Case Report

Tuberculosis of the spine (Pott's disease) presenting as 'compression fractures'

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Study design: Case reports and survey of literature.

Objective: Case reports of two women with tuberculosis (TB) of the spine (Pott's disease) presenting with severe back pain and diagnosed as compression fracture are described. Physicians should include Pott's disease in the differential diagnosis when patients present with severe back pain and evidence of vertebral collapse.

Setting: Ohio, USA

Methods: A review of the literature on the pathogenesis, pathophysiology, clinical presentation, diagnostic methods, treatment and prognosis of spinal TB was conducted.

Results: After initial delay, proper diagnosis of spinal TB was made in our patients. Microbiologic diagnosis confirmed *M. tuberculosis*, and appropriate medical treatment was initiated.

Conclusions: Although uncommon, spinal TB still occurs in patients from developed countries, such as the US and Europe. Back pain is an important symptom. Vertebral collapse from TB may be misinterpreted as 'compression fractures' especially in elderly women. Magnetic resonance imaging scan (MRI) is an excellent procedure for the diagnosis of TB spine. However, microbiologic diagnosis is essential. *Mycobacterium tuberculosis* may be cultured from other sites. Otherwise, biopsy of the spine lesion should be done for pathologic diagnosis, culture and stain for *M. tuberculosis*. Clinicians should consider Pott's disease in the differential diagnosis of patients with back pain and destructive vertebral lesions. Proper diagnosis and anti-tuberculosis treatment with or without surgery will result in cure.

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Introduction

Tuberculosis (TB) is an uncommon disease in the US and the developed world. Extrapulmonary TB may involve any organ systems and signs are non-specific. The presenting symptom of spinal TB (Pott's disease) is usually back pain. When roentgenogram of the spine shows vertebral collapse, it may be mistaken for compression fractures, delaying the true diagnosis of Pott's disease. We report herein two women who had spinal TB, initially treated as compression fractures.

Case reports

Case One

A 73-year-old woman was admitted to another hospital for treatment of congestive heart failure and compression fractures of the spine. She was also found to have a fever with no obvious source. She was referred for work-up because of weakness, malaise, fever, and sweating for several weeks duration. Other symptoms included non-productive cough and backache. She had a history of diabetes mellitus, myocardial infarction, post percutaneous transluminal coronary angioplasty (PTCA), mitral and aortic regurgitation, congestive heart failure and chronic obstructive pulmonary disease (COPD). She had a history of 40 pack years of smoking.

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On examination the temperature was 102°F. There was no lymphadenopathy. Rales were noted in right upper lung field, and the heart rate was regular, with a soft precordial systolic murmur. The abdomen was soft, with no organomegaly or tenderness noted, and pedal edema was absent. Neurological examination revealed no focal motor weakness. The reflexes were equal bilaterally at 2+, and Babinski's reflex was absent. She had mild kyphosis.

Complete blood counts, electrolytes, BUN, creatinine, albumin were normal. The alkaline phosphatase was 36 U/L. The alanine aminotransferase was 140 U/L, aspartate aminotransferase 68 U/L. Erythrocyte sedimentation rate (ESR) was 82 mm/h. Urinanalysis was normal. Serum electrophoresis showed mild increase in IgG, normal IgA. PPD skin test was negative. Two blood cultures had no growth. Sputum was negative for malignant cells and acid-fast bacilli (AFB). Computed tomographic (CT) scan of the chest showed pulmonary infiltrates and fibrosis in the right lung. Abdominal CT scan was normal. Roentgenogram of the thoracic spine showed marked kyphosis of the thoracic spine. Collapse of the mid-thoracic vertebral bodies with marked erosive changes involving the anterior and inferior portions were noted (Figure 1). Vertebral lesion biopsy showed caseating granulomas with multinucleated giant cells. Stain for AFB was positive. Anti-tuberculosis treatment was started with isoniazid (INH), pyrazinamide, and rifampin. The patient underwent bronchoscopy; bronchial washings subsequently grew *M. tuberculosis* susceptible to all drugs tested. The patient was discharged and sent to a rehabilitation hospital and later received 12 months of anti-tuberculosis treatment. The patient did well over the next 4 years of follow-up.
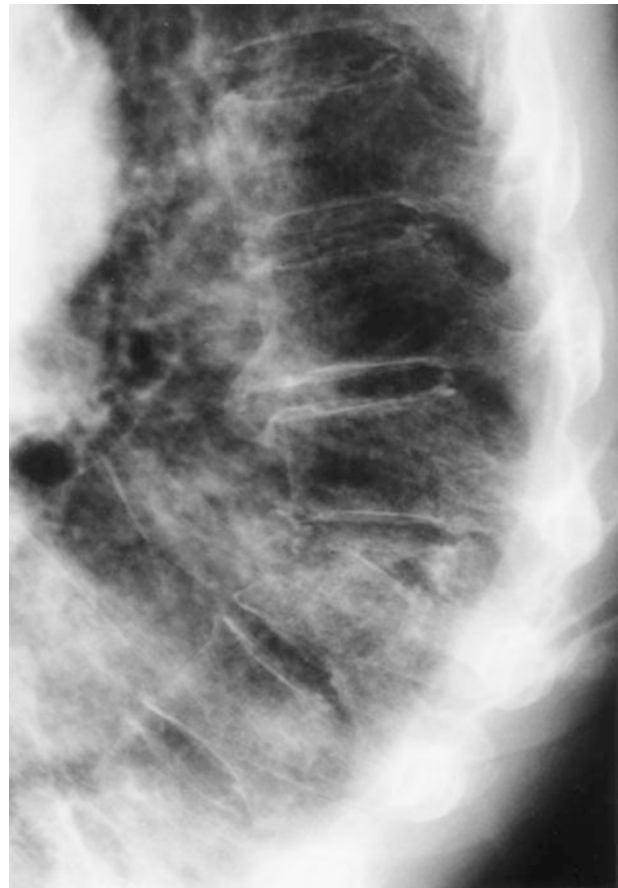
Case Two

A 54-year-old Filipino immigrant was found positive during a PPD skin test administered during her routine medical check-up. Chest radiograph was unremarkable. INH prophylaxis was initiated. Three months later, the patient complained of back pain; no neurological deficit was noted. Subsequently she developed severe weakness of the right leg and difficulty ambulating. She denied fever, rigors, chills, cough and hemoptysis. She was referred to a hospital for management of progressive neurological deficit and persistent back pain. She was found to have compression fractures of T12-L1 and underwent insertion of Harrington rod.

Lesion biopsy of the disc between T12-L1 and bone were negative for AFB by flourochrome stain; gram stain and routine culture were also negative. After the operation she had some weakness in the right lower extremity and some improvement in the pain. She was discharged to a rehabilitation hospital for spinal cord rehabilitation.

On examination, the patient was afebrile. She had good strength in both the upper and left lower extremities, but the right lower extremity showed three

Figure 1 Case one. Roentgenogram of dorsal spines showed marked kyphosis of thoracic spine, collapse of the mild thoracic vertebral bodies with marked erosive changes involving anterior and inferior portion of vertebral body.

over five strength in hip and knee flexion and extension, with four over five in ankle flexion and extension. The lungs were clinically clear, and cardiac examination was normal. Lumbosacral radiography revealed Harrington rod placed at T12 to L3 levels with apparent fusion of bodies of T12 to L2 levels. Bodies of L1-2 were poorly visualized due to superimposed metallic structures. Chest film showed cardiomegaly with no infiltrates. During her stay in the rehabilitation hospital, the patient continued to have severe pain, despite being given adequate doses of narcotics and muscle relaxant.

Because of strong clinical suspicion and continued patient deterioration, repeat biopsy of the lesion between the T12-L1 vertebral bodies was performed; the specimen grew *M. tuberculosis*. She was started on a daily regimen of INH, rifampin, pyrazinamide, and ethambutol. The patient's back pain gradually improved. Ethambutol was discontinued when testing revealed that the organism was susceptible to INH, rifampin, and pyrazinamide. She successfully completed 12 months of anti-tuberculosis treatment with

resolution of her symptoms. She was well during the 4 year follow-up interval.

Discussion

TB of the spine is an ancient disease. In 1782, Sir Percival Pott described spinal TB and surgical treatment of paravertebral abscess. Hence, spinal TB was called 'Pott's Disease'. Spinal TB accounts for 50% of the cases of skeletal TB, 15% of the cases of extrapulmonary TB and 2% of all cases of TB.[1-3] Spinal TB is on the decline in the United States and other developed countries. Most cases of spinal TB in developed countries are seen primarily in immigrants from Africa and Southeast Asia, as noted in our second case. In a study done in London between 1985–1992, 95% of cases of spinal tuberculosis were found in immigrants.[4]

Nowadays, spinal TB is a disease of children in developing nations and the elderly in the United States and Europe.[5] While the HIV pandemic has led to a resurgence of tuberculosis, it has had little impact on the epidemiology of spinal tuberculosis. In a large French study, none of 82 cases of spinal tuberculosis were HIV infected.[6] In other large longitudinal studies among HIV infected patients, few cases of spinal tuberculosis were reported, even after long term follow-up.[7,8] A study conducted in a New York City hospital between 1985–1995 described 26 patients with spinal tuberulosis. Twenty-seven per cent of them were HIV positive.[9]

In a series of patients from the southern United States, identified between 1952 and 1972, the average age of patients with spinal TB was 51 years.[10] In 1966, Friedman noted that the majority of patients in his series from Cleveland, Ohio were over the age of 40 years.[11] The lower thoracic and thoracolumbar spine were the most common areas involved, comprising 48% to 67% of lesions.[10-12]

The initial route of entry of *M. tuberculosis* is usually the respiratory tract, followed by hematogenous dissemination. Secondary hematogenous seeding can occur from a silent focus elsewhere in the body (eg gut, kidney, and tonsil). Another mode of *M. tuberculosis* spread to the vertebral bodies is from involved contiguous para-aortic lymph nodes. Vertebral involvement may be due to hematogenous dissemination via intercostal lumbar arteries. Batson's plexus, which surrounds the vertebral column, has also been implicated in those cases in which an alternating pattern of vertebral involvement has been described.

The tubercle bacillus begins its destruction in cancellous bone and eventually extends to the cortex. The infection gradually spreads to adjacent vertebra via the disc space. In advanced stages of the disease, progressive vertebral collapse occurs, resulting in kyphosis and gibbus formation.

In spinal TB, onset of symptoms is usually insidious and disease progression slow. The usual presentation consists of pain overlying the affected vertebrae, low-grade fevers, chills, weight loss, and nonspecific constitutional symptoms of varying duration. Paraplegia can be the first sign of spinal disease. Varying degrees of weakness, nerve-root compression and sensory involvement can occur. Duration of symptoms prior to diagnosis ranges from 2 weeks to several years. Historically, this interval was at least 12 months on average, decreasing to between 3 and 6 months in the recent era.[5] Weight loss has been recorded in 58% of patients,[13] and 90% to 100% of patients had back pain.[14,15] Neurological involvement has varied in different studies from 32% to 76% with notable differences in severity.[15-17]

Recognition of spinal TB in developed countries can be difficult because it is uncommon. It presents in a similar fashion to malignant deposits in the spine, which are encountered more frequently. The patient usually presents with pain, systemic symptoms like weight loss and raised ESR. This presentation is similar to cases of spinal metastasis.

Other conditions, which should be differentiated from TB of the spine include pyogenic and fungal osteomyelitis, multiple myeloma and eosinophilic granuloma. In our patients, spinal TB was initially mistaken for compression fractures.

A positive PPD skin test has been reported in 62–100% of TB spine cases.[14,17] Even though a positive tuberculin skin test supports the diagnosis, a negative test should not be considered as evidence excluding tuberculosis infection. In patients where there is a high index of clinical suspicion or evidence of impaired cellular immunity, an allergy panel should be performed.

A confident radiological diagnosis is made in less than half of the cases, with the principal alternative diagnosis being malignant disease. The relative merits of different imaging procedures like plain radiography, CT and MRI in the diagnosis of spinal TB have been evaluated. Most of the studies have failed to describe how much information is provided by each test. Conventional radiographs give a good overview; CT visualizes the disco-vertebral lesions and paravertebral abscesses, while MRI is useful in determining the spread of the disease to the soft tissues and spinal canal.[18] The plain radiograph described changes consistent with TB spine in 91–99% of cases.[13,14,16] Radiographs may reveal advanced lesions with vertebral osteolysis and disc space narrowing; these findings are similar in patients with pyogenic TB. However, absence of relative sclerosis is suggestive of TB. Paraspinal abscesses responsible for soft tissue swelling can be visible on plain radiograph. The presence of calcification within the abscess is virtually diagnostic of spinal TB. Several studies have also reported atypical presentations of spinal tuberculosis on radiography, including disease confined to only one vertebra or disease confined to the neural arch with complete sparing of adjacent vertebrae and intervertebral disc.[19] The other unusual site of involvement has been destruction of lateral aspects of vertebral bodies along with the neural arch. This rare

form of disease is associated with rapid onset of paraplegia and has only been reported in one of 123 patients with spinal TB.[20,21] CT scan is of immense value in the diagnosis of spinal TB, as it demonstrates abnormalities earlier than plain radiography. The pattern of bone destruction may be fragmentary in 47% of the cases, osteolytic in 34%, localized and sclerotic in 10%, and subperiosteal in 30%.[15] Other suggestive findings include soft tissue involvement and paraspinal tissue abscess. CT scan is also of great value in the demonstration of any calcification within the abscess or visualizing epidural lesions containing bone fragments. It is ideal for guiding a percutaneous diagnostic needle in potentially hazardous or relatively inaccessible sites.[16,22]

The best diagnostic modality for spinal TB is MRI.[13,18,22,23] MRI is more sensitive than radiography and more specific than CT in the diagnosis of spinal TB. The anatomical pattern revealed by MRI, particularly the soft tissue and disc involvement, yields greater specificity. MRI can also provide the diagnosis of TB of the spine 4–6 months earlier than conventional methods, offering the benefits of earlier detection and treatment.[23] MRI allows for the rapid determination of the mechanism for neurologic compression and can distingush between bone and soft tissue lesion (tuberculoma).

As radiological appearances are commonly non-diagnostic and imaging studies are not fully reliable for differentiating spinal TB from other infections or neoplasm, bacteriologic and/or histologic confirmation must be obtained.[24] Microbiological diagnosis is virtually impossible without aspiration of pus or some form of tissue sampling, as few patients have concomitant renal or pulmonary disease yielding diagnostic urine or sputum. However, collection of a spinal or paraspinal specimen is not absolutely necessary if pulmonary or lymph node TB is present or if extraskeletal sites can be sampled. The diagnosis in our first patient was made by histology and AFB smear of the vertebral body lesion biopsy and culture of bronchial washings growing *M. tuberculosis*. Fine needle aspiration of vertebrae for cytologic, histologic and bacteriologic studies has been recommended. Fine needle aspiration biopsy as a diagnostic tool is accurate, safe, and cost effective because the procedure does not require hospitalization. Fine needle aspiration biopsy done under CT-guidance was successful in diagnosing spinal tuberculosis in 34 out of 38 patients; in the remaining four patients with a previous history of anti-tubercular treatment, acid-fast bacilli could not be isolated.[25] Histologic studies were confirmatory of TB in 59–76% of the cases.[5,26]

Although TB of the spine is an ancient disease, treatment of this disease has undergone major changes since 1950. Treatment of spinal TB with anti-tuberculosis medication is effective in about 90% of the cases. In some cases treatment may be combined with surgery.[26,27] Skeletal TB is treated for 12 months (INH, rifampin and pyrazinamide for 2 months

followed by INH and rifampin for 10 more months). There is substantial impact of patient compliance on treatment success; directly observed therapy (DOT) is recommended. Surgery, which was once the mainstay treatment for spinal TB, is required less frequently, even in patients with cord compression. Chemotherapy alone has also been reported as a successful treatment modality.[28]

Excellent results with treatment of TB spine can be achieved if early diagnosis is made. Increasing back pain should suggest plain radiography of the spine, and perhaps followed by MRI. Our patients present with persistent back pain. Spinal radiographic findings were misinterpreted as compression fractures. The diagnosis of TB spine was delayed. Though spinal TB is uncommon in this country, sporadic cases do occur. Differentiating TB of the spine from osteo-porotic compression fractures of the spine, especially in postmenopausal white women, and from spinal cord involvement in malignancies is important. Timely treatment of spinal TB can avoid extensive investiga-tions, treatment delays and adverse long-term out-comes, including compression fractures with neurological deficits. Treatment of compression frac-tures in spinal TB with functional deficits is similar to compression fractures of the spine due to any other cause, requiring long-term rehabilitation of the patient. In patients with spinal TB pain can be severe. Various measures to alleviate pain during rehabilita-tion include appropriate spinal bracing, miacalcin (may benefit due to its analgesic effect) and narcotic analgesics.

References

1 Fancourt GJ, Ebden P, Garner P. Bone tuberculosis: results and experience in Leicestershire. *Br J Dis Chest* 1986; **80:** 265–272.
2 Wolfgang GL. Tuberculosis Joint Infection. *Clin Orthop Rel Res* 1978; **136:** 257–263.
3 Davies PD, Humphries MJ, Byfied SP. Bone and Joint Tuberculosis. A survey of notifications in England and Wales. *J Bone Joint Surg (Br)* 1984; **66:** 326–330.

4 Hayes AJ, Choksey M, Barnes N, Sparrow OCE. Spinal tuberculosis in developed countries; difficulties in diagnosis. *J R Coll Surg Edinb* 1996; **41:** 192–196.

5 Janssens J-P, De Haller R. Spinal tuberculosis in a developed country. A review of 26 cases with special emphasis on abscesses and neurologic complications. *Clin Orthop* 1990; **257:** 67–75.

6 Cotton A, Flipo RM, Drouot MH. Spinal tuberculosis: Study of the radiological aspects of 82 cases. *J Radiol* 1996; **77:** 419–426.

7 Munoz Fernandez S, Cardenal A, Balsa A. Rheumatic manifestations in 556 patients with human immunodeficiency virus infection. *Semin Arthritis Rheum* 1991; **21:** 30–39.

8 Vassilopoulos D, Chalasani P, Jurado RL. Musculoskeletal infections in patients with human immunodeficiency. *Virus Infection Medicine (Baltimore)* 1997; **76:** 284–294.

9 Leibert E, Schluger NW, Bonk S, Rom WN. Spinal tuberculosis in patients with human immunodeficiency virus infection: clinical presentation, therapy and outcome. *Tuber Lung Dis* 1996; **77:** 329–334.

10 Brashear HR, Rendleman DA. Pott's paraplegia. *South Med J* 1978; **71:** 1379.

11 Friedman B. Chemotherapy of tuberculosis of the spine. *J Bone Joint Surg (Am),* 1996; **48:** 451–474.

12 Dobson J. Tuberculosis of the spine. An analysis of the results of conservative treatment factors influencing prognosis. *J Bone Joint Surg (Br)* 1951; **33:** 517–531.

13 Pertuiset E, Johann B, Liote F. Spinal tuberculosis in adults. A study of 103 cases in a developed country, 1980–1994. *Medicine* 1999; **78:** 309–320.

14 Azzam NI, Tammawy M. Tuberculous spondylitis in adults: diagnosis and treatment. *Br J Neurosurg* 1988; **2:** 85–91.

15 Perronne C, Saba J, Behloul Z. Pyogenic and tuberculosis spondylodiskitis (vertebral osteomylitis) in 80 adult patients. *Clin Infect Dis* 1994; **19:** 746–750.

16 Jain R, Sawhney S, Berry M. Computed tomography of tuberculosis: patterns of bone destruction. *Clin Radiol* 1993; **47:** 196–199.

17 Nussbaum ES, Rockwold GL, Bergman TA. Spinal tuberculosis: A diagnostic and management challenge. *J Neurosurg* 1995; **83:** 243–247.

18 Lindhal S, Nymann RS, Brismar J. Imaging of tuberculosis. IV. Spinal manifestations in 63 patients. *Acta Radiol* 1996; **37:** 506–511.

19 Naim-ur-Rahman. Atypical forms of spinal tuberculosis. *J Bone Joint Surg* 1980; **62B:** 162–165.

20 Monaghan D, Gupta A, Barrington NA. Case report: tuberculosis of spine–an unusual presentation. *Clin Radiol* 1991; **43:** 360–362.

21 Weaver P, Lifeso RM. The radiological diagnosis of tuberculosis of adult spine. *Skel Radiol* 1984; **12:** 178–186.

22 Ridley N, Shaikh MI, Remedios D. Radiology of skeletal tuberculosis. *Orthopedics* 1998; **21:** 1213–1220.

23 Desai SS. Early diagnosis of spinal tuberculosis by MRI. *J Bone Joint Surg (Br)* 1994; **76:** 863–869.

24 Ahmadi J, Bajaj A, Destian S. Spinal tuberculosis: atypical observations at MR imaging. *Radiology* 1993; **189:** 489–493.

25 Mondal A. Cytological diagnosis of vertebral tuberculosis with fine-needle aspiration biopsy. *J Bone Joint Surg* 1994; **76-A:** 181–183.

26 Rezai AR, Lee M, Cooper PR. Modern management of spinal tuberculosis. *Neurosurgery* 1995; **36:** 87–97.

27 Moon M-S. Tuberculosis of the spine. Controversies and a new challenge. *Spine* 1997; **22:** 30–39.

28 Tuli SM. Results of treatment of spinal tuberculosis by 'middlepath regime'. *J Bone Joint Surg (Br)* 1975; **57:** 13–23.