

Tuberculous spondylitis presenting as severe chest pain

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Abstract

This case report describes a 32-year-old male who presented to an emergency department with severe chest pain and a history of cough, fever, night sweats, loss of appetite and weight. Chest radiography revealed a left upper lobe consolidation and multiple compression deformities in the thoracic spine. Magnetic resonance imaging demonstrated significant kyphosis and vertebral plana at two thoracic levels. Anterior compression of the spinal cord and adjacent soft tissue masses were also noted.

Introduction

Tuberculosis (TB), caused by the organism *Mycobacterium tuberculosis* (*M. tuberculosis*), is a disease that can affect any organ system and has devastating impact if untreated.¹ Ordinarily, TB manifests in the chest and is either primary or post primary. Primary TB is often seen in infants and children, but involves 23-34% of all adult cases. In 90% of the cases, postprimary TB, almost exclusively occurs in adolescents and adults the result of reactivation of a previously dormant infection. The other 10% represent continuation of primary TB.¹

The numbers of people affected by TB continue to rise as a result of a rapid increase in the world population.² The World Health Organization reported the incidence of tuberculosis worldwide to be 9.4 million in 2009 with India ranking #1 at 1.6 to 2.4 million cases. The prevalence of tuberculosis in 2009 was 14 million cases. Approximately 1.7 million people died of tuberculosis in 2009.³ While a rapidly increasing world population has increased the numbers of people with TB,⁴ Harisinghani, *et al.* reports that a resurgence of TB has occurred

since the mid 1980s due to the acquired immunodeficiency syndrome epidemic and increasing drug-resistant strains of TB.¹

Tuberculosis is not only a disease that affects an individual's health but it also has social and economic consequences. The perceived risk of transmission leads to stigmatization and isolation of individuals with tuberculosis. In areas where human immunodeficiency virus (HIV) and TB co-exist, individuals are assumed to have HIV if they are positive for TB. TB is often perceived to be associated with malnutrition, poverty, being foreign-born and low socioeconomic status.⁵

Musculoskeletal tuberculosis afflicts the spine in 50% of cases⁶ and is the most common site for osseous tuberculosis accounting for 1-2% of all the patients with TB.⁷ It involves the spine through hematogenous spread^{7,8} and is referred to as tuberculous spondylitis or Potts' disease.¹ Spinal TB is indolent and slow growing⁹ with 10-47% of individuals developing neural complications.⁶ The purpose of this case report is to describe an unusual presentation of tuberculous spondylitis presenting as severe chest pain. This case is unique in that tuberculous spondylitis rarely spreads hematogenously to the spine and when it does neurological disorders, not present in this report, will likely manifest. We also include a discussion of a spectrum of systems affected by tuberculosis with the intent to provide clinicians a large amount of information in one case report.

Case Report

A 32-year-old Indian male presented to an emergency and trauma center with severe posterior chest pain. During the previous 6 months he reported a cough, fever, night sweats, loss of appetite and weight. Physical examination demonstrated a temperature of 38.9°C. Slight tenderness was reported with palpation of the middle chest. Auscultation demonstrated crepitation in the inferior upper lobe and lower lobe of the left lung. The remaining examination of the other body systems was unremarkable.

Lab examinations revealed low hemoglobin 12.0 g/dL (reference range 14.0-17.5 g/dL), thrombocytosis 436,000/mcL (reference range 150,000-300,000), high ESR 135 (reference range <15 mL/hr) and high urine protein 10.0 mg/dL (reference range 0-8.0 mg/dL). A PA chest radiograph demonstrated a consolidation in the inferior aspect of the left upper lobe (Figure 1). Incidentally noted were compression deformities of T11 and T9. No history of trauma was reported. A magnetic resonance imaging (MRI) examination performed the next day demonstrated severe collapse of the

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T9 and T11 vertebral bodies with retropulsion of the vertebral bodies resulting in anterior compression of the spinal cord on T2 weighted sagittal images. Early changes were also observed in the vertebral body of T3 (Figure 2A). Mild gibbus dislocations were also noted at both levels. The superior and inferior intervertebral discs adjacent to the collapsed vertebral bodies appeared unremarkable with minimal desiccation noted. Anterior and lateral to the vertebral bodies, a subligamentous soft tissue mass was noted extending from the superior aspect of T7 caudad to the inferior aspect of T12. A heterogeneous signal was noted within the soft tissue mass with multiple fluid levels consistent with an abscess. Similar to the anterior abscess was a posterior subligamentous heterogeneous soft tissue mass extending from the inferior aspect of T8 caudad to T12. This mass was also characteristic of an abscess with fluid levels. Axial T1 weighted MRI with gadolinium at T11/12 demonstrated the soft tissue abscesses adjacent to the vertebral body. The spinal cord was anteriorly compressed by the subligamentous and epidural abscess collections and narrowing of the lateral recesses was noted (Figure 2B). Coronal T1 weighted MRI with gadolinium demonstrated the paravertebral soft tissue abscess and collapse of the vertebral bodies at T9 and T11 (Figure 2C).

Instrumental stabilization of the spine was

offered, yet intervention was declined. The patient was prescribed ambulant chemotherapy for six months with daily isoniazid and rifampicin. The result of polymerase chain reaction (PCR), from a sputum sample, was positive for *M. tuberculosis*.

Discussion

Tuberculosis affects one-third of the world population and given the latency of the infection these individuals are at risk for reactivation or acute disease.¹⁰ TB forms granulomatous lesions which are focal compact collections of inflammatory cells where mononuclear cells dominate. The granulomas, in healthy individuals contain the infection and are assumed to form as a host defense mechanism.² There is a 10% lifetime risk of developing active clinical TB after inhalation of *M. tuberculosis* from someone infected with active TB. Transmission is most likely in the first few years after infection. Immunocompetent individuals either eliminate the organism or contain it in a latent state.² When an individual's innate immune system cannot eliminate the organism or contain it, it will immediately begin to proliferate resulting in primary TB.¹¹ Tuberculosis is a multisystem disease. It not only involves the pulmonary system but also the musculoskeletal, genitourinary tract and central nervous system. Individuals with HIV are especially prone to involvement beyond the pulmonary system.¹ Sixty percent of patients with HIV will present with skeletal tuberculosis as opposed to 1-2% of HIV negative patients.⁷ Table 1 lists the systems of involvement and the complications.¹

Primary tuberculosis manifests as parenchymal disease, lymphadenopathy, pleural effusion, military disease or atelectasis. Parenchymal lung disease in primary tuberculosis affects the areas where ventilation is the greatest; the middle lobe, the lower lobes and the anterior segment of the upper lobes. Differential diagnosis for tuberculous lymphadenopathy includes metastases and histoplasmosis. Miliary disease differentials include tuberculosis, varicella pneumonia, sarcoidosis, histoplasmosis, metastases, pneumoconiosis or hemosiderosis. Post primary tuberculosis in the lungs presents as parenchymal disease with cavitation, airway involvement, pleural extension and other complications such as chest wall tuberculosis secondary to pleural disease and empyema or hematogenous spread.¹

Chest radiography is often used to exclude clinically active TB. It presents radiographically in the chest as parenchymal disease, lymphadenopathy, pleural effusion, miliary disease or atelectasis. It is important to note, 15%

of chest radiographs may be normal.¹ The American Thoracic Society and the Centers for Disease Control have recommended the use of chest radiography to exclude active TB if an individual has a positive tuberculin skin test (TST) or to detect previous TB infection. One study in a large tuberculosis screening program (2586 individuals) concluded that there was a low yield in the detection of active or latent TB.¹²

Tuberculous spondylitis most commonly affects the upper lumbar and lower thoracic spine.¹⁷ It typically resides in the endplates of the anterior aspect of the vertebral body before involving the entire vertebra. Spread to adjacent vertebral bodies is by subligamentous extension and is a highly specific finding.⁸ The formation of abscesses can result in vertebral body destruction and neurological disorders¹³ which occur secondary to vertebral involvement.¹⁴ Involvement of contiguous vertebral bodies is often the presentation.⁷ The posterior elements are not uncommonly involved and when involvement of the pedicles occur it usually is associated with severe vertebral body and disc destruction, wide prevertebral abscess and severe kyphosis.⁸ Differential diagnosis when considering destruction of a vertebral body includes a primary or secondary neoplasm (lymphoma, multiple myeloma, chordoma, metastases)¹ but the presence of an abscess and bone fragments favor spinal tuberculosis. Pyogenic discitis may be included in the differential diagnosis when preservation of the disc is noted but heterogenous enhancement is typical of tuberculosis whereas a homogeneous enhancement is common with pyogenic discitis (vertebral osteomyelitis), as well as peridiscal bone destruction.⁹ Rare spinal infections (brucellosis, fungal disease



Figure 1. Chest x-ray showed left mid-zone consolidation, consistent with pulmonary tuberculosis. An incidental finding was severe wedging of T11 and to a lesser extent T9.

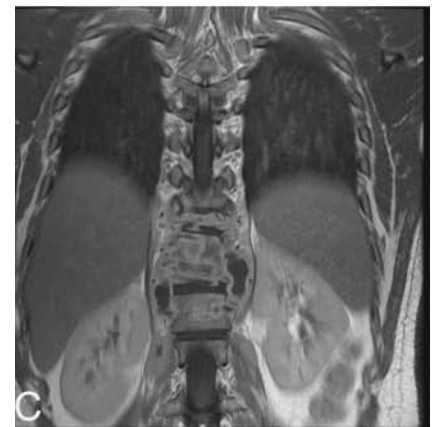


Figure 2. (A) Sagittal T2W SE, (B) axial T1W SE with gadolinium at T11-T12 disk level and (C) coronal T1W SE with gadolinium showed progression of wedging at T9 and T11 in comparison to the chest radiograph. This resulted in mild kyphosis and gibbus dislocation. Subligamentous and epidural abscess collections were noted with compression of the spinal cord and the lateral recesses (A, B). Bone marrow edema and early changes were noted in the vertebral body of T3 (A). Notice the preservation of disk signal and height adjacent to the involved vertebrae (A, C).

Table 1. Extrapulmonary systems affected with tuberculosis and presentation.

System	Presentation
Cardiac	Calcific pericarditis
Musculoskeletal	
Spinal	Tuberculous spondylitis (Pott disease)
Extraspinal	Tuberculous osteomyelitis, dactylitis, arthritis
Gastrointestinal	Ileocecum and colon: ulcerative, hypertrophic or ulcerohypertrophic changes Peritoneum: diffuse or loculated ascitic fluid or caseous nodules, fibrous peritoneal reaction and dense adhesions Lymph Nodes: lymphadenopathy (m/c manifestation of abdominal TB) Liver and Spleen: micronodular (miliary) or macronodular
Genitourinary	Kidneys: hydronephrosis Parenchyma: calcification and scarring Collecting system: ulceration, wall thickening and fibrosis Cortex: scarring Adrenal Glands: unilateral or bilateral masses with central areas of necrosis, enlarged glands (recent or concurrent TB), adrenal atrophy w/calcification (healed prior TB) Ureter: dilatation and ragged appearing, filling defects, thickening of the ureteral wall, periureteral inflammatory changes, short or long segment ulceration Bladder: reduced bladder capacity (m/c finding in tuberculous cystitis), wall thickening, filling defects, Genitalia: Female: endometrial adhesions, obstruction of fallopian tubes and multiple areas of constriction, calcified lymph nodes in the adnexal region Male: necrosis, calcification, caseation and cavitation of prostate gland or seminal vesicles, epididymo-orchitis
Central nervous system	Meningeal involvement: hydrocephalus and infarcts in the middle cerebral artery distribution Parenchymal involvement: solitary or multiple tuberculomas Tuberculous otomastoiditis Ocular tuberculosis

TB, tuberculosis.

and echinococcosis) may also have imaging characteristics similar to spinal tuberculosis.¹

Central nervous system tuberculosis accounts for 2-5% of all cases.¹⁵ It is associated with a high mortality rate and severe neurological sequelae and includes three clinicopathological forms: meningoencephalitis, tuberculomas and abscesses.¹⁵ Clinically, meningitis presents most commonly as headache, nuchal rigidity, fever and vomiting. Less common findings are altered mental status, cranial nerve palsy and other focal neurologic signs. Tuberculin skin test is positive in less than 50% of the cases.¹⁰ An abnormal chest radiograph is identified in greater than 50% but less than a third have the classic miliary pattern of dissemination. Computed tomographic examination demonstrates hydrocephalus, basilar exudates or inflammation, tuberculoma, brain edema or cerebral infarction.¹⁰ The mortality for TB meningitis is between 10% and 50% at 1 year. This is primarily due to a delay in diagnosis. The differential diagnosis for tuberculous meningitis includes other bacterial agents, viruses, fungi and parasites. Noninfectious inflammatory diseases included in the differential diagnosis are rheumatoid disease and sarcoidosis. Neoplastic processes,

primary or secondary, which are similar in presentation to TB meningitis include meningiomatosis, neoplastic meningitis from a peripheral tumor source, cerebrospinal fluid seeding from a primary tumor of the central nervous system.¹

The use of MRI provides high sensitivity and satisfactory specificity. MRI is sensitive for the detection of early vertebral osteomyelitis.⁷ Computed tomographic examinations will demonstrate osseous destruction but fails to accurately define the epidural extension and impact on the neural structures.⁷ Consistent with our case, the spinal lesions are seen clearly on MRI with the vertebral bodies demonstrating low signal on T1 and high signal on T2. There is relative preservation of the discs. Septated abscesses are seen either intra-osseous or pre or paravertebral. Subligamentous extension with breaching of the epidural space is also characteristic of spinal tuberculosis.⁹

In this case, the patient reported no neurological symptoms and none were diagnosed with physical examination. Surgical stabilization was offered to the patient in order to prevent the development of paraplegia by decompressing the spinal cord and stabilizing the

spine. A spine is considered unstable with regards to tuberculosis when there is destruction from the infection and the mechanical insult has resulted in a pathological fracture of the vertebral body or when the facets and posterior complex are destroyed along with the vertebral body.⁶

Early surgical decompression is implemented when extradural compression is due to granulation tissue or caseous tissue and features of cord edema, myelitis or myelomalacia are present.⁹ Early diagnosis is necessary to prevent kyphotic deformity.¹ In addition to stabilizing the spine and preventing neurological deficits, the primary aim of treatment for tuberculosis of the spine is abolition of infection.⁶ Debridement of the area can occur during spinal stabilization or a second nonsurgical approach has been used, in which antitubercular drugs, ambulant chemotherapy and bed rest were used to sterilize the lesion.⁶ Both approaches achieved favorable results with only a slight progression of kyphosis before the lesion healed when surgical stabilization was not utilized.⁶

An obstacle to TB detection is the lack of accurate and rapid diagnosis. Delays between infection and treatment are a problem and must be addressed in order to effectively reduce transmission.^{4,10} In low and middle income countries, where 90% of TB occurs, diagnosis relies on sputum smear microscopy and chest radiology, two techniques that are unsatisfactory and unavailable.² Automated liquid culture systems are the gold standard for the diagnosis of TB. Since the early 1900s, the tuberculin skin test was used for latent infection with tuberculosis. This test was unable to distinguish those individuals who were infected with *Mycobacterium tuberculosis* from those with other mycobacteria. Currently, interferon- γ release assays (IGRAs) are the gold standard for identifying latent TB.² Polymerase chain reaction (PCR) using a variety of tissue samples, including sputum¹⁶ used to diagnose the patient in this report, has shown a sensitivity and specificity of 82.65% and 91.00% respectively.¹⁷ The process of diagnosing TB using PCR can be completed in 3 to 6 h compared to 4-8 weeks using the conventional culture method.¹⁷

Treatment for drug sensitive TB as recommended by WHO includes 6 months of rifampicin, taken daily. Alternatives to the daily doses are permitted if the patient is compliant. Treatment for multi-drug resistant tuberculosis adopted by WHO includes regimens of 4 drugs for a total duration at a minimum of 18 months after culture conversion (2 negative smears and cultures taken 30 days apart) or 24 months in patients with chronic disease and extensive pulmonary damage.²

It has been suggested that vitamin D deficiency is associated with the risk of tuberculo-

sis. The role of vitamin D in the immune response includes promoting formation of phagolysosomes, needed to destroy *M. tuberculosis* bacilli, and antimicrobial peptides which prevent infection.¹⁸ Vitamin D supplementation may enhance antimycobacterial immune function.² The risk of reactivation is seen in individuals who are immunosuppressed due to HIV or treatment with corticosteroids or tumor necrosis factor antagonists.¹¹

Bacillus Calmette-Guérin (BCG) is the only approved vaccine for TB. In endemic countries with TB it is used to protect children against TB meningitis or disseminated infection.¹⁹ This vaccine has been shown to have limitations one of which is that it is becoming too attenuated through culture and modern preparations.¹⁹

Conclusions

The portal system of infection for TB is pulmonary, but extra-pulmonary manifestations are also seen. Systemic risk factors including immunocompromise, poor nutrition and low socioeconomic status have been reported. Tuberculous spondylitis is a common musculoskeletal manifestation of TB infection with high frequency in the thoracolumbar spine. TB in the musculoskeletal system warrants exclusion of pulmonary, renal or intestinal involvement. The use of MRI for the examination of tuberculous spondylitis provides high sensitivity and specificity increasing the likelihood of rapid and successful treatment.

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