

Thoracal Spinal Fusion In Tuberculous Spondylitis With Pulmonary Oedema And Thrombocytosis: A case report

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Abstract

TB is caused by *Mycobacterium tuberculosis* (M. tb). Tuberculosis (TB) prevalence is increasing in developed nations and continuing to cause significant mortality in low and middle income countries. When M. tb disseminates to the vertebral column, it is called Pott's disease or spinal TB. The frequency, symptoms, and severity of the disease range by the location of the spine and the region of the affected vertebrae. The delay in establishing diagnosis and management results in complications such as spinal cord compression and spinal deformity. Tuberculous spondylitis is the most dangerous form of musculoskeletal tuberculosis because it can cause bone destruction, deformity and paraplegia. The spread of tuberculosis infection will cause inflammation in paradiscs, and causes progressive bone destruction and then will make vertebral collapse and deformity arising shaped kyphosis (posterior angulation) called gibbus. The role of surgical debridement and fusion with TB is controversial, and good results may be obtained with either medical treatment or surgery. Surgery may be more appropriate when definite cord compression is documented or for complications such as abscess or sinus formation or spinal instability.

Keywords: M. tb, Pott's disease, Spondylitis tuberculosis, Spinal fusion, Spinal deformity

Introduction

Mycobacterium tuberculosis (M.tb) is the second leading cause of death from an infectious agent worldwide. In 2018, approximately 10 million people became infected with TB, and 1.5 million died due to TB. Of note, more than two thirds of the global TB burden is reported in Africa and Asia, and in absolute terms, six countries accounted for 60% of the new cases: India, Indonesia, China, Nigeria, Pakistan, and South Africa. Primary infection by M. tb often seeds itself in the lungs of its host, causing Tuberculosis (TB), though in rare instances, the infection may spread to the bone and joints. Skeletal TB comprises 10% of extrapulmonary TB cases, of which 50% involve the spinal column. Spinal TB is otherwise known as Pott's disease, named after Pervical Pott, the first patient to present with classic spinal TB in 1779.

Pott's disease has been associated with significant morbidities and serious functional impairments, and comes second as the most common forms of tuberculosis after pulmonary tuberculosis. Although uncommon, spinal TB still occurs even in both developed and developing countries. The diagnosis of spinal TB is difficult and it commonly presents at an advanced stage. Delays in establishing diagnosis and management contribute to higher rates of complications such as spinal cord compression and spinal deformity. Patients mostly present with lower limb weakness, Gibbus deformity, pain, palpable masses, and kyphotic deformity in long-standing cases.

The most common levels involved are the lower thoracic and upper lumbar levels. Has a predilection for the vertebral body, sparing the posterior elements, and characteristically, sparing the intervertebral disc unlike most pyogenic infections. Neurologic deficit develops in 10–47%

of patients, and may be due to medullary and radicular artery inflammation in most cases. The infection itself rarely extends into the spinal canal; however, epidural granulation tissue or fibrosis or a kyphotic bony deformity may cause cord compression.

In some cases, surgical procedures such as laminectomy, abscess drainage, costotransversectomy, or anterolateral decompression may be required. The role of surgical debridement and fusion with TB is controversial, and good results may be obtained with either medical treatment or surgery. Surgery may be more appropriate when definite cord compression is documented or for complications such as abscess or sinus formation or spinal instability.

Research Methods

This study employs a case study approach with a qualitative descriptive method to analyze a case of tuberculous spondylitis accompanied by pulmonary edema and thrombocytosis. The study focuses on evaluating the diagnosis, management, and therapeutic outcomes of patients experiencing complications from Pott's disease, with the objective of understanding the role of thoracic spinal fusion in treatment and recovery.

The data used in this research were obtained from clinical case reports, radiological examinations, and laboratory results. Primary data sources include patient medical records, imaging results (X-ray and MRI), and details of the surgical interventions performed. Meanwhile, secondary data were collected from literature reviews related to tuberculous spondylitis, its associated complications, and medical and surgical management strategies.

Data collection methods involved analyzing patient medical records, which included disease history, physical examinations, and laboratory findings. Additionally, imaging interpretations were conducted to identify the extent of vertebral damage, the presence of abscesses, and potential spinal cord compression. The surgical procedure performed was also documented in detail, including the vertebral fusion techniques applied and the pharmacological therapy administered postoperatively.

Data analysis was conducted using a descriptive approach, where clinical findings were classified based on diagnostic aspects, intervention methods, and patient treatment outcomes. The effectiveness of the therapy was evaluated by comparing the patient's condition before and after medical and surgical interventions. Furthermore, the study findings were compared with existing literature to assess the alignment of the management strategies with established medical protocols.

The results of this study are expected to provide additional insights into the best approaches for managing tuberculous spondylitis with complex complications. Thus, this research can contribute to enhancing clinical understanding of factors influencing therapeutic success and provide recommendations for managing similar cases in the future.

Results and Discussion

A 42-year-old woman came to the emergency room with complaints of shortness of breath that had worsened in the past 1 day. The patient also complained of back pain that radiated to the chest wall and abdomen. The patient complained of weakness in both legs that had been felt since 1 month. The patient said that he was comfortable in a sitting position but the shortness of breath became worse when sleeping. The patient complained of fever up and down, long cough, and weight loss of about 7 kg felt since 1 month. The patient has a history of pulmonary TB 3 years ago and a history of using anti-tuberculosis drugs, but did not complete.

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On examination, she was weak with compos mentis consciousness. Blood pressure 130/90 mmHg, pulse 100 beats per minute, respiratory rate 30 beats per minute, axillary temperature 36.8 OC, and oxygen saturation 97% with nasal cannula 4 liters per minute. On eye examination, there were no anemic conjunctiva or icteric sclera, pupils were 3 mm/3 mm isocoric. The lungs looked flat, vesicular breathing sounds in both lung fields, additional coarse rales were found in both lungs, normal heart sounds and no murmurs were found. On examination of the extremities, weakness was found in the left lower limb with a motor strength of 5/5/3/3/3/3. Spinal palpation found gibbus and tenderness in the thoracic region.

On X-ray examination, cardiomegaly was found with pulmonary oedema and infiltrates in the upper to lower lung fields bilaterally with the impression of pulmonary TB. Laboratory examination showed haemoglobin (Hb) 10.5 g/dL, leucocytes (WBC) 11,100 /uL, platelets 589,000 /uL.

The patient was performed vertebral fusion with pedicle screw at thoracic 4-5 and thoracic 8-9 left and right, then ROD was applied, followed by laminectomy at thoracic 6-7. Biopsy samples were taken from the transpedicular of the left thoracic 7 and then applied sub cutis drain. Patients were also given drug therapy betaloc 50 mg in the morning, spironolactone 100 mg in the morning, furosemide injection 20 mg per 12 hours, meropenem injection 1 gram per 12 hours, budesma nebul 0.5 mg + lasalcom per 8 hours. The patient was also transfused PRC and WB 1 bag.

Discussion

During primary infection, the hematogenous and lymphatic spread can lead to extrapulmonary seeding of *M. tuberculosis*. Usually, infection is subclinical, as adaptive and cellular immune processes confine it to foci. Primary infection by *M. tb* often seeds itself in the lungs of its host, causing Tuberculosis (TB), though in rare instances, the infection may spread to the bone and joints. Extrapulmonary tuberculosis can occur with foci reactivation after primary infection, frequently in latent tuberculosis.

Pott's disease (tuberculous spondylitis) is one of the forms of infection of the spine that is caused by *Mycobacterium tuberculosis*. This disease has been associated with significant morbidities and serious functional impairments, and comes second as the most common forms of tuberculosis after pulmonary tuberculosis. Usually affects more than one level. The most common levels involved are the lower thoracic and upper lumbar levels. Has a predilection for the vertebral body, sparing the posterior elements, and characteristically, sparing the intervertebral disc unlike most pyogenic infections. Psoas abscess is common (the psoas major muscle attaches to the bodies and intervertebral discs from T12–5). Sclerosis of the involved vertebral body may occur.

In the thoracic region, kyphosis is clearly visible due to the presence of normal dorsal curvature; in the lumbar area, it is only slightly visible due to the presence of normal lumbar lordosis where most of the body weight is transmitted posteriorly, resulting in partial collapse; while in the cervical region, collapse is only minimal, and if it is visible, it is because most of the body weight is transmitted through the articular processes. With the increase in the kyphosis angle in the thoracic region, the ribs will pile up, causing a barrel chest deformity.

Usually, infection begins at the anterior aspect of the vertebral body. Once two vertebrae are involved, the intervertebral disc is also affected with necrosis, vertebral narrowing, collapse, and Gibbus deformity (a form of kyphosis) with risk of spinal cord compression. Infection can spread to adjacent soft tissues, with paravertebral abscesses that can also cause spinal cord

compression. The bacteria travel through the vascular system, using the anterior and posterior spinal arteries of each vertebra, to ultimately reach the cancellous bone region of the vertebra, with the most common site being the thoracolumbar junction. Using the valveless Batson's paravertebral venous plexus, intrabdominal and intrathoracic pressures spread the infection to the inferior anterior portion of a vertebral body. From here, the infection may utilize the anterior longitudinal ligament to infect adjacent vertebrae. Paradiscal and central are among the more common presentations of spinal TB, along with anterior/non-osseous and posterior lesions.

In extensive disease, the infection spreads to involve the ligaments, paravertebral soft tissue, the epidural space, and formation of a cold abscess. The abscess may compress the spinal cord or nerve roots with resulting neurological deficits, and may extend into the retroperitoneal space. Spinal TB more frequently presents in children and young adults due to increased vascularization of their spine, often with paradiscal lesions and in immunosuppressed patients.

Type of involvement	Mechanisms of involvement	Radiological appearances
Paradiskal	Spread of disease via the arteries	Involves adjacent margins of two consecutive vertebrae. The intervening disk space is reduced.
Central	Spread of infection along Batson's plexus of veins	Involves central portion of a single vertebra; proximal and distal disk spaces intact.
Anterior marginal	Abscess extension beneath the anterior longitudinal ligament and the periosteum	Begins as destructive lesion in one of the anterior margins of the body of a vertebra, minimally involving the disk space but sparing the vertebrae on either side.
Skipped lesions	Spread of infection along Batson's plexus of veins	Circumferentially involvement of two noncontiguous vertebrae levels without destruction of the adjacent vertebral bodies and intervertebral disks.
Posterior	Spread via the posterior external venous plexus of vertebral veins or direct spread	Involves posterior arch without involvement of vertebral body
Synovial	Hematogenous spread through subsynovial vessels	Involves synovial membrane of atlanto-axial and atlanto-occipital joints

Table 1. Various types of vertebral involvement in spinal tuberculosis

Spinal TB remains the most prevalent spine infection globally, and in cases with extensive spine involvement, vertebral body collapse or severe deformity, surgical options may be considered more appealing, allowing for simultaneous treatment of infection and deformity. Kyphotic deformity as a sequela of spinal tuberculosis results from the destruction of vertebral bodies by *Mycobacterium tuberculosis* abscesses. Three types of reconstitution may result after resolution of active infection: Type A, with minimal vertebral body destruction and an intact posterior column; Type B, which causes 40° to 60° of kyphosis; and Type C, which causes more than 100° of kyphosis. The clinical appearance depends on the number of vertebrae involved causing "knuckle" (1 vertebra), "gibbus" (2 vertebrae), and "rounded kyphosis" (>3 vertebral collapse).

Cold abscess lacks inflammatory features and initially forms in the infective focus. Later,

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it takes the path of least resistance along the natural fascial and neurovascular planes. In the cervical spine, it can present as a retropharyngeal abscess or as a swelling in anterior or posterior triangle of neck or even in the axilla. Retropharyngeal abscess can produce dysphagia, hoarseness of voice, and respiratory stridor. In the thoracic region, the cold abscess usually presents as a fusiform paravertebral swelling, seen radiographically, and it can track along the intercostal vessels and presents as a swelling in the chest wall. Thoracic cold abscess can track down through the arcuate ligament, or via the openings in the diaphragm. The lumbar cold abscess usually present as a swelling in Petit's triangle or in the groin and can track down along the psoas to cause pseudo-flexion deformity of the hip. Rarely they can track femoral or gluteal vessels to present as an abscess in Scarpa's triangle or gluteal region.

Spondylitis TB classification system was based on seven clinical and radiological criteria (abscess formation, disc degeneration, vertebral collapse, kyphosis, sagittal index, instability and neurological problems). It also recommends specific techniques for each type. We have divided tuberculosis of the spine into three types by using GATA (Gulhane Askeri Tip Akademisi) classification of spinal tuberculosis.

Type	Lesion	Treatment
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
	B Abscess formation, one or two level disc degeneration, no collapse, no neurologic deficits.	Abscess drainage and debridement
Type II	1. Vertebral collapse (pathological fracture) 2. Abscess formation 3. Kyphosis (correctable with anterior surgery) 4. Stable deformity with or without neurological deficit. Sagittal index $< 20^{\circ}$	1. Anterior debridement and fusion 2. In existence of neurological deficit decompression should be added 3. Strut cortical graft is used for fusion
Type III	1. Severe vertebral collapse 2. Abscess formation 3. Severe kyphosis 4. Unstable deformity with or without neurological deficit. Sagittal index $< 20^{\circ}$	1. Anterior debridement and fusion 2. Decompression 3. Correction of deformity and internal fixation (anterior, posterior, or both)

Table 2. GATA classification of spinal tuberculosis.

Vitamin D deficiency was also correlated with increased susceptibility to Pott's disease, specifically caseous necrosis-type spinal TB, as well as an increased likelihood of necrosis compared to individuals with normal vitamin D levels. Active vitamin D is thought to enhance innate immunity by promoting the fusion of macrophage and phagolysosome complexes, mediating reactive oxygen species (ROS) generation, and reducing peroxisome proliferator activated receptor expression to inhibit lipid metabolism in TB-infected macrophages. Upon initial infection with *M. tb*, alveolar macrophages (AMs) are the first responders in generating an

immune response. Active vitamin D also enhances adaptive immunity by promoting FoxP3+/IL-10+ Treg cell differentiation and boosting T-cell immune tolerance.

The most common symptom is local pain, increasingly intense over weeks or months, associated with muscle spasm and rigidity. The usual TB symptoms of fever, weight loss, malaise, and night sweats are only present in less than 40% of patients diagnosed with extrapulmonary TB, while back pain and lower limb weakness are the most common symptoms usually present in about 80% and 73% of patients, respectively. Neurological alterations suggestive of spinal cord compression can also occur, as well as constitutional symptoms (fever and weight loss). Movement limitation and kyphosis may also occur. Neurologic deficit develops in 10–47% of patients, and may be due to medullary and radicular artery inflammation in most cases. The infection itself rarely extends into the spinal canal; however, epidural granulation tissue or fibrosis or a kyphotic bony deformity may cause cord compression.

The diagnosis of skeletal tuberculosis is particularly difficult, as samples of affected tissue are difficult to obtain. Diagnosis is difficult and usually associated with a significant delay, either for progressive and indolent nature or for capacity to mimic another diseases, such as primary/metastatic neoplasm, traumatic injury, osteoporosis, bacterial/fungal infections, Paget's disease, and others. Blood investigations used to diagnose TB include complete blood count, erythrocyte sedimentation rate (ESR), enzyme-linked immunosorbent assay (ELISA), and polymerase chain reaction (PCR). ESR has a sensitivity of 60–90% and is used to monitor therapeutic response. PCR with a sensitivity of 75% and specificity of 97% is useful in the paucibacillary state. Skin tests like Mantoux test with 40–55% sensitivity and 75% specificity is of no diagnostic value in endemic areas due to false-positive results in *Bacillus Calmette–Guérin*-vaccinated individuals.



Figure 1. Postoperative X-ray imaging with radiological sign of bone fusion

WBC elevated in only 35% (rarely > 12,000), associated with poor prognosis. ESR: elevated in almost all. Usually > 40 mm/hr. Mean: 85. CRP: may be more sensitive than ESR, and may tend to normalize more quickly with appropriate treatment. Needle biopsy with cultures: can usually be done percutaneously via transpedicular approach with CT or fluoroscopic guidance. May be helpful even if blood cultures are positive (different organisms retrieved in

15%) an attempt at direct culture from the involved site should be made. Cultures should include fungal, aerobic and anaerobic bacterial, and TB. Ideally, cultures should be done before antibiotics are started. The yield of needle biopsy cultures ranges from 60–90%. Open biopsy is more sensitive, but morbidity is higher.

Radiography may locate lesions and evaluate the extension of the disease, but there are no pathognomonic findings. Suggestive findings (demineralization and loss of definition of bony margin) are usually in the anterior aspect of vertebral body. Commonly two adjacent vertebrae are involved. Progression may cause anterior collapse and Gibbus deformity. Some have lytic lesions without involvement of the vertebral disc. CT, myelography, and MRI are also useful in establishing a diagnosis, particularly MRI, as it identifies soft-tissue involvement and spinal cord compression. MRI T1WI confluent low signal in vertebral bodies and intervertebral disc space. T2WI increased intensity of involved VBs and disc space. Contrast enhancement of VB and disc, also look for paraspinal and epidural mass. If MRI is contraindicated CT-myelogram assesses bony anatomy and can demonstrate spinal canal compromise. Bone scan may occasionally be helpful if the diagnosis is still uncertain when suspicion is high. CT scan helpful for demonstrating bony involvement as well as detailed bony anatomy in case instrumentation is required during treatment. CT may be negative if done too early in the course. Plain X-ray changes take from 2–8 weeks from the onset of infection to develop. Earliest changes are loss of cortical endplate margins and loss of disc space height. Bone scan three phase bone scan has reasonably good sensitivity and specificity. Gallium scan has better accuracy, findings include increased uptake in the 2 adjacent VBs with loss of intervening disc. Indium-111 labeled WBC scan: low sensitivity for vertebral osteomyelitis.

The goal of management of spinal TB is to eradicate the infection, prevent or treat neurological deficits, and to correct or halt the progress of spinal deformity. Treatment includes the same regimen used for pulmonary tuberculosis but should be prolonged, as there is a risk of recurrence with short cycle therapy. The ideal duration is not known, but usually, 6–9 months of first-line antitubercular drugs are recommended. The second-line antitubercular treatment (ATT) drugs (kanamycin, amikacin, capreomycin, levofloxacin, etc) have to be used judiciously as they have more side effects and are expensive than the standard first-line ATT drugs (isoniazid [INH], rifampicin, ethambutol, pyrazinamide). 90% of cases can be managed non-surgically with antibiotics and immobilization. Characteristics of potential candidates for non-surgical treatment are listed in Table 3.

organism identified
antibiotic sensitivity
single disc space involvement with little VB involvement
minimal or no neurologic deficit
minimal or no spinal instability

Table 3. Candidates for non-surgical treatment in pyogenic spontaneous spondylodiscitis.

In advanced stages with deteriorating neurologic functions, the medical treatment, that is, directly observed treatment short-course (DOTS) antitubercular treatment (ATT) in conjunction with radical surgery is required. Indications for surgery in acute tuberculous spondylitis: (1) Progressive neurologic deficit, (2) Progressive increase in spinal deformity (coronal or sagittal), (3) Failed conservative treatment including 1 and 2 above or severe pain due to abscess or spinal

instability, (4) Uncertain diagnosis: this could be an inability to obtain microbiological diagnosis from microscopy, culture or even via detection of mycobacterium DNA using polymerase chain reaction (PCR) techniques. The goals of modern surgical management of spinal TB include debridement of diseased vertebrae, decompression of spinal cord, correction of deformities, debridement and fusion with or without instrumentation which can be achieved by spinal reconstruction or fusion procedures (e.g., plate and screws), laminectomy (for posterior spinal disease or in paraplegia due to extradural granuloma/ tuberculoma), microdiscectomy or costotransversectomy.

Various approaches available for the aforementioned procedures include anterior approach, transoral or trans-hyoid approach, posterior approach, extrapleural anterolateral approach, or combined procedures. The cervical spine is best approached by an anterior approach in the supine position. The transoral and transthyrohyoid approach are employed for lesions in atlantoaxial regions. The posterior approach is most commonly used due to familiarity and faster recovery but requires a prone position. The posterior approach offers the advantage of superior posterior stabilization. For the thoracic spine, several options are available including anterior and anterolateral decompression by thoraco-abdominal approach, and posteriorly there is costotransversectomy and laminectomy. Particularly in the thoracic spine, there are more chances of deformity as the weight passes through the anterior column. For lumbar level spinal TB, posterior, anterior and antero-lateral are all viable options.

Pedicle screw fixation is usually avoided in the presence of clear infection, however this may be disrupted if stability of the spinal column is required. Pedicle fixation is contraindicated in cases with high probability of failure such as in cases of pedicle size that is too small, osteoporosis and inadequate anterior column support that is inadequate. Recently, minimally invasive surgery (MIS) has been used either as stand-alone or in combination with open procedures. The MIS procedures include thoracoscopic debridement, posterolateral endoscopic debridement, and MIS transforaminal interbody fusion. Successful outcomes have been reported; however, their role in cases with severe neurological deficits and extensive osseous destruction is questionable.

Conclusion

Pott's disease (tuberculous spondylitis) arises from the extrapulmonary dissemination of *M. tb* to cancellous bone in the vertebrae through the anterior and posterior venous plexuses, ultimately spreading to adjacent vertebrae, which yield the characteristic back pain and neurological deficit symptoms. The goal of treatment in spinal TB is to eradicate the disease and prevent or treat the spine deformity and/or neurologic deficits. Surgery in spinal TB is directed toward achieving adequate decompression and debridement, maintenance, and reinforcement of stability and halting the progress of deformity or finally correcting the deformity in healed disease. The advantages of surgery include thoroughness of debridement, decompression of the spinal cord, and adequate spinal stabilization.

In this patient, there was a neurologic deficit where the motoric score on the left foot dropped to 5/5/5/3/3/3/3. This occurred due to the compression of the paraspinal abscess on the spinal cord. This patient was treated with vertebral fusion at thoracic level 4-5 and thoracic level 8-9 using pedicle screws with a size of 5.5 x 45 cm as many as 8 pieces and a 25 cm rod as many as 1 piece then laminectomy was done at thoracic 6-7.

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