Chapter 7
Tuberculous Spondylodiscitis

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7.1 Introduction and Epidemiology

According to the global tuberculosis (TB) report 2016 of the World Health Organization (WHO) (http://www.who.int/tb/publications/global_report/gtbr2016_executive_summary.pdf?ua=1), the number of the estimated new TB cases worldwide was 10.4 million (56% of them were men), and the estimated number of deaths due to TB was 1.4 million in 2015. TB still remained among the top ten causes of death worldwide in the same year. "Tuberculous spondylodiscitis (TS)"/"spinal tuberculosis (ST)" and "Pott's disease" are synonyms which refer to the infection of the vertebral bones by *Mycobacterium tuberculosis*. Nontuberculous mycobacteria (NTM) rarely cause vertebral osteomyelitis [1, 2]. Before the description of tubercle bacillus by Robert Koch in 1882, Pott's disease was first defined by Sir Percivall Pott in 1779 based on the clinical presentation of a patient with spinal deformity and paraplegia [3]. Musculoskeletal TB accounts for approximately 10% of extrapulmonary TB cases and 1–5% of all TB cases [1, 4–6]. ST is the most common form of skeletal TB (accounts for about half of the cases) followed by tuberculous arthritis and extraspinal tuberculous osteomyelitis [7–10]. Male population is slightly more at risk of developing ST. More than half of the patients are men [11]. Mean age of the patients is 40–50 years (range: 8–60 years). In endemic areas, it occurs in younger subjects, whereas in non-endemic regions, it occurs mostly in adults [11, 12]. ST is endemic in most of the developing countries. During the 10-year period (from 2002 to 2011), the incidence of ST has significantly decreased in the

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USA. However, although it is not common, it still remains a public health issue [13]. In recent decades, its incidence – in context with the total TB incidence – in developed countries has been on the rise due to HIV-infected patients, immigrants from TB-endemic countries [11]. The incidence of ST among HIV-infected patients is comparable to that in HIV-uninfected patients [1, 12, 14]. The history of ST goes back to Egyptian mummies with lesions in skeletal bones. *Mycobacterium tuberculosis* complex DNA was detected in specimens from bone lesions of mummies [15–17].

It is a serious public health problem because the diagnosis of this destructive form of TB is usually delayed due to its indolent course. Delayed diagnosis and treatment result in permanent sequelae such as deformities and neurological deficits [1]. It is still an ongoing cause of mortality and morbidity in the twenty-first century despite advances in diagnostic tools and treatment [18].

7.2 Pathogenesis and Pathophysiology

Hematogenous seeding to bones occurs during the course of primary infection. Local adaptive immune responses play a role in the confinement of primary infectious foci. Contiguous spread from a primary focus or development of infection via lymphatic drainage is very rare. Predisposing conditions such as immunosuppression, advanced age, HIV infection, malnutrition, or chronic renal failure may lead to reactivation of latent infection in those foci in vertebral bones [19, 20]. Both osteomyelitis and arthritis occur in the context of ST. The initial site of infection by tubercle bacilli is the growth plate, followed by the inflammation of intervertebral joint, and then the infection spreads to involve the two adjacent vertebral bodies [21]. Invasion of end arteries leads to bone destruction. In case of contiguous involvement of the intervertebral disc as in so-called spondylodiscitis, vertebral collapse may take place. Sometimes, the avascular intervertebral disc is spared, in which case the term “spondylitis” is preferred. Cold abscesses around vertebral structures may develop. Drainage of the infectious focus into psoas muscle causes myositis and then psoas abscess may develop. Interferon-gamma, CD4+, and CD8+ T lymphocytes are the vital elements of the cellular immune response to TB [22]. Other than lymphocytes, epitheloid histiocytes, giant cells, plasma cells, and fibroblasts can be observed in histopathological specimens of the infected focus. Either exudative caseous necrosis destroys the bone tissue or granulation reaction occurs. Healing process usually happens with fibrous tissue formation and calcification. In contrast to pyogenic osteomyelitis, periosteal reaction or bone regeneration with sclerosis does not take place in ST [23]. Mostly, the anterior portion of the vertebral body is involved [24]. Collapse of the anterior parts of the vertebral bodies leads to “Gibbus deformity” (kyphosis associated with Pott’s disease). The thoracic and lumbar vertebrae are most commonly involved in ST [25, 26].

7.3 Clinical Manifestations

The clinical presentation is insidious and the early phase of the disease is indolent [1]. In a multinational, multicenter study including 314 patients with ST, the median duration from the onset of symptoms to diagnosis has been reported to be nearly 2.5 months [27]. In another study from Europe, the median duration of symptoms before diagnosis was 4 months [28]. The most common symptoms are back pain or pain in the involved area [12]. The severity of pain increases over time. Muscle spasm around the involved vertebrae may occur. Fever, weight loss, and night sweats are uncommon constitutional symptoms present in less than half of the patients, more commonly in advanced disease [29]. But, in the largest case series of 967 patients with ST reported from China, fever and night sweats were common presenting symptoms after back pain [26]. On physical examination, local tenderness over the spinous processes, severe pain induced by range of motion test, and in advanced cases kyphosis and neurologic symptoms such as numbness, tingling, weakness, and even paraplegia may be present. In the largest case series of 967 patients with ST from China, neurological involvement was present in 1/3 of the patients [26]. In another study conducted in France, half of the patients had neurologic symptoms and signs [28]. Wang H et al. have reported the frequencies of sensory and motor deficits as 54% and 28%, respectively, in their study including 329 patients with ST [30]. Neurologic deficit is usually reversible if early diagnosis and prompt treatment with urgent decompression can be performed at presentation [31]. Typical presentation of the disease includes back pain, gibbus deformity, paraplegia, or paraparesis. Atypical presentation involves epidural abscess without significant anterior vertebral involvement, noncontiguous multiple segments of the vertebral column, bilateral psoas abscesses, involvement of posterior segments of the vertebral column only, and sacral ST [32–34].

Thoracic vertebrae are the most commonly involved site, followed by lumbar and cervical spinal vertebrae in decreasing frequency [12]. In a large case series from Pakistan, the frequencies of the involved vertebral levels have been reported as dorsal spine (45%), followed by lumbosacral spine (33%), cervical spine (10%), and multiple levels (12%) [11]. Sharma A et al. have evaluated the clinical characteristics of 312 patients with ST. In their study, thoracic vertebrae were most commonly (46%) involved followed by thoracolumbar vertebrae (28%). In 80% of the patients, only one or two adjoining vertebrae were affected [35]. In immunocompromised patients (such as HIV-infected subjects) multiple vertebral lesions may be seen. NTM infection usually presents with widely spread lesions in the spine in older and/or immunosuppressed patients [36]. The most common NTM isolated as the cause of ST was *M. avium complex* (MAC) followed by *M. xenopi*, regardless of HIV infection. In HIV-infected patients, ST caused by NTM occurred at a younger age. Half of the patients with ST due to NTM had any form of immunosuppression, while 15% had a history of surgery or trauma. Surgery was indicated in 2/3 of those patients [37].

Table 7.1 Symptoms and signs of ST [1, 3, 27, 28]

	Frequency
Chronic back pain	58–87%
Local spinal tenderness	21%
Fever	31–48%
Weight loss	41–48%
Night sweats	18–49%
Cold abscesses Paraspinal Psoas	69% 59–63% 22–29%
Kyphosis/gibbus deformity	46%
Neurologic deficit	40–56%
Weakness of the lower extremities	69%
Paraplegia	10–25%
Spinal instability	21–33%

Neurologic symptoms develop early in the course of cervical ST [38]. Retropharyngeal abscess may the presenting sign of cervical ST [2, 39]. Tuberculin skin test (TST) was positive in ¾ of the patients with ST [27]. QuantiFERON(®)-TB Gold In-Tube test, which is an interferon-gamma releases assay (IGRA), was positive in 75% of patients [40]. Concomitant pulmonary TB has been reported in 3–14% of the cases [26, 27, 31]. Therefore, chest radiography is not useful for the diagnosis of ST. But it should always be obtained to rule out pulmonary tuberculosis which requires isolation of the patient with acido-resistant bacilli (ARB)-positive sputum production. Also, pulmonary involvement may facilitate the diagnosis of ST.

Because of delayed diagnosis, vertebral body collapse can lead to kyphosis or "Gibbus deformity" which is a form of structural kyphosis that may cause spinal cord compression. The cause of paraplegia is spinal cord compression either by gibbus deformity or osteophytes. Spinal cord compression may be present at the time of diagnosis in 40–70% of cases [41]. The most common complications of ST are paraplegia and quadriplegia depending on the nerve roots affected at the involved spinal level and spinal deformity (kyphosis or scoliosis). Compression fractures may also occur in untreated cases. Gibbus deformity causing spinal subluxation may cause such neurologic deficits due to spinal cord compression. If there is any motor deficit at the time of presentation, it is unlikely to recover completely despite therapy.

Paravertebral "cold abscess" (soft tissue mass) develops in most of the cases. Calcification of paravertebral abscesses may occur. Psoas muscles may be involved by spread of the infection [7]. *M. tuberculosis* is a quite common cause of psoas abscess in TB-endemic countries. In comparison to brucellar and pyogenic vertebral osteomyelitis, ST more commonly causes neurologic deficit, spinal deformity, and paravertebral abscesses. Also, thoracic vertebrae are more commonly involved in ST [42]. Involvement of noncontiguous multisegmental vertebrae without intervertebral discs is an atypical form of ST which is quite rare (3–16%) [11, 26, 27, 43, 44]. It may resemble malignant diseases [32, 44]. Symptoms and signs of ST and their frequency in patients diagnosed with ST are listed in Table 7.1.

7.4 Diagnosis

Because of the indolent presentation, the diagnosis is usually delayed. Prompt diagnosis is required to start appropriate therapy on time to prevent permanent sequelae development. Therefore, a high index of suspicion of ST based on epidemiological, past medical and/or contact history, clinical clues, and characteristic imaging findings is of paramount importance in earlier diagnosis [1, 12]. Especially in HIV-infected patients, no other symptoms or signs may be present. In TB-endemic countries, skeletal pain may lead to consideration of ST at initial presentation. However, in developed countries with low TB incidence, the diagnosis may be overlooked and delayed.

Imaging modalities [plain radiography, computed tomography (CT), and magnetic resonance imaging (MRI)] are useful tools to consider ST. Early in the course of the disease, radiography is not sensitive in diagnosis. Pathologic findings on radiography firstly occur in the anterior aspect of a vertebral body with reactive sclerosis, the vertebral end plate becomes demineralized [45]. Subsequent involvement of the adjacent vertebra leads to anterior wedging. Calcifications in abscesses can also be demonstrated on radiography [46]. But, radiography is not helpful early in the course of ST for diagnosis [47]. Bone sclerosis and destruction, lytic lesions and collapse of the intervertebral disc, destruction of the adjacent vertebral corpus, epidural extension (present in more than 60% of the patients), and calcifications of abscesses can be visualized on CT [48, 49]. CT can also be used as a guidance for FNAB and percutaneous drainage. Diagnostic yields of CT-guided bone biopsy for ST are 60–80% and were comparable to surgical biopsy [28, 50]. Specificities of CT-guided biopsy for pathogen detection were 83% and 91% in epidural infiltration and paravertebral abscesses, respectively [51].

MRI is the most sensitive imaging method in the diagnosis of ST. Destruction of the anterior portion of the vertebral body, anterior wedging, and paravertebral cold abscesses seen on MRI favor the diagnosis of ST [11, 46]. Contrast-enhanced MRI also reveals compression of the nerve roots or the spinal cord [52, 53]. MRI is superior to other imaging modalities in diagnosis of ST because of its high-contrast soft tissue resolution, imaging in multiple planes, and high sensitivity to detect early infiltration in bone marrow [11]. On T1-weighted images, hypointense vertebral bone marrow, reduced disc height, paraspinal soft tissue masses, and epidural extension of the inflammation can be demonstrated. On T2-weighted images, involved vertebral bodies, intervertebral discs, and soft tissues are seen as isointense or hyperintense areas [11]. Majority (85%) of the patients have typical MRI findings at presentation [35]. Epidural/paraspinal abscesses on MRI usually favor tuberculous spondylitis rather than pyogenic spondylitis [54, 55]. Radiographic features of ST are summarized in Table 7.2. MRI images of three different patients diagnosed with Pott's disease are shown in Figs. 7.1a–c, 7.2a, b, and 7.3a, b.

The diagnosis is established by microbiological and/or histopathological examination of the involved tissues and affected parts of the skeleton obtained by CT or ultrasound (US)-guided fine-needle aspiration and biopsy (FNAB) [52]. However, FNAB was inadequate for diagnosis in 27% of cases [56]. Microbiological

Table 7.2 Radiographic features of tuberculous spondylitis

Radiographic features
Involvement of adjacent vertebral bodies
Involvement of multiple vertebral levels
Demineralization of vertebral end plate
Lytic destruction of anterior portion of vertebral body
Sparing of the intervertebral disc Disc space narrowing due to disc destruction
Anterior wedging of vertebrae
Vertebral body collapse
Subligamentous spread of infection Paravertebral abscess
Psoas muscle involvement or abscess Heterogenous signal intensity and rim enhancement on MRI

Adapted from reference [42] and [46]

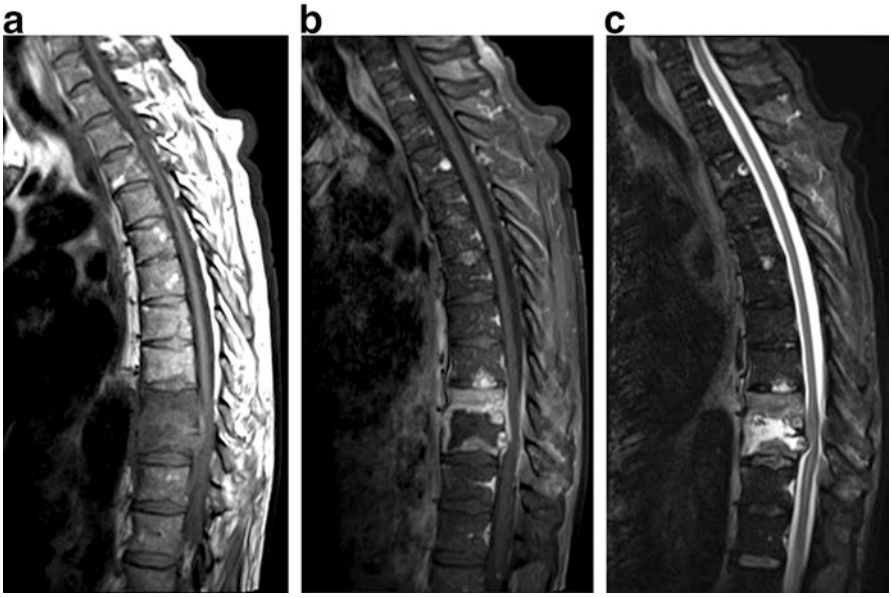

Fig. 7.1 (**a**) T1W sagittal image: Bony destruction at thoracic T10 and T11 vertebrae due to Pott's disease, paravertebral extension of soft tissue component composed of granulation tissue, an abscess is seen between T10-T11 vertebral bodies with intraosseous component, destructing end plates. There is also spinal canal narrowing and significant cord compression due to bulging of posterior margin of destructed T11 body. (**b**) T1W sagittal image (post-contrast): Contrast enhancement of destructive lesions of Pott's disease is seen at thoracic T10 and T11 vertebrae. (**c**) T2W sagittal image: Typical bony destruction at thoracic T10 and T11 vertebrae, paravertebral extension of soft tissue component, an abscess is seen between T10-T11 vertebral bodies, destructing end plates. There is also spinal canal narrowing and cord compression due to bulging of posterior margin of destructed T11 body (compressive myelopathy)

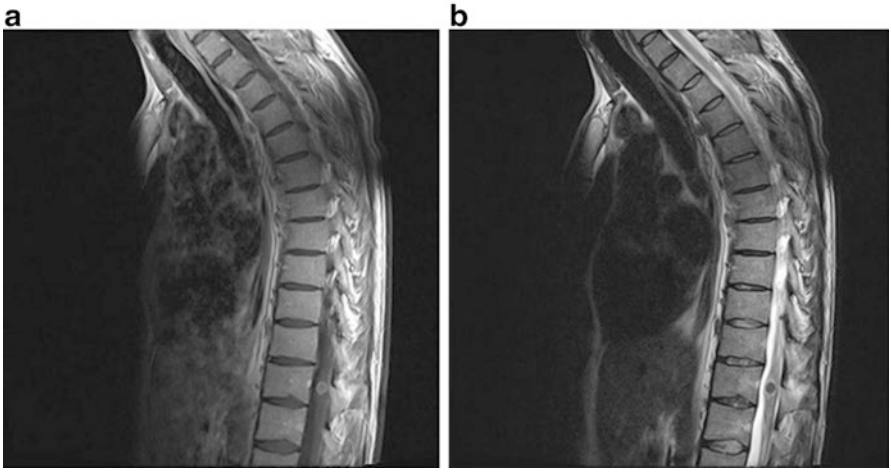

Fig. 7.2 (**a**) T1W sagittal image (post-contrast)**.** Height of the vertebral bodies decreased in multiple levels and intervertebral joint spaces narrowed due to Pott's disease. A spinal tuberculoma can be seen in the distal spinal cord at the level of thoracic T11 vertebra. It shows contrast enhancement peripherally in post-contrast series image. (**b**) T2W sagittal image: The spinal tuberculoma at the level of thoracic T11 vertebra is seen as hypointense centrally and hyperintense peripherally

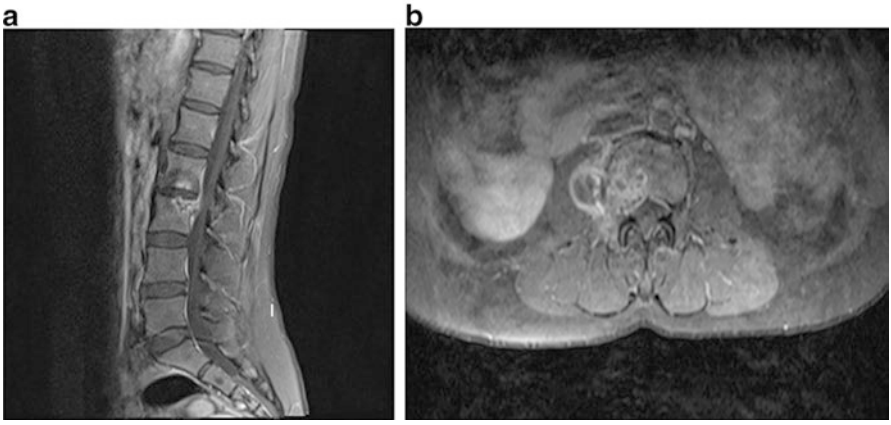

Fig. 7.3 (**a**) T1W sagittal image: Involvement of end plates and bodies of lumbar L2 and L3 vertebrae and L2 and L3 intervertebral disc due to Pott's disease. (**b**) T1W axial post-contrast image: A psoas abscess with contrast enhancement peripherally which developed secondary to Pott's disease of lumbar vertebrae at the right paravertebral area can be seen

examination includes microscopy of acid-fast bacillus (AFB) stained specimen and mycobacterial culture. Microbiologic diagnosis can be established in ¾ of the patients if appropriate specimens can be obtained [12]. If the mycobacteria can be isolated in TB culture, drug susceptibility testing is essential to prescribe optimal therapeutic regimen [57–59]. Culture of the material from draining sinuses may show colonizing microorganisms. Deep bone or soft tissue material is necessary for the correct diagnosis of causative pathogen. If CT- or US-guided FNAB reveals caseating granulomas and AFB, the diagnosis of ST is confirmed. Other granulomatous diseases such as brucellosis, fungal infection, infection with nontuberculous mycobacteria should be considered in the differential diagnosis of ST. Although currently not FDA-approved for use in extrapulmonary TB, molecular diagnostic methods such as nucleic acid amplification can be used to improve the diagnostic probability. They have promising results in diagnosis of musculoskeletal TB, but they lack sensitivity despite having high specificity [59–61]. Furthermore, in TB highly endemic countries, availability of the rapid automated growth systems and molecular tests such as Xpert MTB/RIF assay to detect nucleic acids and rifampin resistance may be limited [60]. The sensitivity and specificity of Xpert MTB/Rif assay are 62% and 100%, respectively [62].

In resource-limited countries with high TB endemicity, the diagnosis of ST is usually based on epidemiological, clinical, and radiological features. Erythrocyte sedimentation rate (ESR) is usually elevated in more than 80% of the patients as in other forms of osteomyelitis [63].

7.5 Differential Diagnosis

The differential diagnosis of ST includes other subacute or chronic granulomatous and non-granulomatous infections of the vertebrae caused by *Brucella* spp., *Candida* spp., other endemic fungi, *Actinomyces* spp., *Burkholderia pseudomallei* (melioidosis), and some bacteria such as *Staphylococcus aureus* [64]. Epidemiological features should be considered in the differential diagnosis of infectious etiology. In comparison to brucellar spondylodiscitis, ST presents with suppurative abscess formation requiring surgical drainage and spinal complications more commonly [65]. Noninfectious diseases such as spondyloarthropathy, degenerative processes, osteoporotic collapse of vertebral bodies, traumatic fractures, and especially primary or metastatic malignancy may mimic ST [7, 66].

7.6 Treatment

The main objectives of treatment are immediate relief of the symptoms (pain, paraparesis, and paraplegia), restoration of neurological and motor function, prevention of development of permanent long-term sequelae, and eradication of the infection.

Early diagnosis and prompt treatment with appropriate anti-TB drug regimen can prevent the development of sequelae such as neurologic deficit and spinal deformity. The principles of pulmonary TB treatment either for drug-susceptible or drug-resistant Mycobacteria are also valid for extrapulmonary TB forms [67]. Modern management strategies of ST have been defined by the British Medical Research Council group which organized randomized trials in patients with ST [68]. A large number of patients were enrolled in those trials, but patients with cervical ST were not included because of its low incidence. Treatment recommendations for patients with cervical ST are mostly based on case series studies [69–71]. Rifampin-based anti-TB treatment for longer courses plus anterior approach surgery when indicated resulted in full recovery in most of the patients with cervical ST. A laminectomy is not as effective in relieving cervical spinal cord compression. Moreover, because of the risk of instability of the cervical spine, it is not recommended [69]. Also, patients with myelopathy were excluded. However, in another study, medical treatment was sufficient for functional or complete resolution of myelopathy [72].

Antituberculous Therapy Medical therapy with anti-TB drugs only is sufficient in most of the patients without any neurological deficit. Standard anti-TB drug combination of isoniazid, rifampin, and pyrazinamide, with or without ethambutol given for 6, 9, or 12 months plus surgery when indicated, constitutes the mainstay of treatment of ST. Selection of the antituberculous (anti-TB) drug regimen differs whether or not the Mycobacteria are drug-resistant and whether the patient is HIV-infected or not. The optimum duration of antimicrobial therapy is uncertain and depends on the susceptibility of isolated Mycobacteria and the composition of the anti-TB drug regimen. Longer treatment duration (12–18 months) has been recommended previously for ST. However, it has been shown that 6–9 months of therapy with rifampin-based first-line anti-TB drugs (i.e., isoniazid plus rifampin for 6 months and streptomycin for the first 3 months) combined with surgical excision and bone grafting is sufficient in patients with susceptible mycobacteria and good response to therapy [7, 73, 74]. Six-month, 9-month, and 18-month regimens after radical surgical debridement gave out similar results. No recurrence or reactivation of TB was observed in patients receiving any of the three regimens [73]. In selected patients who underwent surgical intervention including thorough debridement, bone grafting, and internal fixation, even an ultra-short-course therapy of 4.5 months was as successful as a 9-month therapy [75]. In contrast, a retrospective study reported a high rate (62%) of relapse with a 6-month of therapy, while no relapse was observed with 9 month course of treatment [74]. Nine to 12 months of therapy are necessary in advanced cases with poor therapeutic response, multidrug-resistant tuberculosis (MDR-TB) [67, 76]. Osteoarticular multidrug-resistant tuberculosis (MDR-TB) has rarely been reported in the medical literature. A favorable clinical outcome can be achieved with second-line antituberculous drugs and surgery when indicated in those cases [77].

Immobilization by bed rest and/or body casts/orthosis is recommended for patients with ST of thoracolumbar junction and more than 50% loss in vertebral height and those who have severe pain. Konstam and Blesovsky reported an ambulatory treatment without immobilization or bracing and medical therapy with at

least 12 months course of isoniazid and p-aminosalicylic acid (PAS) for ST [78]. Surgery was performed in only a small percentage of patients who needed abscess drainage. Eighty-six percent of patients recovered completely with chemotherapy alone.

CDC, Infectious Diseases Society of America (IDSA), and American Thoracic Society (ATS) guidelines for treatment of spinal TB recommend medical rather than surgical treatment in uncomplicated cases [67], because surgical debridement in combination with medical treatment did not provide any additional benefit compared to medical treatment alone in uncomplicated cases [72, 79]. In a systematic review and meta-analysis, medical management of spinal epidural abscess failed in nearly 30% of the cases and required surgery [80].

Surgical Therapy There is heterogeneity in the percentage of patients who required surgery [12]. In approximately 2/3–3/4 of the patients, surgery may be necessary [26, 81]. Medical treatment alone was implemented in 1/3 of the patients, while diagnostic and/or therapeutic surgical intervention was required in the remaining 2/3 [27].

The indications for surgery include kyphosis >40 degrees at presentation (kyphotic angle is measured from lateral spinal X-ray using the modified Konstam method) or progressive kyphosis, neurologic deficits due to spinal cord compression in advanced cases, progression of neurological deficits despite appropriate therapy (ongoing deterioration)/poor response to chemotherapy, drainage of cold abscesses, spinal instability, and diagnostic purposes in patients with nondiagnostic FNAB results. Surgery should be performed early in patients with acute neurological impairment and instability of the spinal column. It may be delayed in clinically and neurologically stable patients [67, 68, 82–85].

The purposes of surgical therapy are debridement and removal of infected tissues, relief of pain, and improvement of neurological deficits by decompression and spinal stabilization, correcting any deformities and restoring function [1]. Surgical therapy includes debridement of infected material, drainage of abscesses if present, decompression, bone grafting, and spinal stabilization by hardware use [85, 86]. Depending the portion of the vertebral body and the vertebral level involved, whether cold abscesses are present or not, different surgical approaches have been described [87–89]. In early phase of the disease, posterior stabilization with hardware instrumentation is performed to prevent kyphosis. Anterior surgical approach is preferred in some cases who present with spinal deformity late in the course of the disease to prevent progression of the deformity [68]. Hodgson et al. reported a success rate with anterior approach surgical decompression by resection and autologous bone grafting and anti-TB chemotherapy [90]. Reconstructive surgery for correction of spinal deformities (e.g., kyphosis) may be needed in some cases after completion of anti-TB therapy. Hardware use may be required for stabilization of the vertebral column [89]. Patients with neurological symptoms and/or destructive bone lesions of the thoracic or lumbar vertebrae may benefit from minimally invasive surgical interventions such as video-assisted thoracoscopic anterior surgery [91, 92]. The need for surgery in treatment of musculoskeletal tuberculosis involving

other parts of the skeleton is not always clear [79]. Therefore, surgery is not warranted routinely in all cases of ST [93, 94].

Neurological motor deficit has been graded in four categories as Grade1, negligible; Grade 2, mild; Grade 3, moderate; and Grade 4, severe (including sensory and autonomic dysfunctions). Conservative treatment is recommended in Grade 1 and 2; surgical therapy is favored in Grade 4. Grade 3 patients remain in gray zone in which there is no consensus on decision of therapeutic modality [95].

Management of ST in HIV-negative and HIV-infected patients does not differ [95]. But monitorization for immune reconstitution inflammatory syndrome (IRIS) is highly recommended in HIV-infected patients receiving concomitant anti-TB therapy for ST and antiretroviral treatment (ART) for HIV infection. After initiation of ART, paradoxical progression of clinical and laboratory findings of TB should alarm for the development of IRIS. IRIS presents with new clinical manifestations and/or imaging findings, or reappearance of resolved signs or symptoms [96, 97].

For surgical treatment of thoracic and lumbar ST, either anterior or posterior approach surgery may be preferred, but posterolateral approach allows better correction of the kyphotic angle and improvement in back pain [98]. However, posterolateral surgery takes more operative time and causes more blood loss and postoperative sinus formation [99–101]. Furthermore, patients who have not improved after posterior approach surgery may need anterior approach surgery [88]. For the treatment of thoracic and thoracolumbar ST, single-stage transpedicular debridement, posterior instrumentation, and fusion have been reported to be effective with satisfactory long-term postoperative outcomes [102]. In patients with single-segment spinal tuberculosis, use of titanium mesh cages resulted in comparable clinical efficacy with autologous iliac bone grafts. They can also be used in surgical treatment of multi-segment spinal tuberculosis with good clinical efficacy [103]. In thoracic ST with multilevel contiguous vertebral involvement, posterior instrumentation was more useful for durable correction of kyphosis [99]. Also, posterior surgical approach is effective in treatment of lumbosacral spinal TB [104, 105].

Monitorization of Response to Therapy Clinical response is assessed by resolution of the symptoms and signs and improvement of sensory and motor neurological functions. Inflammatory markers such as C-reactive protein (CRP), ESR have a limited role in evaluation of response to therapy. A two-third reduction in serial ESR measurements and decrease in CRP levels within 6 weeks in the postoperative period may indicate good response to therapy and rapid neurological recovery [106]. Despite appropriate therapy, radiological findings may resolve lately in the course of disease, even progression may be observed. Thus, repeated serial imaging studies are not recommended [107]. Mild weakness/improved muscle power, lower paraplegia scores, sensory-evoked potentials (SEPs), and motor-evoked potentials (MEPs) were the predictors of 6-month favorable outcome in patients with paraplegia due to Pott's disease [108]. Follow-up of the patients should continue until at least 1–5 years year after completion of treatment to determine long-term outcome of therapy.

7.7 Prognosis

Favorable outcome in ST can be defined as "full physical activity of the skeleton with clinical and radiological improvement of the disease, with no functional impairment." Levels of vertebral involvement, the score of the patient according to American Spinal Injury Association Impairment Scale (AIS grade) at presentation, and bladder and bowel involvement affect the final outcome of neurological improvement significantly [35].

Two percent mortality has been reported by Turgut M et al. and by Batirel A et al. in their study including a total of 694 cases and 314 cases with ST, respectively [27, 31]. Permanent sequelae have been reported in ¼ of the patients due to delayed diagnosis. The most common sequelae (4–11%) were kyphosis/gibbus deformity, scoliosis, paraparesis, paraplegia, and loss of sensation [27]. Older age, presence of spinal deformity, and neurologic deficit were found to be predictors of unfavorable outcome [27].

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