<td>60 NS</td>
<td>41</td>
</tr>
</tbody>
</table>
Adjunctive Corticosteroids:

• There was no reduction in residual neurologic deficits and disability among surviving patients at nine months.

• The survival benefit associated with steroid therapy may have been in part due to a reduction in severe adverse events (9.5 versus 16.6%), particularly hepatitis that necessitated changes in TB drug regimens.

• No mortality benefit from dexamethasone was seen in 98 HIV-infected patients included in the study.
Adjunctive Corticosteroids:

- Adjunctive corticosteroid therapy is **recommended** for all children and adults treated for TB meningitis.
- The possible exception being adults with early mild Stage I manifestations.

Adjunctive Corticosteroids:

- Specific clinical indications include:
  - Acute "encephalitis" with high CSF pressure or cerebral edema.
  - Exacerbation of signs after beginning therapy.
  - Spinal block or incipient block (CSF protein >500 mg/dL and rising).
  - CT evidence of marked basilar enhancement.
  - Patients with symptomatic tuberculoma.
Adjunctive Corticosteroids:

- Be aware that concurrent treatment with Rifampin may reduce effective levels of corticosteroids by induction of the hepatic cytochrome P-450 system.

Recommended Corticosteroid regimens:

- Children — Prednisone 2 to 4 mg/kg per day tapered over 4wks.
- Adults either:
  - Prednisone 60 mg per day tapered gradually over six weeks or
  - Dexamethasone IV for the first three weeks (initially 0.4 mg/kg per day, tapering to 0.1 mg/kg per day) followed by oral 4 mg per day, tapered over 3 - 4 weeks at the rate of 1 mg decrease dose each week.
Surgery

- Patients with hydrocephalus may require urgent shunting.
- Serial LP and steroid therapy may suffice for Stage I pts awaiting response to chemotherapy.
- Shunting should not be delayed in patients with stupor, coma or progressive neurologic signs.

SPINAL TUBERCULOUS ARACHNOIDITIS

- More commonly seen in countries outside Europe and the Americas.
- Pathogenesis is similar to that of meningitis, with
  - focal inflammatory disease at single or multiple levels
  - gradual encasement of the cord by a gelatinous or fibrous exudate.
SPINAL TUBERCULOUS ARACHNOIDITIS

• Symptoms progress over weeks to months and may terminate with meningitis syndrome.
• Patients present with subacute onset of root and cord compression signs:
  – spinal or radicular pain,
  – hyperesthesis or paresthesias;
  – lower motor neuron paralysis; and
  – bladder or rectal sphincter dysfunction.

Vasculitis may lead to thrombosis of the anterior spinal artery and infarction of the spinal cord.
• Other forms include extradural or intradural tuberculoma and epidural abscess.
• TB is a prevalent cause of paraplegia in the developing world.
**SPINAL TUBERCULOUS ARACHNOIDITIS**

- The diagnosis is based upon the findings of
  - abnormal CSF protein indicative of spinal block,
  - MRI changes of nodular arachnoiditis, and
  - compatible tissue biopsy.
- Treatment is the same as for TB meningitis.

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**Skeletal Tuberculosis:**

- Vertebral Osteomyelitis – Pott’s Disease
- Arthritis
- Myositis
- Osteomyelitis
History:

- 39 y.o. undocumented Hispanic male presented with F, wt. loss & upper back pain which was worse with cough.
- No prior TB Hx or treatment.
- Pt found to have far advanced TB on CXR.
- Sputum AFB heavily positive, PCR – MTb.

History 2:

- CT spine and MRI spine revealed discitis, vertebral osteo. and epidural abscess at T2 – 4.
- Sterile pyuria, anemia and hyponatremia also noted.
History 3:

- Patient treated with RIPE.
- Proposed and accepted for transfer 11/3/03 to TCID due to “preserved neurologic exam.”

Exam:

- T 102.0°F, HR 88, BP 84/42, 5’3”, 111 lbs.
- Lungs – occ. faint upper lobe crackles.
- Neurologic:
  - Strength 4/5 BLE, weak ankle dorsiflexors.
  - Sensory numbness below T8.
  - DTR’s 3/4.
  - Plantar flexor reflex on Left, equivocal on Right.
Hospital Course:

- Patient treated with RIPE.
- Continued daily fevers.
- Pt had decreased LE strength and was transferred in isolation for neurosurgical assessment.
- Neuro exam revealed anti-gravity strength Rt > Lt, weak right ankle dorsiflexor, numbness below nipples, and intact toe position sense.

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Laminectomy T2 – T5:

- Posterior laminectomy was performed.
- No discrete epidural mass was discovered.
- Having decompressed the thecal sac the surgeon judged not to go further.
- Corticosteroids were given during the early post operative phase then discontinued.

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Further Management:

• Pt returned to TCID, afebrile with diminished cough, able to do transfers and with improved LE strength.
• Pt completed a 1 year course with prolonged continuation phase (INH & RIF).
• Pt was able to walk on discharge.

Potts Disease:
Potts T10 & T11:  

24 yo Nigerian Female with 1 yr history of back pain:
Psoas Abscess:

Needle Biopsy of Vertebral Osteomyelitis under CT guidance:
TB Osteomyelitis Distal Femur:

Tuberculosis of the right hip. Plain film of the right hip in a 28 year old woman with painful joints and a productive cough demonstrates complete loss of the joint space and destruction of the cartilage and adjacent joint surfaces with severe pararticular bony demineralization (arrows). Courtesy of Jonathan Kruskal, MD.
Tb of the ankle:

TB Arthritis of the wrist:
TREATMENT

• **Duration of therapy** —
  • Traditionally, 12 to 18 month courses of therapy have been advocated.
  • There has been concern about drug penetration into necrotic bone and fibrous tissues.
  • Several studies have advocated shorter courses of treatment.

TREATMENT

• Many specialists, continue to advise at least nine months Rx in patients with skeletal TB.
• Longer treatment regimens (at least 12 months) are recommended for patients
  – with advanced or extensive disease,
  – concern for vital adjacent structures (spinal cord) or
  – if the response to therapy is uncertain.
Role of surgery

• In many countries, orthopedic surgery is neither affordable nor available.
• When available, surgery is a useful adjunct for:
  – drainage of an abscess,
  – debridement of infected material, and
  – decompression and stabilization of vital structures such as the spinal cord.

Role of surgery

• Judicious use of surgery may improve early mobilization and reduce morbidity.
• Once treated, reconstructive surgery and even prosthetic joint replacement can be considered.
• Unlike typical bacterial osteomyelitis, early surgical debridement is not beneficial in skeletal TB.
Monitoring of clinical response

• The response to therapy is best monitored by clinical indicators such as
  – pain,
  – constitutional symptoms,
  – mobility, and
  – accompanying neurologic signs.

Monitoring of clinical response

• There is often radiographic progression of disease during the first six months despite appropriate treatment.
• X-ray worsening is not an indication to change therapy.
Lymphadenitis:

- Cervical – Scrofula
- Peripheral
- Visceral
Peripheral Tuberculous Lymphadenitis
Polesky, Medicine, 2005; 84:350

106 Patients with TB lymphadenitis
(Santa Clara Co., CA, 1994-1999)

Female 66%
Mean Age 34 years
Foreign Born 92% (Vietnam 60%)
Years in US 5.2 (mean)
HIV Seropositive 5%
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Necrotic Lymph Node by CT:

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43 y.o. HIV+ (CD4=46)

Pulmonary TB

Started on RIPE

Started on HAART – AZT/3TC/efavirenz

17 Days later….

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Immune Reconstitution TB LAD:

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TB LAD:

Pleural Tuberculosis:

- Exudate
- Empyema
- Empyema necessitans
- Broncho-pleural Fistula
PLEURAL TUBERCULOSIS

- The most common cause of pleural effusion in many areas of the world.
- In the US, 1/30 patients with TB have effusion.
- Frequently a manifestation of primary TB.
- True incidence unknown.
Therapy of Pleural TB:

- Standard RIPE therapy advised.
- **AVOID CHEST TUBE**: Fluid continues to form, fluid frequently loculated, may create BPC fistula.
- **NO DECORTICATION FOR 6 MONTHS**: Pleural thickening decreases with time and treatment.
Therapy of Pleural TB:

• BP FISTULA (air-fluid level in pleural space):
  Does not heal spontaneously.
  90-120 days therapy before decortication.
  ± Thoracoplasty, open external drainage.

Tb Empyema:
Empyema Necessitans:
Empyema necessitans (SQ):
Pericardial Tuberculosis:

- Effusive
- Constrictive
- Calcific
Effusive:

Pericardial Calcification:
**M-mode echocardiogram of a large pericardial effusion** Left panel: The echo image at the level of the aortic root (Ao) and left atrium (LA) demonstrates a large echo free space representing a pericardial effusion (PE) behind the LA. LA wall motion is markedly increased (black arrows). Right panel: A large echo free space, representing the effusion, is present behind the left ventricle (LV) throughout systole and diastole. RV = right ventricle, MV = mitral valve; IVS = intraventricular septum.

**NORMAL HEMODYNAMICS:**

**TAMPONADE:**

**Pericardial effusion** Subcostal four-chamber view from a two-dimensional echocardiogram shows a moderate-sized pericardial effusion (PE) in a patient with normal hemodynamics at cardiac catheterization. The effusion surrounds the right atrium (RA), right ventricle (RV) and left ventricle (LV). Courtesy of Ralph Snobetai, MD.

**Pericardial effusion** Subcostal four-chamber view from a two-dimensional echocardiogram shows a massive pericardial effusion (PE) surrounding the left ventricle (LV), right atrium (RA), and right ventricle (RV) which is strikingly reduced and shows diastolic collapse. The patient had severe hemodynamic compromise and tamponade physiology. Courtesy of Ralph Snobetai, MD.
Constrictive pericarditis occurred in 16 of 18 untreated patients in a series reported in 1948. This complication now occurs in only 10 to 20% of treated cases. A trial from South Africa found that corticosteroids added for the first 11 weeks of Tb therapy reduced mortality and the need for subsequent pericardiocentesis.
Role of corticosteroids

• The following benefits were noted in the prednisolone group:
  – More rapid reduction in pulse rate and JVP
  – Improved functional status
  – Lower mortality during follow-up (4% versus 11%)
  – Lower need for pericardiectomy (21% versus 30%)

Role of corticosteroids

• Among the same 240 patients with TB pericarditis and effusion, prednisolone treatment with open drainage were beneficial.
• Mortality rate was 3% in those treated with drainage, TB therapy and prednisolone.
• Mortality in those treated only with drainage and TB therapy was 14%.
Role of corticosteroids

• The ATS, CDC, and IDSA joint guidelines recommend corticosteroids as an adjunct during the first 11 weeks of therapy for TB pericarditis.

Role of corticosteroids

• The recommended regimens are as follows:
  – For adults, prednisone 60 mg/day for 4 weeks,
    • 30 mg/day for 4 weeks,
    • 15 mg/day for 2 weeks, then
    • 5 mg/day for week 11.
  – For children, prednisone 1 mg/kg daily as the initial dose for 4 weeks, with a decreasing dose over time as described for adults.
Pericardiectomy

- Reserved for pts with recurrent effusions or elevated of CVP after 4 to 6 weeks of TB and corticosteroid therapy.
- Surgery is more easily accomplished and has a lower mortality if performed “early”.
- The procedure has less beneficial outcomes in patients who have end-stage constriction.

The preferred technique for pericardiectomy.

Through a left anterior thoracotomy, the pericardium is removed from the level of the left pulmonary veins to the right side of the mediastinum.

The left phrenic nerve is preserved on a pedicle of pericardium.
Genitourinary Tuberculosis:

- Renal
- Ureters & Bladder
- Genital
Ureteral TB with stenosis.
Blunting of the calyces (caliectasis) and two long ureteral strictures (arrows) are seen. Although the caliceal changes can be seen in other disorders (such as reflux nephropathy), the concurrent ureteral abnormalities are virtually diagnostic of tuberculosis.

TB Testis:
Diagnosis

• Supported by the demonstration of AFB in the urine.
• Dysuria, sterile pyuria, hematuria, and the characteristic IVP findings are highly suggestive.
• NTM may cause false (+) AFB smears.
• Urine TB culture is the gold standard
  – Three to six first morning midstream specimens.
  – Bacilli are shed into the urine intermittently
  – Only 30% to 40% of single specimens are positive.

Gastrointestinal Tuberculosis:

• Peritoneal
• Hollow Viscus
• Hepatosplenic
• Other
Tuberculosis Peritonitis:

- Sx: Acute or chronic abdominal pain, swelling, constitutional symptoms, intestinal obstruction.
- Ascitic fluid: Exudate, 150-400 WBC, lymphocyte predominance (can be neutrophilic).
- (+) MTB culture <20%.
- Elevated ascitic fluid adenosine deaminase (ADA).
- Laparoscopy and biopsy.
GI Tuberculosis:

- Chronic pain (R L Q), weight loss, anorexia, fever
- Colitis (ulcerative, hypertrophic) with predilection for ileocecal region (duodonitis-PUD)
- Stricture, bowel perforation
- Endoscopy- Visual findings not pathognomic, requires biopsy with AFB smear and culture

Duodenal TB (Miliary Case):
• TB Ascending Colon:

• TB Splenic Flexure at onset of treatment.
• TB Splenic Flexure after 10 weeks of Rx.

84 y.o. WM with change in bowel habits and an normal CXR.
Anatomic distribution of GI TB:

<table>
<thead>
<tr>
<th>Site</th>
<th>Autopsy %</th>
<th>Clinical %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Esophageal</td>
<td>0.14</td>
<td>0.3</td>
</tr>
<tr>
<td>Stomach</td>
<td>0.69</td>
<td>2</td>
</tr>
<tr>
<td>Duodenum</td>
<td>2.5</td>
<td>0.3</td>
</tr>
<tr>
<td>Ileocecal</td>
<td>66.1</td>
<td>42</td>
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<tr>
<td>Colon</td>
<td>53.8</td>
<td>12</td>
</tr>
<tr>
<td>Anorectum</td>
<td>11.9</td>
<td>7</td>
</tr>
</tbody>
</table>

Hepatic or Splenic TB:
ENT Tuberculosis:

- Laryngitis
- Otitis
- Mastoiditis
TB Otitis Media:

Extensive Mastoiditis on L
Other EPTB:

- Adrenal
- Thyroid
- Myocardium
- Retina – choroidal tubercles
- Skin – Lupus vulgaris

Adrenal Calcifications from Tb:
Adrenal tuberculosis in 49-year-old man of 2 months duration. Contrast-enhanced CT shows the bilateral mass-like enlargement with peripheral enhancement.

Choroidal Tubercles:

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Lupus vulgaris

Reddish-brown plaque. Note nodular infiltration, scaling of the helix, and atrophic scarring in the center of the plaque.

16 y.o. male with PTB & Skin TB x 5yrs:
Cutaneous TB:

21-yo female with 2-yr hx of a progressive ulceration of the nose and lips.

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A 70-year-old man presented to hospital with a two month history of dull back pain. He had a history of pulmonary tuberculosis 50 years previously.

Preoperative chest computed tomography showing a descending aortic aneurysm with TB destruction of the vertebrae from T8 to T10

TB Mycotic Aortic Aneurysm:
Miliary TB:

• Wide dissemination throughout the body.
• Tiny size of the lesions.
• Distinctive pattern seen on chest X-ray.
  – With or without other types of infiltrates.
• Appearance similar to millet seeds, thus the term "miliary" tb.
Guidelines for EPTB Treatment:

• In general, EPTB can be treated with the same regimens as pulmonary disease.
• Evidence suggests that 6- to 9-month regimens that include INH and RIF are effective.
• If PZA cannot be used in the initial phase, continuation phase must be increased to 7 months.

Guidelines for EPTB Treatment:

• For disseminated TB and TB meningitis, 9--12 months of treatment is recommended.
• Prolongation of therapy also should be considered for TB in any site that is slow to respond.
• The addition of corticosteroids is recommended for patients with TB pericarditis and TB meningitis.