SPINAL TUBERCULOSIS DIAGNOSIS & MANAGEMENT

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INTRODUCTION

- Evidence of spinal TB dates back to Egyptian times and has been documented in 5000-year-old mummies.

- In 1779, Percival Pott published the first modern description of spinal deformity and paraplegia resulting from spinal TB.

- According to WHO(2006), about one third of the world’s population is infected by *Mycobacterium TB*, and 9 million individuals develop TB each year.
• One fifth of TB population is in India.
• Three percent are suffering from skeletal TB.
• Vertebral TB is the most common form of skeletal TB and accounts for 50% of all cases of skeletal TB.
• Almost 50% are from pediatric group.
• Every day 1000 die of tuberculosis in India.
• Neurological complications are the most crippling complications of spinal TB (Incidence: 10 to 43%).
## SPINAL TUBERCULOSIS

### REGIONAL DISTRIBUTION

<table>
<thead>
<tr>
<th></th>
<th>Region</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Cervical</td>
<td>12%</td>
</tr>
<tr>
<td>2</td>
<td>Cervicodorsal</td>
<td>5%</td>
</tr>
<tr>
<td>3</td>
<td>Dorsal</td>
<td>42%</td>
</tr>
<tr>
<td>4</td>
<td>Dorsolumbar</td>
<td>12%</td>
</tr>
<tr>
<td>5</td>
<td>Lumbar</td>
<td>26%</td>
</tr>
<tr>
<td>6</td>
<td>Lumbosacral</td>
<td>3%</td>
</tr>
</tbody>
</table>
SPINAL TUBERCULOSIS

Pathology

• Spinal tuberculosis is usually a secondary infection from a primary site in the lung or genitourinary system.
• Spread to the spine is hematogenous in most instances.
• Delayed hypersensitivity immune reaction.
• Initially: a pre-pus inflammatory reaction with Langerhan’s giant cells, epithelioid cells, and lymphocytes.
• The granulation tissue proliferates, producing thrombosis of vessels.
SPINAL TUBERCULOSIS

- Tissue necrosis and breakdown of inflammatory cells result in a paraspinal abscess.
- The pus may be localized, or it may track along tissue planes.
- Progressive necrosis of bone leads to a kyphotic deformity.
- Typically, the infection begins in the anterior aspect of the vertebral body adjacent to the disk.
- The infection then spreads to the adjacent vertebral bodies under the longitudinal ligaments.
- Noncontiguous (skip) lesions are also seen occasionally.
SPINAL TUBERCULOSIS

SIGNS & SYMPTOMS

Spine Deformity
- Kyphosis
- Scoliosis

Neurological
- Pain
- Motor deficits
- Spasticity

Constitutional
- Sensory deficits

Local
- Bladder involvement
- Cold abscess / Sinuses

Local / Radicular / Dysesthetic
SPINAL TUBERCULOSIS

DIAGNOSIS

HISTORY

- Presentation depends on:
  - Stage of disease,
  - Site
  - Presence of complications such as neurologic deficits, abscesses, or sinus tracts.
- Average duration of symptoms at the time of diagnosis is 3 – 4 months.
- Back pain is the earliest & most common symptom.
- Constitutional symptoms.
- Neurologic symptoms (50 % of cases).
**SPINAL TUBERCULOSIS**

**DIAGNOSIS**

- Physical examination of the spine:
  - Localised tenderness and paravertebral muscle spasm,
  - Kyphotic deformity,
  - Cold abscess swelling / sinus tract
  - Cervical spine TB is a less common presentation, characterized by pain & stiffness with dysphagia / stridor more common in lower cervical spine involvement
SPINAL TUBERCULOSIS

DIAGNOSIS

LAB STUDIES

• **Mantoux / Tuberculin skin test** (purified protein derivative {PPD})
  
  A positive test can be observed, one to 3 months after infection.

  Positive in 84 – 95 % of patients who are HIV negative

  Negative in almost 20 per cent patients with active disease if the disease is disseminated, or if the patient is immunocompromised or suffering from exanthematous fever.

• ESR may be markedly elevated (neither specific nor reliable).

• **ELISA**: for antibody to mycobacterial antigen-6, sensitivity of 60 – 80%.

• **PCR**: sensitivity of 40% only.

• Brucella complement fixation test (useful in endemic areas as brucella can clinically mimic tuberculosis).
SPINAL TUBERCULOSIS

DIAGNOSIS

Microbiology studies to confirm diagnosis:

- **Ziehl-Neelsen staining:**
  a quick and inexpensive method.

- Obtain bone tissue or abscess samples to stain for acid-fast bacilli (AFB), & isolate organisms for culture & drug susceptibility.

- Culture results are available only after a few weeks.

- Positive only in 50% of cases.
SPINAL TUBERCULOSIS
DIAGNOSIS

LAB STUDIES

• IFN – Release assays (IGRAs)

Recently, two in vitro assays that measure T-cell release of IFN – in response to stimulation with the highly specific tuberculosis antigens ESAT- 6 & CFP-10 have become commercially available.
**SPINAL TUBERCULOSIS**

**DIAGNOSIS**

- **RADIOLOGICAL DIAGNOSIS**
  - 1. PLAIN RADIOGRAPH
  - 2. CT SCAN
  - 3. MRI SPINE
  - 4. BONE SCAN

TB bacilli are rarely found in CSF, therefore imaging plays pivotal role in suggesting the diagnosis.
PLAIN RADIOGRAPH

- More than 50% of bone has to be destroyed before a lesion can be seen on X-ray. This process takes approximately six months.

- The **classic roentgen triad** in spinal tuberculosis is primary vertebral lesion, disc space narrowing and paravertebral abscess.

- **Typical tubercular spondylitic features in long standing paraspinal abscesses** =
  a) produce concave erosions around the anterior margins of the vertebral bodies producing a scalloped appearance called the **Aneurysmal phenomenon**.
  b) fusiform paraspinal soft tissue shadow with calcification in few.

- **Skip lesions** as involvement of non contiguous vertebrae (7 – 10 % cases).

- **DEFORMITIES:**
  1. Anterior wedging
  2. Gibbous deformity.
  3. Vertebra plana = single collapsed vertebra.
**CT SCAN**

- Patterns of bony destruction.
- Calcifications in abscess (pathognomic for Tb)
- Regions which are difficult to visualize on plain films, like:
  1. Cranio-vertebral junction (CVJ)
  2. Cervico-dorsal region,
  3. Sacrum
  4. Sacro-iliac joints.
  5. Posterior spinal tuberculosis because lesions less than 1.5cm are usually missed due to overlapping of shadows on x rays.

**MRI**

- Lack of ionizing radiation, high contrast resolution & 3D imaging.
- Detect marrow infiltration in vertebral bodies, leading to early diagnosis.
- Changes of discitis
- Assessment of extradural abscesses / subligamentous spread.
- Skip lesions
- Spinal cord involvement.
- Spinal arachnoiditis.
LOCATION OF VERTEBRAL BODY DESTRUCTION

PATTERNS OF VERTEBRAL BODY DESTRUCTION ON CT:

1. Fragmentation
   with pathologic fracture
   without pathologic fracture
2. Sequestra within a lucent defect
3. Reactive sclerosis
MRI

- MRI is the modality of choice as delineates leptomeningeal disease better, direct evaluation of intramedullary lesions, associated osseous signal change and epidural abscesses.

- Typical (spondylo-discitis) and atypical (spondylitis without discitis) types.
Patterns of vertebral involvement

- The primary focus of infection in the spine can be either in the vertebral body or in the posterior elements.

- Four patterns:
  - Paradiscal (Commonest)
  - Central
  - Anterior, &
  - Appendiceal
1. Paradiscal Lesions:

- Most common pattern of spinal tuberculosis.
- It is adjacent to the I/V disc leading to a narrowing of the disc space.
- Disk space narrowing is caused either by destruction of subchondral bone with subsequent herniation of the disc into the vertebral body or by direct involvement of the disc.
- MR imaging shows low signal on T1-weighted images and high signal on T2-weighted images in the endplate, narrowing of the disc, and large paraspinal and sometimes epidural abscesses.
SPINAL TUBERCULOSIS

Paradiscal Lesions
2. **Anterior Lesions:**

- The anterior type is a subperiosteal lesion under the ALL.
- Pus spreads over multiple vertebral segments, stripping the periosteum and ALL from the anterior surface of the vertebral bodies.
- The periosteal stripping renders the vertebrae avascular and susceptible to infection.
- Both pressure and ischemia combine to produce anterior scalloping.
SPINAL TUBERCULOSIS

• Collapse of the VB & diminution of the disc space is usually minimal & occurs late.

• This lesion is relatively more common in thoracic spine in children.

• MR imaging shows the subligamentous abscess, preservation of the discs, and abnormal signal involving multiple vertebral segments representing vertebral tuberculous osteomyelitis.
SPINAL TUBERCULOSIS

Anterior Lesions
SPINAL TUBERCULOSIS

3. **Central Lesions**:  
- Centred on the vertebral body.  
- Disc is not involved.  
- Infection starts from the center of the vertebral body; reaches there through Batson’s venous plexus or through posterior vertebral artery.  
- Vertebral collapse can occur, producing a vertebra plana appearance.  
- MR imaging shows a signal abnormality of the vertebral body with preservation of the disc.  
- **DD**: *The appearance is indistinguishable from that of lymphoma or metastasis.*
SPINAL TUBERCULOSIS

Central Lesions
SPINAL TUBERCULOSIS

4. **Appendicular type**:

- Isolated infection of the pedicles & laminae (neural arch), transverse processes, & spinous process.
- Uncommon lesion (< 5%).
- Occur in isolation or in conjunction with the typical paradiscal variant
- Radiographically, they appear as erosive lesions, paravertebral shadows with intact disc space.
- Rarely, present as synovitis of facet joints.
SPINAL TUBERCULOSIS

Appendicidal lesion
**Paravertebral abscess:**

- With collapse of the vertebral body, tuberculous granulation tissue, caseous matter, and necrotic bone accumulate beneath the anterior longitudinal ligament.

- Gravitate along the fascial planes and present externally at some distance from the site of the original lesion.

- In lumbar region, along the psoas fascial sheath points into the groin just below the inguinal ligament.

- In thoracic region, the longitudinal ligaments limit the abscess, which is seen in the radiogram as a fusiform radiopaque shadow at or just below the level of the involved vertebra or may reach the anterior chest wall in the parasternal area by tracking via the intercostal vessels.
LOCATION OF PARAVERTEBRAL ABSCESS
DEFORMITIES IN SPINAL TUBERCULOSIS

• "POTT’S SPINE": Bony deformities occur in tubercular spine

• Kyphotic deformity (more common in thoracic spine) occurs as a consequence of collapse in the anterior spine.

• Collapse is minimal in cervical spine because most of the body weight is borne through the articular processes.

• Knuckle Kyphosis: forward wedging of one or two VB causing small kyphos.

• Angular Kyphosis: wedge collapse of 3 or more VB
Fig. 23.2: Method of measurement of angle of kyphosis (Dickson 1967). A line is drawn along the posterior margins of the bodies of the healthy vertebrae above and below the site of disease; angle 'K' is the angle of kyphosis. Angle 'K' increases with increase in the degree of kyphosis. Another method is by determining the angle between the upper end-plate of the normal vertebra proximal to the affected vertebrae and the lower end-plate of the normal vertebra distal to the affected vertebrae (c).
INTRADURAL EXTRAMEDULLARY INVOLVEMENT

1. **Intradural extramedullary tuberculoma**

   - Uncommon form of tuberculosis with only 18 cases have been reported in literature till date.

   - Intradural extramedullary tuberculomas can further be separated into two groups:

     1. The first type comprises of hard rounded lesions generally 2 to 3 cm in diameter, having relatively thin membranes often containing granular calcified material, and are generally attached to dura but are relatively easily separated from the underlying cord.

     2. The second form is diffuse involvement of subdural space by masses of relatively avascular grayish tubercular granulation tissue.
2. **Spinal Arachnoiditis**

- Frequently involves the spinal cord, meninges and the nerve roots and is more appropriately referred to as radiculomyelitis (TBRM).
  
  Should be suspected when patient develops spinal cord symptoms.

- **PATHOGENESIS:**
  
  1. Hematogenous spread from extra CNS source.
  3. Secondary intraspinal extension from tuberculous spondylitis.

  Gross granulomatous exudates fill the subarachnoid space.

  With time exudates get organized and fibrin coated nerve roots adhere to each other.

  Vasculitis of spinal arteries may cause spinal cord ischemia.
The MR imaging features of spinal tuberculous arachnoiditis consist of:

1. CSF loculation and obliteration of the spinal subarachnoid space.
2. Loss of outline of the spinal cord against CSF in spinal canal on T1.
3. Matting of the nerve roots in the lumbar region.
4. Contrast-enhanced imaging reveals nodular, thick, linear intradural enhancement which can completely fill the subarachnoid space, sometimes giving the appearance of a normal unenhanced MR image.

Key point: when T1 contrast enhanced images look like T2 images.
Syringomyelia can occur as a complication of arachnoiditis.

- Early syrinx formation is due to spinal cord ischemia
- Late onset syrinx in chronic arachnoiditis is due to focal scarring of the subarachnoid space by adhesions which impedes free circulation of CSF thus forcing CSF into the central canal of the spinal cord via VR spaces

- **The differential diagnosis of nodular or diffuse thickening in spinal canal on MRI:**
  
  1. meningeal carcinomatosis.
  2. lymphoma.
Intradural intramedullary involvement

- Intramedullary *tuberculomas* is due to hematogenous spread from other primary site in the body.
- Predominantly in the young patients.
- Thoraco-lumbar region is the most common site of involvement.
- MRI shows low or intermediate signal intensity on T1W images and low signal on T2W images (Low signal on T2W images is due to caseous necrosis in the tuberculoma, which has high protein content).
- Post Gadolinium study shows ring enhancement.
- Sometimes associated with changes of myelitis with altered cord signal intensity.
<table>
<thead>
<tr>
<th>STAGE</th>
<th>CLINICO-RADIOLOGICAL FEATURE</th>
<th>DURATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Pre-destructive</td>
<td>straightening of curvatures</td>
<td>&lt; 2 months</td>
</tr>
<tr>
<td></td>
<td>Spasm of perivertebral muscles</td>
<td></td>
</tr>
<tr>
<td></td>
<td>MRI: marrow edema</td>
<td></td>
</tr>
<tr>
<td>2) Early destructive</td>
<td>decreased disc space+ paradiscal erosion</td>
<td>2-4 months</td>
</tr>
<tr>
<td></td>
<td>MRI: marrow edema; break of osseous margin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>CT: marginal erosion or cavitations</td>
<td></td>
</tr>
<tr>
<td>3) Mild angular kyphosis</td>
<td>2-3 vertebrae involvement</td>
<td>4-9 months</td>
</tr>
<tr>
<td></td>
<td>(K: 10-30 degree)</td>
<td></td>
</tr>
<tr>
<td>4) Moderate angular kyphosis</td>
<td>&gt; 3 vertebral involvement</td>
<td>6-24 months</td>
</tr>
<tr>
<td></td>
<td>(K: 30-60 degree)</td>
<td></td>
</tr>
<tr>
<td>5) Severe kyphosis</td>
<td>&gt; 3 vertebrae involvement</td>
<td>&gt; 2 years</td>
</tr>
<tr>
<td></td>
<td>(K: &gt; 60 degree)</td>
<td></td>
</tr>
</tbody>
</table>

(K: angle of Kyphosis)
BONE SCAN (Technitium (Tc) – 99 m)

- Increased uptake in up to 60 per cent patients with active tuberculosis.
- >= 5mm lesion size can be detected.
- Avascular segments and abscesses show a cold spot due to decreased uptake.
- Highly sensitive but nonspecific.
- Aid to localise the site of active disease and to detect multilevel involvement.
DIFFERENTIAL DIAGNOSIS

The differential diagnosis of the tuberculous spine includes:

1. SPINAL INFECTIONS- pyogenic, brucella & fungal.
2. NEUROPATHIC spine
3. NEOPLASTIC commonly lymphoma/ metastasis
4. DEGENERATIVE

No pathognomonic imaging signs allow tuberculosis to be readily distinguished from other conditions. Biopsy is definitive.
DD: Pyogenic Spondylitis

<table>
<thead>
<tr>
<th><strong>Tubercular</strong></th>
<th><strong>Pyogenic</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Long standing history of months to years</td>
<td>History of days to months.</td>
</tr>
<tr>
<td>Presence of active pulmonary tuberculosis - 60%</td>
<td>Not present.</td>
</tr>
<tr>
<td>Most common location thoracic spine followed by thoraco-lumbar region.</td>
<td>Most common location lumbar spine.</td>
</tr>
<tr>
<td>&gt; 3 contiguous vertebral body involvement common - 42%</td>
<td>19% only. Mostly involves 1 spinal segment – 2 vertebrae &amp; intervening disc.</td>
</tr>
<tr>
<td>Vertebral collapse - 67%</td>
<td>21% only.</td>
</tr>
<tr>
<td>Bone destruction : 73%</td>
<td>48%</td>
</tr>
<tr>
<td>Posterior elements involvement common</td>
<td>Rare</td>
</tr>
<tr>
<td>Skip lesions common</td>
<td>Rare</td>
</tr>
</tbody>
</table>
**TUBERCULAR**

- Disc is involved with less frequency and severity. Disc spared in central type TB.

- Paraspinal and epidural abscesses-60%

1. large involving many contiguous vertebral bodies level.

2. calcification if present is pathognomonic.

3. Smooth rim enhancement -74%

- TO SUMMARISE: atypical features + abscess character.

**PYOGENIC**

- Disc destruction is most often seen in pyogenic osteomyelitis.

- 30%

1. Rare

2. Not seen.

3. Heterogenous enhancement. Thick irregular Rim enhancement only 9% cases.
A well-defined paraspinal lesion with abnormal signal intensity; a thin, smooth ring enhancing abscess wall; subligamentous spread to three or more vertebral levels; and multiple/skip vertebral bodies or entire-body involvement are findings more suggestive of tuberculous spondylitis than of pyogenic spondylitis.
DD: BRUCELLA SPONDYLITIS

1. Predilection for the *lumbar spine*.

2. *Intact vertebral architecture* despite evidence of diffuse vertebral osteomyelitis.


4. Smaller paraspinal abscesses

5. Facet joint involvement
DD: NEUROPATHIC SPINE

- Patients with diabetes mellitus, syringomyelia, syphilis, or another neuropathic disorder are prone to this disease

- **Destructive changes in the vertebral bodies**
  - large osteophytes,
  - Extensive vertebral sclerosis.
  - Loss of disk space but no hyperintensity or enhancement
  - No paraspinal soft-tissue lesion
DD: NEOPLASTIC

when 2 contiguous vertebral bodies are involved without intervening disc, it is difficult to differentiate tubercular spondylitis (central type) from neoplastic condition.

**TUBERCULAR**

- A destructive bone lesion associated with a *poorly defined vertebral body endplate*, with or without a loss of disk height, suggests an infection, which has a better prognosis.

**LYMPHOMA/ METASTASIS**

- The saying "*good disk, bad news; bad disk, good news*" describes the idea that a destructive bone lesion associated with a *well-preserved disk space with sharp endplates* suggests neoplastic infiltration.
Most important d/d for this central type tuberculosis is a neoplasm commonly lymphoma/metastasis.
COMPICATIONS OF SPINAL TUBERCULOSIS

- Paraplegia
- Cold abscess
- Spinal deformity
- Sinuses
- Secondary infection
- Amyloid disease
- Fatality
TUBERCULOUS SPINE WITH PARAPLEGIA

• Incidence: 10 – 30%

• Dorsal spine most common

• Motor functions affected before / greater than sensory.

• Sense of position & vibration last to disappear.
PATHOLOGY OF TUBERCULOUS PARAPLEGIA

• **Inflammatory Edema:**
  Vascular stasis, Toxins.

• **Extradural Mass:**
  Tuberculous osteitis of VB & Abscess.

• **Meningeal Changes:** “Dura as a rule not involved”.

Extradural granulation → Contraction / Cicatrization → Peridural fibrosis → Recurrent Paraplegia
PATHOLOGY OF TUBERCULOUS PARAPLEGIA

• **Bony disorders**: Sequestra, Internal Gibbus

• **Infarction of Spinal Cord**:
  Endarteritis, Periarteritis or thrombosis of tributary to ASA.

• **Changes in Spinal Cord**:
  Thinning (Atrophy), Myelomalacia & Syrinx.
SEDDON’S CLASSIFICATION OF TUBERCULOUS PARAPLEGIA:

GROUP A (EARLY ONSET PARAPLEGIA) a/k/a Paraplegia associated with active disease:

• During the active phase of the disease within first 2 years of onset.

• Pathology can be inflammatory edema, granulation tissue, abscess, caseous material or ischemia of cord.

GROUP B (LATE ONSET PARAPLEGIA) a/k/a Paraplegia associated with healed disease:

• Usually after 2 years of onset of disease.

• Can be due to recrudescence of the disease or due to mechanical pressure on the cord.

• Pathology can be sequestra, debris, internal gibbus or stenosis of the canal.
**KUMAR’S CLASSIFICATION OF TUBERCULOUS PARA/TETRAPLEGIA** (Predominantly based on motor weakness)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Negligible</td>
</tr>
<tr>
<td>II</td>
<td>Mild</td>
</tr>
<tr>
<td>III</td>
<td>Moderate</td>
</tr>
<tr>
<td>IV</td>
<td>Severe</td>
</tr>
</tbody>
</table>
BASIC PRINCIPLES OF MANAGEMENT

• Early diagnosis
• Expeditious medical treatment
• Aggressive surgical approach
• Prevent deformity
• Expect good outcome
WHAT IS MIDDLE PATH REGIME?

• Rest in hard bed
• Chemotherapy
• X-ray & ESR once in 3 months
• MRI/CT at 6 months interval for 2 years
• Gradual mobilization is encouraged in absence of neural deficits with spinal braces & back extension exercises at 3 – 9 weeks.
• Abscesses – aspirate when near surface & instil 1gm Streptomycin +/- INH in solution
• Sinus heals 6-12 weeks after treatment.
• Neural complications if showing progressive recovery on ATT b/w 3-4 weeks: surgery unnecessary.
• Excisional surgery for posterior spinal disease associated with abscess / sinus formation +/- neural involvement.
• Operative debridement–if no arrest after 3-6 months of ATT / with recurrence of disease.
• Post op spinal brace → 18 months-2 years.
<table>
<thead>
<tr>
<th>Phase</th>
<th>Duration</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intensive</strong></td>
<td>5 – 6 months</td>
<td>INH + Rifampicin &amp; ofloxacin</td>
</tr>
<tr>
<td>(for replicating mycobacteria)</td>
<td></td>
<td></td>
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<tr>
<td><strong>Continuation</strong></td>
<td>7 – 8 months</td>
<td>INH + Pyrazinamide x 3-4 months f/b Rifampicin x 4-5 months</td>
</tr>
<tr>
<td>(forPersisters, slow growing or dormant or intracellular mycobacteria)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Prophylactic</strong></td>
<td>4 – 5 months</td>
<td>INH + Ethambutol</td>
</tr>
</tbody>
</table>
VARIOUS REGIMES

- 3(HRZE) / 3(HRZS) + 3(HRZ) + 12(HR)

  Pediatric age group, streptomycin (for two months) replaces ethambutol to avoid optic neuropathy.

- 4(HRZE) + 14(HR)
<table>
<thead>
<tr>
<th>Treatment category</th>
<th>Intensive phase (daily or three times a week)</th>
<th>Continuation phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe forms of EPTB (category I)</td>
<td>2HRZE (2HRZS)</td>
<td>6HE</td>
</tr>
<tr>
<td></td>
<td>$2\text{H}_3\text{R}_3\text{Z}_3\text{E}_3$ ($2\text{H}_3\text{R}_3\text{Z}_3\text{S}_3$)</td>
<td>4HR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$4\text{H}_3\text{R}_3$</td>
</tr>
<tr>
<td>Less severe forms of EPTB (category III)</td>
<td>2HRZ</td>
<td>6HE</td>
</tr>
<tr>
<td></td>
<td>$2\text{H}_3\text{R}_3\text{Z}_3$</td>
<td>4HR</td>
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<tr>
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<td></td>
<td>$4\text{H}_3\text{R}_3$</td>
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</table>
**Evolution of Treatment**

**Pre-antitubercular era :**

- Artificial abscess- **Pott (1779)**
- Laminectomy & laminotomy: **Chipault (1896)**
- Costco-transversectomy: **Menard (1896)**
- **Calves** operation (1917)
- Lateral rhachiotomy of **Capener (1933)**
- Anterolateral decompression of **Dott & Alexander (1947)**
SURGICAL INDICATIONS:

• No sign of neurological recovery after trial of 3-4 weeks therapy
• Neurological complications develop during conservative treatment
• Neuro deficit becoming worse on drugs & bed rest
• Recurrence of neurological complication
• Prevertebral cervical abscess with difficulty in deglutition & respiration
• Advanced cases- Sphincter involvement, flaccid paralysis or severe flexor spasms
OTHER INDICATIONS:

- Recurrent paraplegia
- Painful paraplegia—d/t root compression, etc
- Posterior spinal disease
- Spinal tumor syndrome resulting in cord compression
- Rapid onset paraplegia (due to thrombosis, etc)
- Doubtful diagnosis & for mechanical instability after healing, &
- Cauda equina paralysis
<table>
<thead>
<tr>
<th>SURGERY</th>
<th>INDICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Decompression (+/- fusion)</td>
<td>Too advanced ds, failure to respond to conservative therapy</td>
</tr>
<tr>
<td>2 Debridement +/- decompression +/- fusion</td>
<td>Recurrence of disease or of neural complication</td>
</tr>
<tr>
<td>3 Anterior transposition of cord (Extrapleural anterolateral approach)</td>
<td>Severe kyphosis (&gt;60°) + neural deficits</td>
</tr>
<tr>
<td>4 Laminectomy</td>
<td>Extradural granuloma / tuberculoma (STS), Old healed disease presenting as secondary canal stenosis / posterior spinal disease</td>
</tr>
</tbody>
</table>
## SURGICAL APPROACHES:

<table>
<thead>
<tr>
<th>WORKERS</th>
<th>C1-C2</th>
<th>CERVICAL</th>
<th>C7-D1</th>
<th>DORSAL</th>
<th>DORSO-LUMBAR</th>
<th>LUMBAR</th>
<th>L5-S1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kirkaldy-Willis (1965)</td>
<td>-</td>
<td>Anterior</td>
<td>Transpleural through bed of 3rd rib</td>
<td>Anterolateral or transpleural</td>
<td>Anterolateral</td>
<td>Retroperitoneal sympathectomy or ureter approach</td>
<td>Transperitoneal, paramedian incision in Trendelenburg position</td>
</tr>
<tr>
<td>Hodgson (1969)</td>
<td>Transoral / transthyroid</td>
<td>Through anterior or posteriorΔ</td>
<td>Transpleural via bed of 3rd rib / split sternal for extensive lesion</td>
<td>Anterior transpleural decompression</td>
<td>Bed of 11th rib extrapleural extraperitoneal / left transpleural via bed of 9th rib</td>
<td>Renal approach</td>
<td>Transperitoneal in Trendelenburg position. Lower midline incision</td>
</tr>
<tr>
<td>Smith &amp; Robinson (1985)</td>
<td>Anterior</td>
<td>Anterior</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mc Afee et al (1987)</td>
<td>Retropharyngeal extramucosal</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Tuli et al (1988)</td>
<td>Transoral for drainage</td>
<td>Anterior</td>
<td>Low anterior cervical</td>
<td>Anterolateral or transpleural</td>
<td>Anterolateral</td>
<td>Retroperitoneal approach</td>
<td>Retroperitoneal or Retropsoas transverse vertebrotomy</td>
</tr>
</tbody>
</table>
Tuli’s recommended approach

- Cervical spine – T1
  - Anterior approach
- Dorsal spine – DL junction
  - Anterolateral approach
- Lumbar spine & Lumbosacral junction
  - Extraperitoneal Transverse Vertebrotomy
ANTERIOR APPROACH TO THE CERVICAL SPINE (C2 to D1)

Smith & Robinson

• Oblique / transverse incision.

• Plane b/w SCM & carotid sheath laterally & T-O medially.

• Longitudinal incision in ALL open a perivertebral abscess, or the diseased vertebrae may be exposed by reflecting the ALL & the longus colli muscles.

Hodgson approach via posterior triangle by retracting SCM, Carotid sheath, T & O anteriorly & to the opposite side.
SURGICAL APPROACHES TO DORSAL SPINE

- Anterior transpleural transthoracic approach (Hodgson & Stock, 1956)
- Anterolateral extrapleural approach (Griffiths, Seddon & Roaf, 1956)
- Posterolateral approach (Martin, 1970)

\{Dura is exposed by hemilaminectomy first & then extended laterally to remove the posterior ends of 2 – 4 ribs, corresponding transverse processes & the pedicles\}.
TRANSTHORACIC TRANSPLEURAL

- Left sided incision preferable
- Incision is made along the rib which in the mid-axillary line, lies opposite the centre of the lesion (i.e. usually 2 ribs higher than the centre of the vertebral lesion).
- For severe kyphosis, a rib along the incision line should be removed.
- A J-shaped parascapular incision for C7 – D8 lesions, scapula uplift & rib resection.
- After cutting the muscles & periosteum, rib is resected subperiosteally.
• Parietal pleural incision applied & lung should be freed from the parieties & retracted anteriorly.

• A plane developed b/w the descending aorta & the paravertebral abscess / diseased vertebral bodies by ligating the intercostal vessels & branches of hemiazygos veins.

• T-shaped incision over the paravertebral abscess.

• Debridement / decompression with or without bone grafting.
ANTEROLATERAL DECOMPRESSION

- **Griffith** et al -- prone position

- **Tuli** --- Right lateral position

- Advantage:
  1. avoid venous congestion
  2. avoid excessive bleeding
  3. permits free respiration
  4. Lung & mediastinal contents fall anteriorly

- Parts to remove:
  - Posterior part of rib (~8cm from the TP)
  - Transverse process (TP)
  - Pedicle
  - Part of the vertebral body
• Semicircular incision

• For severe kyphosis, additional 3-4 transverse processes and ribs have to be removed.

• Intercostal nerves serve as guide to the intervertebral foramina & the pedicles.
ANTERO-LATERAL APPROACH TO LUMBAR SPINE (LUMBO-VERTEBROTOMY)

• Left side approach

• Semicircular incision

• Expose and remove transverse process subperiosteally.

• Preserve lumbar nerves
EXTRA PERITONEAL ANTERIOR APPROACH TO LUMBAR SPINE

• 45° right lateral position with a bridge centered over the area to be exposed.

• Similar incision as nephroureterectomy or sympathectomy

• Strip peritoneum off posterior abdominal wall and kidney, preserving ureter.

• Longitudinal incision along psoas fibers for abscess drainage

• Retract the sympathetic chain

• Double ligation of lumbar vessels.
EXTRA PERITONEAL APPROACH TO
LUMBO-SACRAL REGION

• Left side preferred (left Common iliac vessels longer & retracted easily).

• Lazy “S” incision

• Strip & reflect the parietal peritoneum along with ureter & spermatic vessels towards right side.
TRANSPERITONEAL HYPOGASTRIC/SUPRAPUBIC ANTERIOR APPROACH TO LUMBO-SACRAL REGION

- Supine position
- Midline incision from umbilicus to pubis.
- Lumbo-sacral region identified distal to aortic bifurcation and left common iliac vein.
- Longitudinal incision on parietal peritoneum over lumbo-sacral region in midline.
- Avoid injury to sacral nerve & artery and sympathetic ganglion.
POSTERIOR SPINAL ARTHRODESIS

• By– Albee & Hibbs

• **Albee**– Tibial graft inserted longitudinally in to the split spinous processes across the diseased site.

• **Hibbs**– overlapping numerous small osseous flaps from contiguous laminae, spinous processes & articular facets

• **Indications**–
  1. Mechanical instability of spine in otherwise healed disease.
  2. To stabilize the craniovertebral region (in certain cases of T.B.)
  3. As a part of panvertebral operation
SURGERY IN SEVERE KYPHOSIS

• **HIGH RISK PATIENTS:**
  - Patients < 10 years
  - Dorsal lesions
  - Involvement of >= 3 vertebrae

• Severe deformity in presence of active disease, especially in children is an absolute indication for decompression, correction and stabilization.

• **Staged operations** -
  1. Anteriorly at the site of disease,
  2. Osteotomy of the posterior elements at the deformity &
  3. Halopelvic or halofemoral tractions post-operatively.
TREATMENT OF PARAPLEGIA IN SEVERE KYPHOSIS

• Griffiths et al (1956): anterior transposition of cord through laminectomy

• Rajasekaran (2002): posterior stabilization f/b anterior debridement and bone grafting (titanium cages) in active stage of disease and vice versa for healed disease.

• Antero-lateral (Preferred approach).
SURGICAL CORRECTION OF SEVERE KYPHOTIC DEFORMITY

• Fundamentals of correction:

1. to perform an osteotomy on the concave side of the curve and wedge it open (secured with strong autogenous iliac grafts).

2. to remove a wedge on the convex side and close this wedge (Harrington compression rods and hooks)
## CLINICAL FACTORS INFLUENCING PROGNOSIS IN CORD INVOLVEMENT

<table>
<thead>
<tr>
<th>CORD INVOLVEMENT</th>
<th>BETTER PROGNOSIS</th>
<th>RELATIVELY POOR PROGNOSIS</th>
</tr>
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<tbody>
<tr>
<td>Degree</td>
<td>Partial</td>
<td>Complete (stage IV)</td>
</tr>
<tr>
<td>Duration</td>
<td>Shorter</td>
<td>Longer (&gt;12 months)</td>
</tr>
<tr>
<td>Speed of onset</td>
<td>Slow</td>
<td>Rapid</td>
</tr>
<tr>
<td>Age</td>
<td>Younger</td>
<td>Older</td>
</tr>
<tr>
<td>General condition</td>
<td>Good</td>
<td>Poor</td>
</tr>
<tr>
<td>Vertebral disease</td>
<td>Active</td>
<td>Healed</td>
</tr>
<tr>
<td>Kyphotic deformity</td>
<td>&lt;60°</td>
<td>&gt;60°</td>
</tr>
<tr>
<td>Cord on MRI</td>
<td>Normal</td>
<td>Myelomalacia / Syrinx</td>
</tr>
<tr>
<td>Peroperative</td>
<td>Wet lesion</td>
<td>Dry lesion</td>
</tr>
</tbody>
</table>
CVJ TUBERCULOSIS

• Less than 1% of all spinal tuberculosis.
• Young patients (14 to 65 years), F: M = 2:1.
• Infection from primary sites (paranasal sinuses, nasopharyngeal or retropharyngeal lymph nodes) spreads retrograde via lymphatic route.

PRESENTATION:

2 months to 2 years to produce symptoms
  – Cervico-medullary compression,
  – Cranial nerve deficits
  – Atlanto-axial instability,
  – Abscess formation
? Tuberculosis of retropharyngeal space

Infiltration

Atlanto-axial ligaments +/-

Hyperemia

Bony architecture → AAD → Tuberculous granulation tissue

Compression

Cold Abscess

Gross bony destruction & angulation
DIAGNOSIS:

X-ray / CT Scan / MRI:

- Destruction of lateral masses,
- Secondary atlanto-axial subluxation
- Basilar invagination,
- Adjacent bony destruction,
- Increase in the pre-vertebral shadow/ prevertebral enhancing soft tissue mass.
- Spinal cord signal changes / compression.
## STAGES OF CVJ TUBERCULOSIS (Lifeso et al)

<table>
<thead>
<tr>
<th>STAGES</th>
<th>LIGAMENTS</th>
<th>BONY DESTRUCTION</th>
<th>C1-C2 INVOLVEMENT</th>
<th>TREATMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Intact</td>
<td>Minimal</td>
<td>No e/o anterior displacement of C1 on C2</td>
<td>Cervico-thoracic orthosis until stability</td>
</tr>
<tr>
<td>II</td>
<td>Disruption</td>
<td>Minimal</td>
<td>Anterior displacement of C1 on C2 +/- proximal translocation of odontoid</td>
<td>Halo traction for normal alignment, f/b posterior fusion &amp; Halo vest immobilization</td>
</tr>
<tr>
<td>III</td>
<td>Marked disruption</td>
<td>Marked</td>
<td>Complete obliteration of anterior arch of C1 with complete loss of odontoid process, marked AAD / A-O instability</td>
<td>Halo traction for alignment f/b posterior fusion, halo vest immobilization until stability</td>
</tr>
</tbody>
</table>
TREATMENT

CONSERVATIVE:

- Absolute bed rest
- Cervical traction for unstable spine
- Prolonged external immobilization
- ATT X 18 months

SURGERY:

- Gross bony destruction with instability
- Abscess formation
- Severe or progressive neurological deficit
- Unstable spine following conservative therapy (failed therapy)
- Doubtful diagnosis (esp. with neoplasm)
PREVENT DISEASE

CARELESS SPITTING, COUGHING, SNEEZING, SPREAD INFLUENZA AND TUBERCULOSIS