INTRODUCTION

Spinal tuberculosis infections (TB) have been reported as early as the time of Egyptian mummies, and encompass almost 1% of the tuberculosis cases. It may have wide range of presentations that not only affect the spinal cord and its covering but also the adjacent bony and soft tissue elements. The need for accurate and prompt diagnosis and treatment has improved the outcome in terms of both deformity and neurology (24).

Spinal TB infections today occur in endemic areas with increasing frequency in all age groups. In young adults, the disease appears to have increased in recent decades because of immunodeficiency syndromes and intravenous (IV) drug abuse, especially in the developing countries.

PRESENTATION OF SPINAL INFECTION

It may present as the classic form or spondylodiscitis, and an increasingly common atypical form which is spondylitis without disc involvement (16). In adults it usually involves the end plates while in children, it can attack the discs directly as the disks are more vascular. The infection may involve the adjacent structures, and form large collections or cold abscesses, and cause symptoms by bony destruction, deformity, and pressure, by epidural abscess, or granulation tissue (5). Neurologic deficits may result from neural compression.

The patient commonly presents with back pain, along with deformity and neurological symptoms, depending upon the level. The disorder may be accompanied with other constitutional symptoms, like fever, weight loss, and night sweats, etc.

The disease is endemic in developing and underdeveloped countries, and may be the first differential in most of the cases. The classic clinical presentation of spinal tuberculosis (Triad of Pott) includes Gibbus, spinal abscess and paraparesis.

RISK FACTORS

Other than living in an endemic area, a compromised immune status was identified with certain risks factors in over 50% of such patients. These include chronic renal failure, diabetes mellitus, alcoholism, IV drug abuse, malignancy, recurrent UTI, Pott’s disease and positivity for HIV. Chronic steroid use, recent spinal procedure and trauma (such as gunshot wound, etc.) are also risk factors.

LABORATORY WORK UP

No single laboratory study is pathognomonic of this condition. CBC shows leukocytosis. The Mantoux test is not very helpful in endemic areas. ESR is usually elevated >30. CRP may also be raised. PCR may be helpful once the samples have been taken by needle biopsy for culture and histopathology, although the yield of cultures ranges from...
45-95 % in different studies. Nowadays, serum and CSF cytokines, and matrix metalloproteinases and monocytes are being experimented with and may have a role in the diagnosis (15).

**RADIOGRAPHIC DIAGNOSIS**

Plain X-ray films usually show bony destruction and kyphosis, and some times with large psoas abscesses may also be seen. Osteomyelitis of adjacent vertebral bodies is seen more commonly in infections anterior to dura. There is evidence of lytic lesions, demineralization, and scalloping of end plates that may take about 4 to 6 weeks after onset of infection.

CT scan is better in defining these subtle osseous changes, more than one level may be seen involved with a predilection for the vertebral body and disc space (sparring posterior elements), with sclerosis of the involved vertebral body endplates. It is important to remember that disc space is most often involved in TB osteomyelitis, where as it is spared in primary or metastatic tumors. (Nucleus pulposus is avascular providing a haven for the mycobacterium).

"A bad disc is a good sign whereas a good disc is a bad sign"

MRI is the imaging study of choice. It not only helps in excluding other differentials (such as herniated disc, hematoma, neoplasm, and transverse myelitis) but also provides better anatomical details including its rostro-caudal extension. There is a decrease in signal intensity of the involved bone and soft tissues on T2-weighted images and the increase in intensity of a uniform thin rim enhancement is a pathognomonic finding suggesting either caseation necrosis or a cold abscess in tuberculosis. It is also helpful to check for the response to the treatment (1,3,7,8,15). PET Computed Tomography may play a role in early detection and in cases where we have atypical multiple lesions.

**CLASSIFICATION**

Throughout history multiple classification systems have been used to quantify the degree of deformity and anterior and posterior involvement along with neurological involvement to plan for the best treatment option. Kumar introduced a 4-point classification system based on stage of disease and site of involvement in 1985. Mehta and Bhojraj in 2001 gave a classification system based on MRI findings and classified in 4 groups, but it lacked to address areas other than dorsal spine. Orguz et al classified TB spine as follows:

1. Type I, one-level disc involvement and soft tissue infiltration without abscess, collapse and neurologic deficit.
2. Type I-A, lesions only limited to vertebra and therefore, manageable with fine needle biopsy and medical therapy.
3. Type I-B, abscess formation exceeds the vertebra and the treatment is debridement using an anterior, posterior or endoscopic approach.
4. Type II, one- or two-level disc degeneration, abscess formation and mild kyphosis correctable with an anterior surgery (14,18).

**MANAGEMENT GOALS**


Prevent or reverse neurological deficit. Relieve pain

Establish spinal stability

**TREATMENT**

Treatment is required after confirmation of the diagnosis, if there is controllable pain, with no increasing deformity, and stable neurological deficit, then a trial of conservative management can be given and surgery is nor necessary, in case there is increasing deformity, increasing neurological deficit, on increasing pain, then surgical intervention is required, with debridement and stabilization (1,2,4,10,14,18).

**NON-OPERATIVE TREATMENT**

This involves immobilization till the pain improves or the instability is excluded. Later mobilization is with brace therapy. ATT is continued for a minimum of 9-18 months, depending on the extent of the infection and organism.

**MEDICAL THERAPY**

Medical therapy with anti tuberculous drugs is the cornerstone in Pott's spine. The standard drugs consists of; Isoniazid (5 mg/kg; maximum 300 mg/day), Rifampicin 15 mg/kg; maximum 600 mg/day, Ethambutol (15 – 25 mg/kg; maximum 2 g/day), Pyrazinamide (15 – 30 mg/kg; maximum 2 g/day). Pyridoxine was also added to the regimen prophylactically. This regimen is given for two months followed by withdrawal of Pyrazinamide and Ethambutol for the rest of the period. The period ranges from 9-18 months (1,2,4,9,10,16,20-24,26). In case of MDR TB 6 drugs may be required with the duration of up to 18 to 24 months.
OPERATIVE TREATMENT

Surgery may be necessary in about one-eighth of the patients. The options include: radical debridement with or without bone grafting, or radical debridement, bone grafting along with instrumentation. Indications for surgical intervention are to obtain diagnosis when closed biopsy is unsafe or is unsuccessful, clinically significant abscess with cord compression, progressive neurological deficits, significant deformity of vertebral body, and in medical refractory cases (i.e. high ESR, persistent pain and progressive neurology). An increasing number of articles indicate that instrumentation is not contraindicated in cases where radical debridement is achieved. The additional stability instead promotes clinical resolution of the infection and related symptoms.

The optimal approaches are either anterior approach, posterior approach or a combined approach. Anterior approach is usually recommended to access for debridement, grafting and stabilization with reconstruction of anterior column. Posterior approach may be adequate in lumbar spine.

The correction of kyphosis is necessary as in the long term even stable patients will progress to increasing deformity. In children the correction is more important because a growing skeleton causes increased deformity (17). In types I, II, and III, progression occurs approximately in 39%, 44%, and 17% of pediatric cases respectively, during the growth spurt. A spinal instability score higher than 2 seems to reliably predict patients who will have an increase of more than 30° kyphosis and a final deformity greater than

<table>
<thead>
<tr>
<th>TYPE</th>
<th>LESION</th>
<th>TREATMENT</th>
<th>SAMPLE</th>
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</table>
| Type I | A | The lesion located in vertebra, one level disc degeneration, no collapse, no abscess, no neurologic deficits. | Fine needle biopsy and drug therapy | [Image]
| | B | Abscess formation, one or two level disc degeneration, no collapse, no neurologic deficit | Abscess drainage & debridement | [Image]
| Type II | Vertebral collapse (pathological fracture) | 1. Anterior debridement & fusion | [Image] |
| | Abscess formation | 2. In existence of neurologic deficit decompression should be added | |
| | Kyphosis (correctable with anterior surgery) | 3. Strut cortical graft is used for fusion | |
| | Stable deformity, with or without neurological deficit. Sagittal index < 20° | | |
| Type III | Severe vertebral collapse | 1. Anterior debridement & fusion | [Image] |
| | Abscess formation, Severe kyphosis | 2. Decompression | |
| | Instable deformity, with or without neurological deficit. Sagittal index ≥20° | 3. Correction of deformity & internal fixation (anterior, posterior or both) | |

Table 1: GATA CLASSIFICATION, E Oguz et al. Int Orthop. 2008 Feb; 32(1): 127-133
Table 2: WHO recommended treatment regimens for different disease categories [14]

<table>
<thead>
<tr>
<th>Disease category</th>
<th>Tuberculosis patient definition</th>
<th>Treatment regimen</th>
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<tbody>
<tr>
<td>I</td>
<td>New smear positive</td>
<td>Initial phase (daily or three times weekly): 2 HRZE</td>
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<tr>
<td></td>
<td>New smear negative with extensive parenchymal involvement</td>
<td>continuation phase (daily or three times weekly): 4 HR or 6 HE daily</td>
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<tr>
<td></td>
<td>New severe extra pulmonary tuberculosis or severe concomitant HIV infection</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>Previously treated sputum</td>
<td>2 HRZE/1 HRZE</td>
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<tr>
<td></td>
<td>Smear-positive pulmonary tuberculosis</td>
<td>5 HRE</td>
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<tr>
<td></td>
<td>- Relapse</td>
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<td></td>
<td>- Treatment after interruption</td>
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<tr>
<td></td>
<td>- Treatment failure</td>
<td></td>
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<tr>
<td>III</td>
<td>New smear-negative pulmonary tuberculosis</td>
<td>2 HRZE</td>
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<tr>
<td></td>
<td>Extra pulmonary tuberculosis</td>
<td>4 HR or 6 HE daily</td>
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<tr>
<td>IV</td>
<td>Chronic and MDR tuberculosis</td>
<td>Specially designed standardized or individualized regimens</td>
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Figure 1: MRI sagittal view T2WI of cervicodorsal spine showing tuberculous vertebral osteomyelitis with collapse and anterior cord compression at two levels with a normal segment in between (skip lesions).

Figure 2: Gibbus seen on the lower back of a patient with Pott’s spine.
60°, the posterior approach is not sufficient to correct severe deformity with kyphosis and Kyphosis angle of more than 90 deg.

New techniques like 360 deg fusion, wedge osteotomies both anterior and posterior and pedicle subtraction osteotomies have been done for correction of these extreme cases with poor neurological results (11-13, 17, 20-22, 24-26).

**Figure 3:** MRI sagittal view T2WI of dorsolumbar spine showing vertebral collapse at D12 with hyperintense signal intensity of the disc space above and below, and anterior to the vertebral body, along with compression at the conus and kyphotic deformity.

**Figure 4:** MRI axial contrast view showing abscess formation within the vertebral body and para spinal region with anterior cord compression and canal compromise.

**Figure 5:** MRI sagittal and axial views of lumbosacral spine showing erosion of disc at L3/4 with fragmentation at adjacent disc level. Evidence of high signals in left para vertebral region representing an abscess. This patient had Pott's spine and was managed conservatively on anti-tuberculous therapy.
**Prognosis:**

With new diagnostic modalities, and proper, medical and appropriate surgical treatment, the disease has a favorable outcome even in the presence of deformity and neurological deficits which are usually reversible up to 75%.

Personal series of 350 patients who underwent surgery over 11 years, only 3 patients have remained plegic despite 25% presenting with plegia.

**Figure 6:** Cervical tuberculous osteomyelitis managed surgically. Left most is X-ray cervical spine lateral view showing collapse at C4 with soft tissue swelling anteriorly, middle one showing collapse with evidence of abscess formation anteriorly and severe cord compression. Right most X-ray showing surgical fusion and fixation of the level involved after anterior decompression.

**Figure 7:** Left most showing MRI axial view of dorsal Pott's spine. Middle and right most are intra operative pictures of evacuating pus after performing thoracotomy and opening the wall of abscess.
REFERENCES


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