Spinal tuberculosis: a diagnostic and management challenge

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Until the past decade, the incidence of tuberculosis in the United States had declined steadily for over 40 years; however, since 1985 a resurgence of this disease has occurred, and prompt recognition and treatment of its protean manifestations have become important. Spinal tuberculosis, traditionally referred to as Pott’s disease, occurs in fewer than 1% of patients with tuberculosis. The insidious nature of this infection, its gradual progression, and its varied clinical presentations often result in delayed diagnosis. The frequent absence of concurrent pulmonary involvement and a general lack of familiarity with the disease, especially in developed countries, heightens the diagnostic challenge. The fact that spinal tuberculosis is a potentially curable illness, which may be increasing in frequency, prompted us to review our experience with this disease over the past 20 years.

Clinical Material and Methods

Patient Population

A retrospective review identified 29 patients with spinal tuberculosis who were treated at the University of Minnesota Hospital and Clinic and the Hennepin County Medical Center from 1973 to 1993. Nine cases were treated during the first 10 years (1973–1983), and 20 cases were evaluated during the second 10-year period (1984–1993). There were 12 males and 17 females ranging in age from 2 months to 79 years. Twelve were Caucasians; eight were Native Americans; and nine were immigrants from Korea, Vietnam, or Laos. Hospital records, neuroimaging studies, operative reports, and follow-up clinic notes were available in all cases. No patient was lost to follow-up review. One patient died within 1 month of treatment. Excluding this case, the average length of follow up was 7.4 years (range 1 to 20 years).

Key Words: spinal tuberculosis • Pott’s disease • tuberculous spondylitis • osteomyelitis
Results

Clinical Features and Diagnosis

At the time of presentation, clinical findings, in descending order of frequency, included back pain, paraparesis, kyphosis, fever, sensory disturbance, and bowel and bladder dysfunction (Table 1). Twenty-two patients (76%) presented with neurological deficit; 12 (41%) were initially misdiagnosed. In 14 cases (48%), spinal infection was the initial manifestation of disease. Fifteen individuals (52%) had been diagnosed previously with extraspinal tuberculosis and three (10%) had concurrent pulmonary involvement. Five patients (17%) had a known family history of tuberculosis. A tuberculin skin test was positive in 18 cases (62%). The erythrocyte sedimentation rate was measured in 12 individuals and was uniformly elevated. We encountered six patients (21%) who had no previous or concurrent diagnosis of extraspinal tuberculosis, no family history of tuberculosis, and a negative tuberculin skin test.

Radiographic Examination

Diagnostic imaging studies included plain roentgeno-

![Fig. 1.](image)

**FIG. 1.** Left: Midsagittal, T2-weighted magnetic resonance image demonstrating marked collapse of the L-1 vertebral body with kyphotic angulation, effacement of the epidural space, and compression of the thecal sac. Right: Axial computerized tomography scan in the same patient confirming severe disruption of the vertebral body with paraspinal soft-tissue involvement.

![Fig. 2.](image)

**FIG. 2.** Upper: Thoracic myelogram obtained for evaluation of progressive paraparesis and sensory disturbance revealing a mass lesion blocking the flow of contrast material, with a relative absence of bone destruction. The patient responded well to laminectomy and debridement of a granulomatous epidural abscess. Lower: Computerized tomography with intrathecal contrast in another patient demonstrating the presence of a ventral epidural mass displacing the thecal sac with preservation of the bony architecture of the vertebral body. A tuberculous epidural abscess was encountered at the time of surgery.
Spinal tuberculosis

![Image](559x306 to 753x544)

Fig. 3. Midsagittal, T₁-weighted magnetic resonance (MR) images. Left: Image revealing expansion of the spinal cord at the T12–L1 level consistent with an intramedullary mass lesion. Right: Comparable gadolinium-enhanced image in the same patient showing a ring-enhancing lesion within the spinal cord. With no history of infection, this was preoperatively misinterpreted as a neoplasm.

grams in all cases (100%), computerized tomography (CT) scans in 22 (76%), myelogram with CT scan in 12 (41%), and magnetic resonance (MR) imaging in eight (28%). Chest x-ray films were obtained in all cases and revealed evidence of active or healed pulmonary disease in 10 (34%). Plain roentgenograms revealed endplate disruption and bone destruction, CT delineated bone involvement and paravertebral abscess extension, intrathecal contrast administration showed the degree of thecal sac compression, and MR imaging demonstrated intramedullary lesions and the extent of soft-tissue involvement. The focus of infection was cervicothoracic in one case (3%), thoracic in 16 (55%), thoracolumbar in three (10%), and lumbosacral in eight (28%). One patient had noncontiguous lesions in the cervical and thoracic regions. In 13 cases (45%), associated paravertebral abscesses were present.

Sixteen individuals (55%) had predominant vertebral body involvement; nine had marked bone collapse with neurological compromise (Fig. 1). Eleven patients (38%) had intraspinal granulomatous tissue causing neurological dysfunction in the absence of bone destruction (Fig. 2). The focus of infection was an epidural “abscess” in nine cases and a subdural “empyema” in two. Two patients (7%) presented with intramedullary tuberculosis (one thoracic, one lumbar); one had widely disseminated tuberculosis, the other had no history of prior or concurrent mycobacterial infection (Fig. 3). One patient with a marked cervicothoracic kyphosis presented with progressive neurological deficit and a large syrinx, which was demonstrated on MR imaging. The radiographic and operative findings in these patients are summarized in Table 2.

**Treatment and Outcome**

All patients received varying combinations of the anti-

tuberculous medications isoniazid, rifampin, ethambutol, streptomycin, and pyrazinamide for at least 3 months. Courses of antibiotic therapy shorter than 6 months were inevitably associated with disease recurrence. Needle biopsy procedures were performed in 12 cases and were diagnostic in five instances (42%). Culture of material obtained at the time of an open procedure was diagnostic in 17 (68%) of 25 cases. In five cases, the diagnosis of tuberculosis was established presumptively based on the clinical setting and culture results from other sites of infection or from sputum samples.

Seven patients who were neurologically normal and had only minimal bone involvement were initially treated with external bracing after diagnostic biopsy. Four responded well, but three later required surgery for inadequately treated osteomyelitis. Six patients with minimal neurological deficit and bone destruction were initially managed by external bracing, but all eventually required surgery for worsening neurological function and increasing bone involvement. Therefore, of 13 patients initially treated with biopsy and bracing alone, nine (69%) eventually required fusion procedures. Progression of disease despite bracing was associated with the presence of vertebral body destruction, greater than 50% loss of body height, and wedge compression with kyphotic deformity.

Eight patients underwent laminectomy and debride-

ment of extra- or intradural granulomatous tissue; one (13%) later required fusion. Eleven patients were treated with isolated anterior or posterior fusion; five (45%) later required combined anterior–posterior procedures. Anterior fusion failure was associated with marked preoperative kyphosis and multilevel disease requiring a graft that spanned more than two disc spaces. Three patients initially treated with combined procedures did not require further surgery. The majority of patients with severe bone destruction had associated intraspinal granulomatous tissue that required extensive debridement at the time of surgery. Two patients underwent microsurgical excision of an intramedullary tuberculoma with intraoperative ultrasound guidance. One recovered normal neurological function; the other was left with a spastic paraparesis.

Thirteen patients were readmitted 2 months to 14 years after initial diagnosis, with progression of inadequately treated osteomyelitis; 12 (92%) required fusion procedures. One patient with a residual cervicothoracic kyphosis after posterior spinal fusion returned 7 years later with progressive neurological deficit and a large syrinx. He

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<td><strong>Radiographic and operative findings in 29 patients with spinal tuberculosis</strong></td>
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<tr>
<td>Focal of Infection</td>
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<tr>
<td>vertebral destruction*</td>
</tr>
<tr>
<td>intraspinal granulomatous “abscess”†</td>
</tr>
<tr>
<td>epidural</td>
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*Patients with bone destruction typically had extensive intraspinal granulomatous tissue associated with osteomyelitis. † Intraspinal tissue producing mass effect with relative absence of bone involvement.
was initially treated with a syringosubarachnoid shunt but eventually underwent an anterior kyphectomy with strut grafting to optimally decompress the spinal cord. On follow-up examination, 17 patients (59%) had mild or no deficit, 10 (34%) had significant deficit, and 2 (7%) were dead.

Discussion

It has been estimated that there are approximately 27,000 cases of tuberculosis reported annually in the United States.6,26 The recent increase in the incidence of tuberculosis may be related to a growing number of immunocompromised patients and a rise in the number of immigrants from areas where tuberculosis remains endemic.2,3,6,7,26–28 In the present series, two-thirds of the patients were encountered after 1983, suggesting that the frequency of spinal tuberculosis may be increasing as well. Tuberculosis spondylitis is uncommon but accounts for up to 60% of all bone and joint tuberculosis and typically affects contiguous vertebral bodies and their intervening disc spaces.11–14,17 There is an apparent predilection for the anterior vertebral body although the posterior elements may be involved as well.11,12,18,30 The thoracic and lumbar regions are most often affected, and paraspinal soft-tissue involvement is common.4,11,12

Diagnostic Considerations

Because the consequences of a delay in diagnosis may include irreversible neurological deficit and even death, it is incumbent on the physician to become familiar with the varied manifestations of this infection. Based on clinical and radiographic findings, tuberculosis spondylitis may be impossible to differentiate from pyogenic or fungal vertebral osteomyelitis and from primary or metastatic tumors including myeloma and lymphoma.16,33,34 A history of tuberculosis, a positive skin test, and an elevated erythrocyte sedimentation rate are useful diagnostic clues, but these are not uniformly present.3,10,11,17 Although diagnosis is often complicated by difficulties associated with culturing these fastidious organisms, the use of DNA amplification techniques may allow for more rapid and accurate identification of tuberculosis infections in the future.5

Radiographic changes associated with tuberculous spondylitis include rarefaction of the vertebral endplates, disc-space narrowing, anterior wedging, and bone destruction, but early findings may not be visible on plain roentgenograms for up to 8 weeks.1 Although significant bone destruction will be evident on plain roentgenogram or CT scan, epidural granulomatous tissue or tuberculosis of the spinal cord may not be detected by these imaging studies. Therefore, in the face of neurological deficit, the diagnostic procedures of choice are myelogram followed by CT or MR imaging. Postmyelogram CT will reveal thecal sac compression and has the added advantage of detailing the degree of bone involvement. Magnetic resonance imaging is noninvasive and will demonstrate intramedullary or extramedullary lesions as well as soft-tissue infection.

Treatment Options

The optimum treatment of spinal tuberculosis remains controversial and should be individualized in each case. Patients who are neurologically intact without significant bone destruction are generally treated with biopsy, antituberculous medications, and external bracing.11 Based on present guidelines, it is recommended that antituberculous chemotherapy, consisting of at least two medications, be administered for a period of at least 12 months.11,13,23 The addition of a third agent for the initial 2 to 6 months is also advised. Surgical intervention may be indicated for diagnostic biopsy, drainage of a large paraspinal abscess, decompression of neural elements, correction of spinal deformity, and/or stabilization of the spine.

In the majority of our cases, the presence of neurological deficit resulted from either marked bone collapse with spinal canal compromise (true tuberculous spondylitis) or a granulomatous epidural or subdural “abscess” producing mass effect without significant bone involvement. Because both epidural infection and bone destruction typically progress for a variable length of time after antituberculous chemotherapy is instituted, the presence of even mild neurological deficit may be taken as a strong indication for surgical intervention.4,11,12

It has been our experience that patients with epidural or subdural granulomatous tissue in the absence of significant bone collapse may be treated satisfactorily with simple laminectomy and debridement. However, when vertebral body involvement is significant enough to produce wedging and kyphosis, an aggressive debridement and fusion procedure is indicated. In the present series, 12 of 13 such patients who were initially managed nonsurgically later required fusion procedures for progression of inadequately treated osteomyelitis. Large controlled trials have examined the efficacy of various treatment regimens for spinal tuberculosis, confirming the advantage of surgical intervention combined with chemotherapy in such instances.20,33 As noted in prior reports, anterior fusion failure was associated with marked preoperative kyphosis and multilevel disease requiring a graft that spanned more than two disc spaces. Therefore, it is suggested that these patients may be treated best by combined anterior–posterior fusion to prevent delayed instability.22,24

It is worthwhile to note that our series includes two of only four cases of intramedullary tuberculomas reported in North America since 1960.7,21 These lesions are exceedingly rare, being present in only two of every 100,000 cases of tuberculosis.5,18 The diagnostic procedure of choice is MR imaging, which reveals a ring-enhancing, hypo- to isointense lesion on T1-weighted images and a hypointense area with a variable central hyperintensity (reflecting caseating necrosis) on T2-weighted images.15 Although intramedullary tuberculomas often respond to chemotherapy alone, some patients have progressive neurological deficits despite treatment, possibly due to lesion swelling as the bacilli release irritative material in response to treatment.6 In cases in which the patient is deteriorating or when the diagnosis is uncertain, microsurgical excision with intraoperative ultrasound guidance is recommended.

Conclusions

As the incidence of tuberculosis in the United States increases, spinal involvement of this disease will undoubt-
Spinal tuberculosis
dedly become more frequent. We reviewed 29 cases of spi-
nal tuberculosis treated over a 20-year period. In our expe-
rience, most patients fell into two categories: those with 
granulomatous epidural “abscess” who responded well to 
simple decompression and debriodement, and those with 
significant bone destruction (true tuberculous spondylitis) 
who required definitive fusion procedures to prevent pro-
gression of disease. With aggressive treatment, a satisfac-
tory outcome can be obtained in the majority of cases.

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