Spinal Tuberculosis Treatment: An Enduring Bone of Contention

Divyani Garg, Vinay Goyal[1]

Department of Neurology, Lady Hardinge Medical College, New Delhi, [1]Department of Neurology, Institute of Neurosciences, Medanta, Gurugram, Haryana, India

Abstract

Spinal tuberculosis is the most common form of extrapulmonary tuberculosis. It is of great importance to neurologists because of the potentially devastating complication of paraplegia, which may set in during active disease or the healed phase. Due to the deep-seated nature of the disease, definitive diagnosis is often challenging. There is no clear consensus on the appropriate duration of therapy for spinal tuberculosis, with various guidelines recommending treatment from as short as 6 months to up to 18 months. In this article, we present a critical appraisal of the evidence on the same. In our opinion, the duration of antitubercular therapy needs to be individualized and the decision to terminate therapy should be multifactorial (clinical, radiological, pathological/microbiological where possible) rather than being enmeshed within any particular guideline.

Keywords: Antitubercular therapy, Pott's disease, spinal tuberculosis

Introduction

Spinal tuberculosis (TB) accounts for 1–2% of all cases of TB and is a common extrapulmonary form. Musculoskeletal TB comprises ten percent of all TB cases, 50% of which are spinal.[1,2] Although it is primarily a skeletal disease, secondary involvement of the nervous system may lead to diverse neurological disabilities. The incidence of neurological complications in spinal TB ranges variously from 10 to 41%.[3] Paraplegia is the most dreaded complication of this disease. It affects the intervertebral disc space and adjacent vertebral bodies, leading to skeletal deformities. Considering the potentially devastating nature of the disease, antitubercular regimens for treatment of spinal TB have characteristically been longer duration, usually ranging between 9 and 24 months or more. Although various international guidelines deem 6 months to be sufficient, they do provide provisions for extended therapy beyond the guidelines, based on the clinical scenarios. In light of a recently published randomized trial comparing 6 months versus 12 months of antitubercular therapy (ATT) to treat definitively diagnosed (pathological or radiologically diagnosed) spinal TB,[4] we reexamine the evidence on this issue and challenges in treatment.

Spinal Tuberculosis: Why should we be Worried?

Tubercular involvement of the dorsal vertebral column poses a potential threat as the spinal canal in this region is narrow. Additionally, the physiological kyphosis at the thoracic level pushes tubercular tissue into the spinal canal causing compressive myelopathy. A tubercular abscess may enter the spinal canal also via the intervertebral foramen. In the lumbar region, the abscess tends to enter the psoas muscle.[5] Another uncommon issue is of multilevel noncontiguous involvement of the vertebrae by TB without the involvement of adjoining intervertebral discs or vertebral bodies.[6] However, this condition has not been associated with drug resistance or HIV status or chronic disease duration. The treatment regimen also does not differ. The only additional caveat in this condition is that surgical planning may need care due to multiple levels of involvement.

Spinal TB is seeded by hematogenous spread, either from a pulmonary or genito-urinary source.[7] This may be via arterial or venous circulation. The subchondral arterial plexus, derived from the anterior and posterior spinal arteries, facilitate the spread of the infection to a region adjacent to the vertebral disc. Batson's venous plexus also transmits infection between vertebrae. Central vertebral body infection may occur via an intraosseous venous system. Hence, the infection usually begins in the anteroinferior vertebral body from where it spreads to the central vertebral body. The central body of involvement usually spares the intervertebral disc due to the segmental nature of spinal arteries supplying two adjacent vertebrae, explaining the area of two adjacent vertebral involvement in TB. Disc-based involvement is common in younger patients due to its rich vascular supply which reduces with age. Hence, the pattern of involvement in older individuals tends to be central body.[8] Due to the collapse of various spinal structures, a skeletal deformity in the form of gibbus is produced.[1]

Address for correspondence: Dr. Vinay Goyal,
Institute of Neurosciences, Medanta, Gurugram, Haryana, India.
E-mail: drvinaygoyal@gmail.com

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Spinal TB may be complicated by spinal tuberculoma, myelitis, myeloradiculopathy, syrinx, vertebral TB and spinal abscess. The upper lumbar and lower thoracic spine are most commonly affected. Paraplegia is the most dreaded complication of spinal TB. As per Hodgson's seminal paper, paraplegia may be classified as paraplegia of active disease and paraplegia of healed disease.[9] In an active disease, mechanical instability and inflammation (abscess, caseous or granulomatous tissue) results in cord compression. The spinal cord may also develop edema as well as myelomalacia. Tuberculous endarteritis affecting the spinal arteries may also lead to myelopathy. In healed disease, long-standing deformities, dural fibrosis, and constriction lead to mechanical changes in the spinal cord, contributing to myelopathy. Spinal cord develops edema and even a secondary syrinx may form in long-standing cases.[2,5,8] Mechanisms of paraplegia in spinal TB are summarized in Table 1.

Cold abscesses are collections of pus that arise from tuberculous vertebrae (usually paravertebral in location) and lack an associated inflammatory response, which may occur in up to 70% of spinal TB.[10] Clinical effects are consequent to mass effect and depend on the location of the cold abscess. Even longitudinally extensive transverse myelitis has been reported in spinal TB.[11]

Spinal TB may occur in an isolated fashion or combination with TB elsewhere. Up to 30% of patients may have concomitant pulmonary disease. In retrospective series of 597 patients with spinal TB, 38 had associated extrapulmonary involvement: meningitis (8), joints (6), lymph nodes (2), genito-urinary TB (20), TB of rib (1) and splenic TB (1). This is also important to identify as associated sites may offer a more convenient sampling of tissue.

How Effective is Antitubercular Therapy in Spinal TB?

Antitubercular drugs have good penetration into vertebrae affected by TB. The effectiveness of three ATT drugs, isoniazid (INH), rifampicin, and pyrazinamide has been evaluated in tuberculous vertebral lesions. It has been determined that in patients who do not have a sclerotic wall around the tuberculous lesion, INH reaches bactericidal concentrations and rifampicin and pyrazinamide reach minimal inhibitory concentration.[12] However, in patients who have a sclerotic rim around the tuberculous focus, drugs do not penetrate within four mm of the osseous sclerotic rim, which hence necessitates surgical removal.[13] The success of ATT alone in the absence of surgery is high, ranging from 82 to 95%.[14] Even in patients with paraplegia, recovery (pain, neurological deficits as well as spinal deformity) may occur in 40% of the cases with medical management alone.[15]

What about Multi-Drug Resistant Spinal TB?

Like central nervous system (CNS) TB, diagnosis of drug-resistant spinal TB is challenging because not only is the disease deep-seated, it is also paucibacillary in nature, making procurement of tissue onerous for pathological and microbiological diagnosis. Acid-fast bacilli are demonstrable in only 10 to 30% of cases.[16] Repeat sampling is certainly difficult. As a result, Prof. Tuli had defined certain clinical criteria to suspect drug resistance in spinal TB.[17] According to these, in a patient with spinal TB who has been on ATT for at least 5 months, resistance should be suspected in the presence of poor clinical and radiological response, the appearance of a new tubercular lesion, worsening of spinal deformity, formation of a discharging sinus, and dehiscence of the previous scar of surgery for spinal TB.

Li *et al*.[18] from China reported the rate of drug resistance in histologically definite spinal TB to be as high as 30.7%. They also reported an average delay of 8.43 ± 2.12 months in the diagnosis of drug-resistant spinal TB. This was similarly 30.3% in the study by Xu *et al*., with the average delay in the diagnosis being 8.52 ± 6.15 months, and additionally 8.25 ± 2.76 months in case of drug-resistant spinal TB.[19] In India, these were reported to be 11.7% (for multi-drug resistance)[20] and 16.2% (resistance for at least one drug)[21] in two studies. In another retrospective study from a tertiary center in the southern part of India, 243 patients admitted over a period of 14 years (up to 2014) were analyzed to assess changing trends in the presentation of central and spinal TB.[22] This study observed an increasing occurrence of spinal TB compared to CNS TB which showed a declining trend. Additionally, there was the emergence of drug resistance up to 37%, particularly in spinal TB. These considerable rates of drug resistance even in spinal TB suggest that all patients of spinal TB should ideally be treated based on drug sensitivity reports. However, in a resource-limited country such as ours with inaccessibility to universal drug sensitivity testing, patients are often empirically

Table 1: Mechanisms of paraplegia in spinal tuberculosis[8]

Paraplegia of active disease (early-onset paraplegia)	
Mechanical factors	Compression due to tuberculous granulation tissue, abscess, vertebral instability, concertina collapse, gibbus
Tuberculous granuloma	Tuberculomas in intramedullary or extramedullary space
Tuberculous myelitis	Due to abnormal immune activation; uncommon
Tuberculous arachnoiditis	Meningeal thickening, fibrosis, and inflammation with nerve root entrapment
Vascular: spinal artery thrombosis	
Paraplegia of healed disease (late-onset paraplegia)	
Bony ridge causing spinal cord transection	Occurs due to severe kyphosis
Pachymeningitis	Fibrotic, thickened dura mater surrounding cord

treated based on clinico-radiological findings. Drug resistance is probed only in cases of suspected drug failure. Patients may even be empirically initiated on second-line ATT on presumptive drug resistance. Due to the paucibacillary nature of spinal TB, even patients with drug failure may be culture negative. In addition, the tissue has a higher diagnostic yield than pus.[15] Chen *et al*.[23] from Taiwan have, however, given pointers to clinically aid spinal TB diagnosis. They identified five key pointers: predisposing factors for spinal TB, symptoms favoring spinal TB, appropriate radiological features, laboratory tests, and clinical findings.

Due to these challenges, even in the absence of drug sensitivity reports, but with the appropriate clinical picture, patients are deemed to be clinically drug-resistant and may be treated as multidrug-resistant spinal TB. However, attempts to obtain tissue sampling should be made as often as possible. This may be done either through percutaneous aspiration or surgical debridement. Whenever possible, surgical debridement should be preferred, not only to procure sufficient tissue and pus but also to reduce bacteriological lesion load. Some role for immunotherapy has also been posited. Gupta *et al*.[24] evaluated the role of immunotherapy for non-responders in spinal TB. Fourteen non-responders on ATT for spinal TB deemed non-responders were administered an immunotherapy regimen incorporating a single intramuscular injection of vitamin D 600,000 IU, 200 mg daily of albendazole for 3 days, and intramuscular salmonella and influenza vaccine, in addition to ATT. Thirteen patients showed a good clinical response in terms of dependence and ambulation, although not objectively quantified.

Length of Drug Regimens for Spinal TB

The duration of drug regimen, as well as the number of drugs that should be used for spinal TB, have long been a matter of debate. This is because there is no appropriate definition for "healed status" and what parameters this definition should be based on. Repeat histological sampling at the end of a defined duration of therapy constitutes ideal proof of cure. However, this is not practical in spinal TB. The World Health Organization (WHO) guidelines for the treatment of TB indicate treatment as per the category.[25] Spinal TB belongs to category I and, as such, necessitates treatment in two phases: the intensive phase and the maintenance phase. In the intensive phase that lasts for 2 months, four first-line antitubercular drugs are administered: INH, rifampicin, pyrazinamide, and streptomycin. In the continuation phase, two drugs (INH and rifampicin) are given for 4 months. However, for bone/joint TB, the WHO recommends extending treatment for a total of 9 months. This is due to the potentially serious nature of complications as well as difficulty in assessing response in these conditions. As per the American Thoracic Society (ATS) guidelines, spinal TB in adults should be treated for 6–9 months.[26] The National Institute for Health and Care Excellence (NICE) guidelines recommends a daily six-month regimen, with the first 2 months consisting of four drugs (INH, rifampicin, pyrazinamide, ethambutol/streptomycin).[27] INH and rifampicin are to be continued for the remaining duration, with provision to modify the regimen as per drug sensitivity. However, several experts have recommended a longer duration of therapy, guided by radiological or pathological clearance of the disease. In a randomized trial in India that compared 6-month versus 12-month therapy for biopsy-proven spinal TB, similar clinical outcomes were achieved at 24 months of study.[4] In this study, 100 patients with spinal TB were randomized to either six or 12 months of ATT. All patients were followed up for at least 24 months. One patient crossed over from the 6 months to the 12-month arm. All patients had a biopsy-proven diagnosis. The primary endpoint was clinical cure with the absence of recurrence at 24 months of completion of therapy. No recurrence of disease occurred in either arm at 24-month follow-up. The presence of biopsy-proven diagnosis strengthened the study. However, it had an open-label design. Additionally, more patients in the 12-month treatment arm required surgery at presentation, despite randomization, skewing the study in favor of the 6-month arm.

The Index-TB guidelines for the treatment of extra-pulmonary TB in India state that bone and joint TB should be treated with extended courses of ATT with a 2-month intensive phase consisting of four drugs (isoniazid, rifampicin, pyrazinamide, and ethambutol), followed by a continuation phase lasting 10–16 months, depending on the site of disease and the patient's clinical course.[28] The recommended regimen as per this guideline is initial 2 months of INH, rifampicin, pyrazinamide, and ethambutol, followed by 10 months of INH, rifampicin, and ethambutol. We feel that since the disease is potentially disastrous in its complications, stopping ATT at blanket 6 months may not be feasible at this time but should be guided by multiple factors, including clinical response as well as neuroimaging and no single factor should be used to determine the end-point of therapy.

Wang *et al*.[29] explored the feasibility of ultra-short course ATT in patients with spinal TB. They included 185 patients with spinal TB requiring surgical management. Patients with ultra-short course chemotherapy (average duration of 4.5 months) were compared with standard chemotherapy (average duration of 9 months). Both groups underwent surgery and were followed up for 61–87 months. The efficacy of ultra-short course ATT was found to be similar to the standard regimen in terms of improvement in inflammatory markers, kyphosis, recovery for work and activities of daily living, as well as post-op bone healing. Guo *et al*. from China, reported 46 patients requiring retreatment who underwent extensive surgery to debride the tubercular load, leading to successful outcomes with ATT of 9–12-month duration.[30]

In a study from Delhi that assessed practice trends in the treatment of central nervous system TB, ATT regimens were often guided by individual physician experience as well as neuroimaging rather than strictly guideline-based regimens.[31]

Role of Imaging in Spinal TB

Imaging is of immense diagnostic value in spinal TB. Plain X-rays of the spine offer an overview. Computed tomography (CT) scan details skeletal involvement and magnetic resonance imaging (MRI) provides soft tissue and spinal cord involvement.

Plain radiography

Spine X-rays continue to be a screening tool although they may be normal in the initial stages of the disease. Initial X-rays may reflect changes in 70–99% of patients.[32] These findings include loss/blurring of plate margins as well as radiolucency. This is followed by features of vertebral destruction, with loss of anterior vertebral height, endplate erosion, the formation of vertebral geodes, soft tissue masses, and bony sclerosis. The occurrence of calcification within the paravertebral soft tissue highly favors TB.[33] Vertebral height may remain preserved till advanced stages of the disease. Spread to adjoining vertebral segments gives rise to multilevel disease. X-rays also reflect late findings including bony ankylosis, sclerosis, and vertebral body collapse. Certain nonclassical findings may also be observed and include anterior vertebral scalloping, noncontiguous vertebral involvement, craniovertebral junction involvement, and reactive sclerosis leading to the development of "ivory" vertebrae.[34]

Computed tomography

CT scans provide better radiographic detail compared to X-rays. Vertebral destruction of four types may be delineated. These include fragmentary, osteolytic, subperiosteal, and localized.[35] The most common of these is the fragmentary type in which bony splinters migrate into the soft tissue mass and is highly characteristic of TB.[35] In addition, the administration of contrast agents permits enhancement of tuberculous tissue and abscess wall, better defining the pathology. Paraspinal soft tissue mass and abscess are observed early in the course of the disease, occurring in 45–100% of cases.[35] CT is superior to MRI in the detection of calcification.

Magnetic resonance imaging

This is the imaging modality of choice in spinal TB. MRI enables early detection of signal change, as well as delineation of the extent of involvement, including myelopathy. Based on MRI, there are four patterns of involvement in spinal TB: anterior, posterior, central, and paradiscal.[36] MRI demonstrates the involvement of the vertebral body, disc, paraspinal soft tissue, and abscess formation. It may also demonstrate hitherto unsuspected multilevel vertebral involvement as well as skip lesions. The choice of surgical approach, whether anterior or posterior, is made based on MRI findings as it enables disease localization in various planes. Vertebral bodies demonstrate hypointense signal on T1 weighted image and hyperintense signal on T2 and short tau inversion recovery sequence [Figure 1 to 4] Abscesses appear hyperintense of T1 sequence and hypointense on T2. Contrast enhancement shows thin wall enhancement. A thoracic abscess can track into the iliopsoas muscles, thigh, and retroperitoneum. Despite typical radiological features, there are none which are sine qua non, and tissue diagnosis may be necessary. However, certain features do strongly support the diagnosis of TB: a paraspinal collection with/without thin-walled abscess, subligamentous extension beyond two vertebrae, multilevel vertebral involvement, dorsal vertebral lesions, and T2 hyperintense signal change.[37]

Spinal TB in Special Situations

Spinal TB in HIV-infected persons: A double conundrum

TB is the most common opportunistic infection in HIV patients with 37 times higher risk.[38] The principles of management, duration of treatment as well as the outcome of spinal TB in HIV patients are the same as for immunocompetent patients.[39]

Issues that especially concern this subgroup are risk of drug interactions and potential for immune reconstitution. Most protease inhibitors and non-nucleoside reverse transcriptase inhibitors used in antiretroviral therapy regimens interact with rifampicin. In immune reconstitution syndrome, initiation of antiretroviral therapy in a patient being treated for TB leads to improvement in the inflammatory response and paradoxical worsening of TB features. Similarly, initiation of antiretroviral therapy in an ATT naive patient may also unmask latent TB.

Spinal TB in Pregnancy

Recognition of spinal TB in pregnancy may be delayed with lower back pain being mistaken for pregnancy-related back pain. Antitubercular drugs pose a little hazard in terms of risk of congenital anomalies. In advanced pregnancy, early decompression and instrumented fusion may support favorable outcomes in spinal TB with paraplegia.[40]

Role of Steroids

There is no definitive role of steroids in the treatment of spinal TB except in cases associated with arachnoiditis or paraplegia to non-osseous spinal TB.[41]

Role of Surgery

The role of surgery in spinal TB has been a matter of lasting debate. The indications for surgery in spinal TB declined with the advent of effective chemotherapy. A Cochrane review of trials in 2006 identified two trials with a total of 331 patients and concluded that evidence was insufficient to recommend routine surgery in addition to medical therapy in patients with spinal TB.[42] The Medical Research Council compared patients with spinal TB to chemotherapy alone versus debridement versus radical debridement with fusion.[43] All three groups had similar functional outcomes.

Indications for surgery in a patient with spinal TB[44] who has associated neurological deficits include: worsening of existent deficits or development of new deficits while on therapy for 3–4 weeks, spinal tumor syndrome, rapidly developing

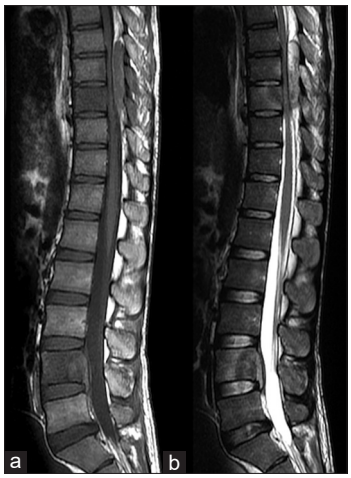

Figure 1: MRI of the spine showing tuberculous involvement. (a) T1-weighted (b) T2-weighted image shows T11 vertebral body involvement with the posteriorly placed epidural collection

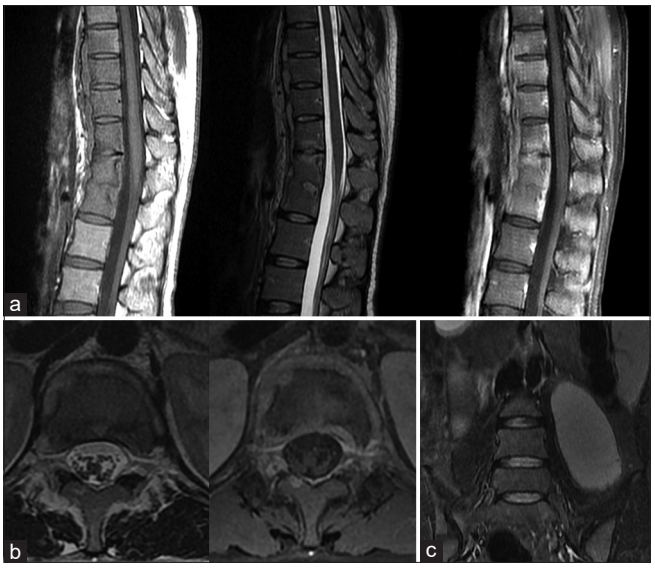

Figure 2: MRI of the spine showing tuberculous involvement. (a) Sagittal, (b) axial, (c) coronal sections showing vertebral body destruction with endplate involvement and slight gibbus formation

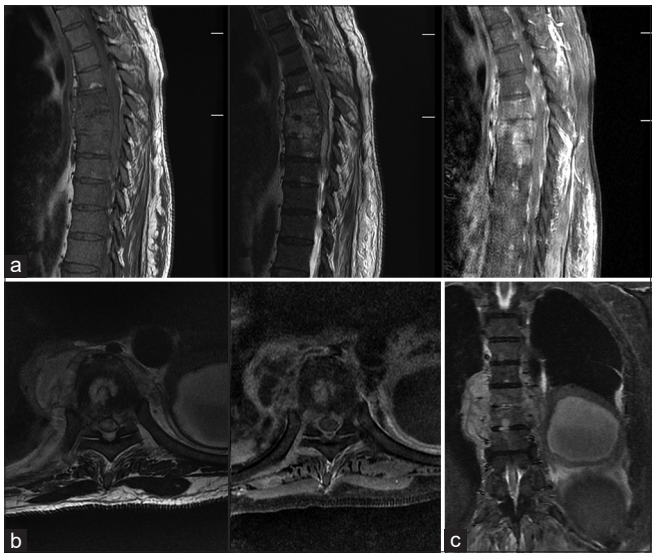

Figure 3: MRI of the spine showing tuberculous involvement. (a) Sagittal, (b) axial, (c) coronal sections showing vertebral body destruction with endplate involvement and slight gibbus formation

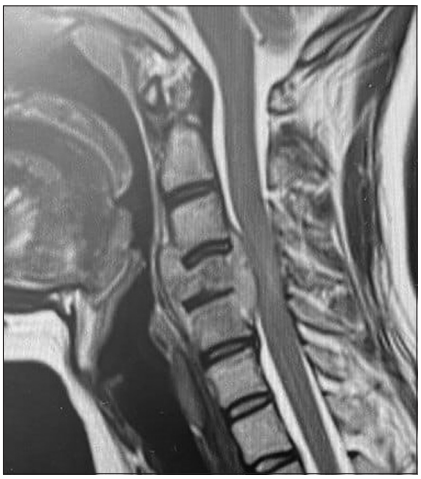

Figure 4: MRI of the spine showing tuberculous involvement. Saggital section showing C4 vertebral involvement with retropharyngeal and epidural collections

paraplegia, severe paraplegia, defined in the INDEX-TB guidelines as 'flaccid paraplegia, paraplegia in flexion, complete sensory or motor loss for greater than 6 months, presence of painful paraplegia in elderly patients, neural arch disease. This is similar to Tuli's "middle path" approach which balanced medical and surgical management and came about in the 1970–80s. Surgery is also necessary to prevent severe kyphosis. The degree of final kyphosis can be estimated with the help of the following formula: Y = a + bx . a and b are consonants 5.5 and 30.5, and x is the loss of vertebral body height. Y represents the final angle of kyphosis. Kyphosis exceeding 60 degrees is associated with repeated cord injury and late neurological deficits and must be prevented.[45]

In the absence of neurological deficit, surgery is indicated in the following conditions: diagnostic uncertainty, mechanical instability, the disease involves both the body and the posterior complex or bilateral facet joint involvement, suspicion of drug resistance and spinal deformity (severe kyphosis or kyphosis in children which may worsen with growth).

Indications for instrumented stabilization include pan-vertebral involvement, lumbar and cervical spine, kyphosis correction surgery is planned, junctional area lesion and in the dorsal spine, if a long graft >4–5 cm is necessary to bridge the gap following surgical stabilization. Some of these indications are summarized in Table 2.[46]

The surgical approach may be determined using various classification schemes. The GATA classification is based on radiological findings to determine the surgical approach which may range from biopsy to decompression (if neurological

compromise exists).[47] However, the severity and progression of neurological paucity are not considered in this system. Bhojraj and Mehta proposed a more pragmatic approach, based on clinical features and involvement of posterior vertebral elements.[48] Anterior and posterior approaches may be used and have similar results. The posterior approach is preferred in case of deformity.

Anterior approach

Since spinal TB predominantly affects the anterior column, anterior approach permits adequate exposure and debridement.[46] The anterior approach is employed in T4–T10 involvement. This is because, above T4, exposure is suboptimal and limited by the great vessels. The anterior approach is recommended when the posterior elements are unperturbed and should not be performed in panvertebral disease. The anterior transthoracic approach has higher morbidity than the posterior approach and may lead to pulmonary and pleural injury.

Posterior approach

Due to the morbidity associated with the anterior approach, the posterior approach has been described for patients with a significant deformity in whom the anterior approach may not suffice.[46] This approach provides greater stability as the disease process is anterior. Additionally, it can be used in patients who have respiratory compromise, elderly or multiple comorbidities.

Table 2: Indications for surgery in spinal TB[46]

In the presence of deformity:
Angle of kyphosis >60 degree
Angle of kyphosis 30-60 degree with frank neurological deficits
Worsening of deformity while on therapy
In the presence of abscess:
Large abscess causing local symptoms
Worsening neurological deficits on therapy
Rapid onset of paraplegia
Severe degree of paraplegia/spinal tumor syndrome
In the presence of instability:
Presence of spine at risk in children
Biopsy/aspiration (open):
Doubtful diagnosis
Lack of improvement after 6-8 weeks of antitubercular therapy
Suspicion of drug-resistant TB
Recalcitrant pain

Combined approach

Posterior instrumentation is combined with anterior decompression and fusion performed in one or two stages. Single-stage procedure is associated with higher morbidity. During the staged procedure, posterior fixation followed by anterior fusion or vice versa may be performed. Initial anterior fusion is associated with the risk of graft slippage while posterior fixation is pending. Posterior instrumentation in addition to anterior debridement/graft placement has also been advocated.

A Great Masquerade: Under and OverDiagnosis

Owing to the largely clinic-radiological nature of spinal TB diagnostics, there is an inherent risk of both over and underdiagnosis. Underdiagnosis, which is clinically less common, may occur in conditions like pregnancy, or in the elderly where low backache is often attributed to mechanical factors. Overdiagnosis is relevant in our scenario due to the widespread epidemiology of TB *per se*. In a recent study, nearly 25% of patients with the alternative diagnoses were radiologically reported as TB or TB formed a differential diagnosis.[49] The most common alternative diagnoses in this series were pyogenic spondylitis, *Brucella* spondylodiscitis, rheumatoid arthritis, etc., Other significant misdiagnosed entities were metastases and lymphoma. This highlights the notion that obtaining a microbiological or pathological diagnosis may be vital, especially if the radiology is not highly typical for TB, rather than empirical therapy. We summarize some of the features that may help in this distinction in Table 3.

Prognosis

Prognosis is considered to be good in individuals who do not develop complications. With medical therapy alone, patients experience relief in pain and even deficits as well as deformity. In a study from Pakistan involving 47 patients of spinal TB treated with ATT for 12 months, 93.6% of patients had complete recovery including neurological deficits with ATT. 19.1% required surgical input. 85% of these patients had a motor

Table 3: Differentiating features between spinal tuberculosis (TB) and its common mimics

Feature	Spinal TB	Pyogenic spondylitis	Metastatic spine disease	Brucella spine involvement
Disease location	Lumbar and dorsal	Lumbar	Dorsal	Lumbar
Predilection	Involvement of vertebral disc and bodies	Involvement of vertebral disc and bodies	Posterior body wall, pedicles, lamina	Involvement of vertebral disc and bodies
	Soft tissue involvement prominent	Soft tissue involvement minimal		Soft tissue involvement minimal; sacroiliitis present
Risk factors	Exposure to tuberculosis	Underlying diabetes, etc. predisposing to infection	Known systemic malignancy	Exposure to unpasteurized milk
Radiological features	Destruction of vertebral body and disc with extensive soft tissue involvement with rim enhancement	Destruction of vertebral body and disc, epidural abscess, prominent contrast enhancement	Lesions have T1 hypo- and T2-hyperintense signal, heterogeneous enhancement	Vertebral architecture preserved despite extensive vertebral osteomyelitis

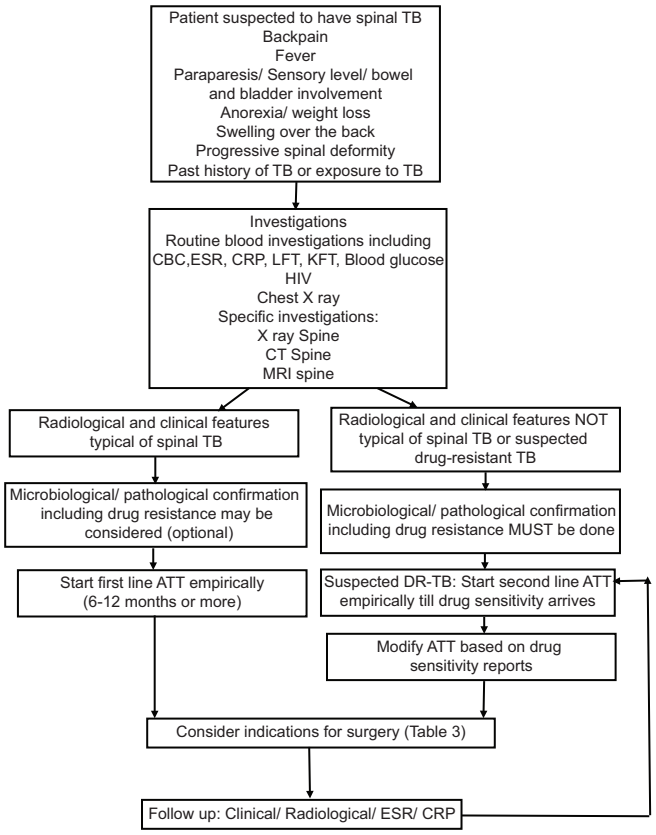

Figure 5: Algorithmic approach to a patient with spinal tuberculosis

weakness at presentation and 12% had sphincter involvement.[50] In another study from South Africa, 82 patients with spinal TB were followed up.[51] Of these, 52% were in non-ambulatory state at presentation and 21% had mild neurologic deficits. Among the patients with neurological deficits, 92% had significant recovery and 74% became ambulatory from an initially non-ambulatory state. In another study, out of 79 patients, 61% had a severe neurological impairment.[52] All were managed using the anti-tuberculous treatment. Almost one-third of patients required operative treatment as well. 70% showed significant improvement within 6 months of treatment. In a Korean study, of 116 patients with spinal TB, 35% had significant symptoms. 62% required surgical management.[53] After ATT, 94 patients showed favorable outcomes. Age and surgery correlated with a favorable outcome.

Conclusion

Spinal TB offers diagnostic as well as management challenges due to the difficulties in establishing a microbiological or pathological diagnosis. The possibility of TB mimics must be carefully considered in all cases and efforts made to rule out these possibilities. The assessment of response to therapy is another challenge. Hence, even the diagnosis of drug-resistant spinal TB may be presumptive. It would be best to let the patient's clinical picture dominate the cut-off point of therapy rather than any arbitrary guideline. We have summarized our approach in Figure 5.

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Conflicts of interest

There are no conflicts of interest.

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