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 continues 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. report 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 affects 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 spread8 and is referred to as tuberculous spondylitis or Pott’s disease.1 Spinal TB is indolent and slow growing9 with 10-47% of individuals developing neurological complications.10 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 crepitant 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 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 sublaminous 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 sublaminous 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 sublaminous 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 chemothera-
py 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 infec-
tion these individuals are at risk for reactiva-
tion or acute disease. TB forms granuloma-
tous lesions which are focal compact collec-
tions of inflammatory cells where mononu-
clear cells dominate. The granulomas, in
healthy individuals contain the infection and
are assumed to form as a host defense mecha-
nism. There is a 10% lifetime risk of develop-
ing 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. Immuno-
competent individuals either eliminate the
organism or contain it in a latent state. When
an individual’s innate immune system cannot
eliminate the organism or contain it, it will
immediately begin to proliferate resulting in
primary TB. Tuberculosis is a multisystem
disease. It not only involves the pulmonary sys-
tem but also the musculoskeletal, genitouri-
nary tract and central nervous system.

Individuals with HIV are especially prone to
involvement beyond the pulmonary system. Sixty percent of patients with HIV will present
with skeletal tuberculosis as opposed to 1-2% of HIV negative patients. Table 1 lists the sys-
tems of involvement and the complications.

Primary tuberculosis manifests as
parenchymal disease, lymphadenopathy, pleu-
ral effusion, military disease or atelectasis.
Parenchymal lung disease in primary tubercu-
losis affects the areas where ventilation is the
greatest; the middle lobe, the lower lobes and
the anterior segment of the upper lobes. Dif-
ferential diagnosis for tuberculous lym-
phadenopathy includes metastases and histo-
plasmosis. Military disease differentials
include tuberculosis, varicella pneumonia, sar-
coidosis, histoplasmosis, metastases, pneu-
monconiosis or hemosiderosis. Post primary
tuberculosis in the lungs presents as
parenchymal disease with cavitation, airway
involvement, pleural extension and other com-
plications such as chest wall tuberculosis sec-
dary to pleural disease and empyema or
hematogenous spread. Chest radiography is
frequently used to exclude clinically active TB. It presents radiographically
in the chest as parenchymal disease, lym-
phadenopathy, pleural effusion, military dis-
ease or atelectasis. It is important to note, 15%

![Figure 1. Chest x-ray showed left mid-
zone consolidation, consistent with pul-
monary tuberculosis. An incidental find-
ning was severe wedging of T11 and to a
lesser extent T9.](image1)

![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 wedg-
ing at T9 and T11 in comparison to the
chest radiograph. This resulted in mild
kyphosis and gibbus dislocation. Sublig-
mentous and epidural abscess colle-
ctions 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).](image2)
and echinococcosis) may also have imaging characteristics similar to spinal tuberculosis.6

Central nervous system tuberculosis accounts for 2-5% of all cases.16 It is associated with a high mortality rate and severe neurological sequelae and includes three clinicopathological forms: meningencephalitis, 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.16 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.16 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 meningiomas, 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.5 Computed tomographic examinations will demonstrate osseous destruction but fails to accurately define the epidural extension and impact on the neural structures.5 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.5

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.5

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.6 Early diagnosis is necessary to prevent kyphotic deformity.5 In addition to stabilizing the spine and preventing neurological deficits, the primary aim of treatment for tuberculosis of the spine is abolition of infection.5 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.1 Automated liquid culture systems are the gold standard for the diagnosis of TB. Since the early 1990s, 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.4 Polymerase chain reaction (PCR) using a variety of tissue samples, including sputum8 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 hours 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 tuberculosis.
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. Vitamin D supplementation may enhance antimycobacterial immune function. The risk of reactivation is seen in individuals who are immunosuppressed due to HIV or treatment with corticosteroids or tumor necrosis factor antagonists.

Bacillus Calmette-Guerin (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. This vaccine has been shown to have limitations one of which is that it is becoming too attenuated through culture and modern preparations.

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.

References

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