REVIEW

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Spinal tuberculosis deserves a place on the radar screen

ABSTRACT

Spinal involvement may be the first manifestation of tuberculosis and the problem that drives the patient to seek medical care. Spinal tuberculosis (often called Pott's disease) is by definition advanced disease, requiring meticulous assessment and aggressive systemic therapy. Physicians should keep the diagnosis in mind, especially in a patient from a group with a high rate of tuberculosis infection.

KEY POINTS

In the United States, approximately 20% of patients with tuberculosis have extrapulmonary disease. Ten percent of those with extrapulmonary tuberculosis have skeletal involvement, and half of these have spinal infections.

Symptoms of spinal tuberculosis are back pain, weakness, weight loss, fevers, fatigue, and malaise.

Magnetic resonance imaging can define the extent of abscess formation and spinal cord compression. The diagnosis is confirmed through percutaneous or open biopsy of the spinal lesion.

Surgery is necessary as an adjunct to antibiotic therapy if the vertebral infection produces an abscess, vertebral collapse, or neurologic compression. Some patients need aggressive supportive care owing to tuberculous meningitis or encephalopathy.

IN APRIL, A HEALTHY 48-year-old office administrator was active and happy, complaining only of a progressive, nagging backache over the past 6 months. Her only risk factor for tuberculosis was a brother who had traveled overseas a decade before.

By the end of May, she lay in intensive care, virtually blind, obtunded, and paraparetic, awaiting the second of a series of spinal operations to drain the extensive abscesses that had accumulated along the lumbar spinal column within the psoas muscles.

Over the months that she had been followed by her local physician, her only complaint was of persistent, focal midlumbar back pain, worse with activity but never completely relieved by rest. The most prominent physical finding was persistent weight loss.

Despite the long course of her back pain symptoms, the first radiographs obtained were taken when she presented to the emergency department with acute worsening of her symptoms (**FIGURE 1, FIGURE 2**).

The indolent clinical course, coupled with the severe vertebral destruction and the systemic symptoms of weight loss and fatigue, put spinal tuberculosis at the very top of the differential diagnosis.

Within 48 hours of transfer to our facility, she became obtunded and had a seizure. Cranial nerve damage became apparent. Tuberculous meningitis was diagnosed.

NO CAUSE FOR COMPLACENCY ABOUT TUBERCULOSIS

This patient's rapid deterioration startled even experienced physicians and serves as a reminder of just how dangerous extrapul-

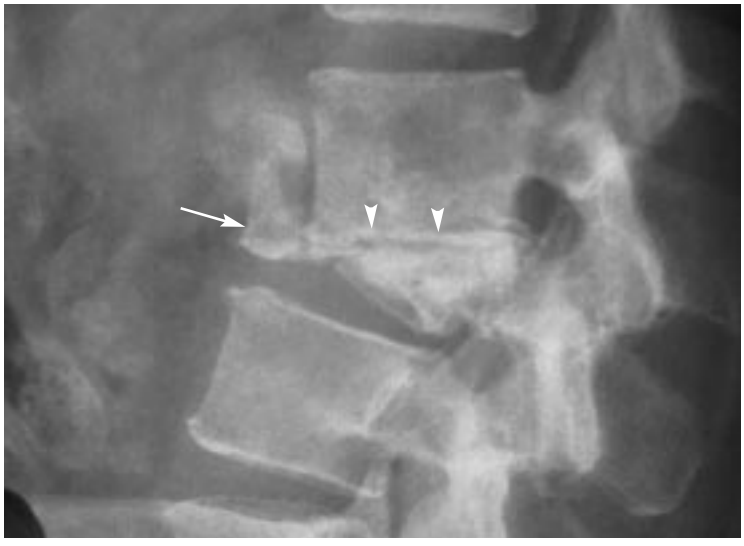

FIGURE 1. Plain radiograph of a 48-year-old woman with 3 to 4 weeks of mild and 1 week of intense low back pain. The classic Pott deformity results from destruction of the L3 vertebral body (arrow) and obliteration of the L2–3 intervertebral disc (arrowheads).

monary tuberculosis can still be. Perhaps even more alarming was that this disease could erupt in middle-class America, in a healthy person, with little warning.

Untreated, neurologic involvement may progress to complete paraplegia

In the United States, the incidence of tuberculosis infection declined for most of the 20th century, from 84 cases per 100,000 population in 1953 to 22 per 100,000 in 1984. The incidence rose in the late 1980s to a peak of 27 per 100,000 in 1992, but has since declined to 5.2 per 100,000 in 2002.[1]

There is no cause for complacency about tuberculosis, however. In developed countries, rates of active tuberculosis disease now account for a greater percent of cases of tuberculosis infection, owing to antimicrobial resistance, drug and alcohol addiction, human immunodeficiency virus (HIV) infection, and therapeutic immunosuppression.[2–4] Moreover, rates of tuberculosis infection and active disease are higher in certain groups, such as foreign-born people and non-Hispanic blacks born in the United States. These groups now account for approximately three fourths of US cases.

TUBERCULOSIS OF THE SPINE: KNOWN FOR 200 YEARS

Percival Pott first described tuberculosis of the spinal column in 1779. The classic destruction of the disc space and the adjacent vertebral bodies, collapse of the spinal elements, and severe and progressive kyphosis (FIGURE 3) subsequently became known as Pott's disease.

Skeletal involvement occurs in approximately 10% of all patients with extrapulmonary tuberculosis, and half of these patients develop infection within the spinal column.[5,6] Up to 45% of patients with spinal involvement develop corresponding neurologic deficits.[7]

In developing countries, spinal tuberculosis affects mostly children; however, adult infection has become more common in North America, the Middle East, and Europe, places where the overall incidence of the disease is lower.[8,9]

Spinal involvement is usually a result of hematogenous seeding either from a pulmonary lesion or from an infection of the genitourinary system.[10] Contiguous extension from a pulmonary abscess can result in thoracic spondylitis.

DELAY IN DIAGNOSIS IS COMMON

Because spinal tuberculosis remains uncommon in the US population as a whole, physicians are unaccustomed to entertaining the diagnosis in appropriate situations. Diagnostic delay is common, and the results can be disastrous.

The progression of spinal tuberculosis is usually slow and insidious, and its main symptom—backache—is nonspecific. Therefore, spinal tuberculosis is more difficult to recognize than pyogenic infection (eg, osteomyelitis due to staphyloccal or streptococcal organisms, which tends to be acute in presentation). Considerable delay in diagnosis may occur before an infectious process is even considered. Even when spinal tuberculosis is considered, it may be difficult to confirm.

Before the diagnosis is established, the vertebral bone and disc material may sustain extensive destruction. Progressive vertebral collapse and fracture can lead to spinal deformity and a classic Pott kyphosis. Neurologic deficits are common with long-standing thoracic and cervical involvement, and if untreated, neurologic involvement may progress to complete paraplegia.

When tuberculous meningitis is superimposed, the disease is lethal and devastating, even though effective drugs are available.[11,12]

CLINICAL PRESENTATION

The clinical presentation of spinal tuberculosis is extremely variable. The type and intensity of symptoms depends on the level of involvement, the severity of the disease, and the duration of the infection.

Patients typically present with a combination of systemic manifestations such as weight loss, fever, fatigue, and malaise, as well as focal back pain. Many patients present with relatively moderate and chronic symptoms despite severe vertebral destruction (FIGURE 1, FIGURE 2).

The pain varies from mild and constant to severe and activity-related. Pain is usually localized to the site of involvement and is most common in the thoracic spine. It can be constant and indolent, reflecting the progressive destruction of the involved disc space and vertebral elements, or it can be intense and directly linked to spinal motion and weight-bearing, which is caused by more advanced disc disruption and spinal instability, nerve root compression, or pathological fracture.[13,14]

Patients with lumbar disease may develop an anterior abscess, which may track into the psoas muscle (FIGURE 1, FIGURE 2). Patients with a "psoas sign" tend to lie with the leg drawn up in a flexed position, and they experience exquisite pain when the hip is extended to a neutral position.

An abscess within the spinal canal may compress the cord or cauda equina, and neurologic symptoms may develop rapidly. Depending on the level of involvement, a spinal abscess may cause nerve root symptoms mimicking a herniated disc, or it may produce acute and progressive spinal cord compression, resulting in paraplegia or quadriplegia if untreated.[15–17]

In rare cases, meningitis develops in association with spinal disease, but most experts feel that meningeal involvement represents hematogenous spread rather than direct infection. Meningitis is suggested by headache, photophobia, and changes in level of consciousness. More severe symptoms, including seizure, cranial nerve palsy, and cavernous sinus syndrome, are associated with a higher mortality rate and frequent neurologic sequelae.

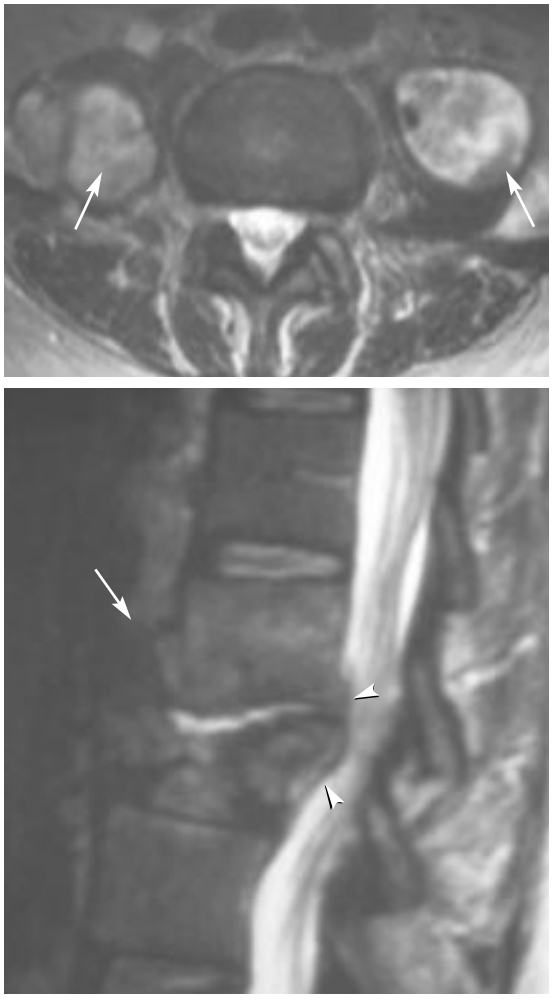

FIGURE 2. TOP, magnetic resonance imaging (MRI) in our patient demonstrates bilateral psoas abscesses, contiguous with granulomatous abscess of the vertebral body. **BOTTOM,** MRI in the sagittal view demonstrates anterior abscess (arrow), segmental instability, and epidural compression (arrowhead) due to abscess and vertebral fragments. This patient had quadriceps and dorsiflexor weakness on the left side. Before surgery could be planned she became obtunded, then manifested seizures and cranial nerve palsies with the onset of tuberculous meningitis. Despite immediate support and quadruple antibiotic therapy, the patient suffered an infectious injury to the visual cortex which rendered her blind.

Pain in the thoracic spine is most common

Tuberculosis of the spine

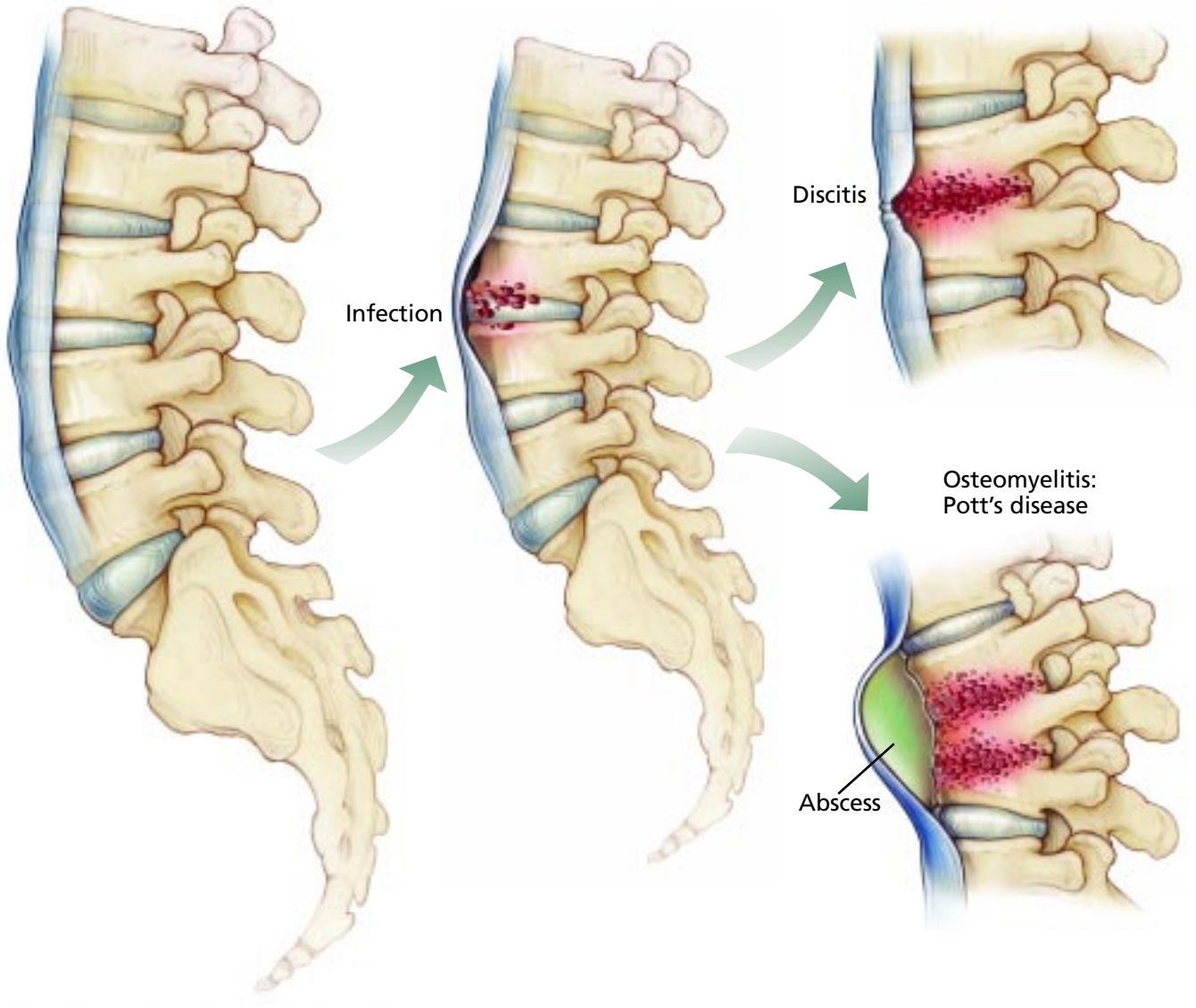

FIGURE 3. Tuberculous bacilli spread to the disc space from surrounding tissues (contiguous spread) or through the vascular supply (hematogenous spread). Over time the disc may be completely digested (discitis), or the infection may progress to involve the bone of each of the adjoining vertebral bodies (osteomyelitis). As the vertebrae degenerate and collapse, a kyphotic deformity results (Pott's disease).

CLUES FROM THE MEDICAL HISTORY

Manifestations of primary infection

The physician should seek out any evidence of primary tuberculosis infection in the recent past. Recurrent fever, chills, night sweats, or weight loss suggest systemic disease of either a pyogenic (eg, due to staphylococci, streptococci, *Haemophilus*, or *Escherichia coli*) or granulomatous nature. A more indolent course of disease progression favors a diagnosis of granulomatous disease over infection with common bacteria such as staphylococci or streptococci.

A persistent, productive cough may suggest the primary focus of infection is in the lungs; persistent urinary tract symptoms suggest the urinary tract. Shortness of breath is uncommon, but a cough that is productive of tannish, purulent, or blood-tinged sputum should raise the suspicion of tuberculosis and should be investigated via sputum cultures or bronchoscopy or both.

History of personal exposure

Some patients who emigrate from areas where tuberculosis is endemic carry chronic tuberculosis with them when they move. Among large immigrant families, one family member might carry active disease and infect other members of the family over time, owing to noncompliance with treatment or distrust of health authorities and western medicine.

Immigrants, the chronically ill, the homeless, and IV drug abusers are at higher risk of spinal tuberculosis

Similarly, patients who have recently traveled to developing countries, who have visited friends or family members from those areas, or who have a known exposure to someone with tuberculosis also are at higher risk.

Within cities, the homeless, intravenous drug abusers, alcoholics, and the chronically ill are at higher risk of contracting active tuberculosis. These people are often immunosuppressed, suffer from malnutrition, have a higher incidence of exposure to other infected individuals, and either shun medical care or fail to complete treatment when it is offered. Cumulatively, these factors increase the risk of chronic infection and of infection resistant to multiple drugs.

History of immunosuppression or immunodeficiency disease

Patients who are immunocompromised, due to either therapeutic medical suppression or HIV infection, are at high risk of developing active tuberculosis, as well as a number of other granulomatous infections otherwise rarely seen. Organ transplant recipients, patients on long-term prednisone therapy, and patients undergoing chemotherapy for cancer treatment are all at increased risk of disease.

FINDINGS ON PHYSICAL EXAMINATION

Pulmonary evaluation

The pulmonary evaluation may reveal evidence of pneumonia or simply a productive cough, from which sputum may be cultured.

Genitourinary evaluation

Evaluation of the genitourinary system should include a clean-catch urinalysis. If the patient is febrile, urine cultures for acid-fast bacilli should also be done; blood cultures may confirm a pyogenic bacterial infection but will not reveal tuberculosis as the source of fever.

Spinal examination

Examination of the spine may reveal focal tenderness over the spinous processes, with more diffuse back spasm in the region of pain. Fluctuance, redness, or focal heat are rare, as the spinal infection typically involves the anterior column of the spine.

Range-of-motion testing, on the other hand, may produce severe pain, and the patient may guard aggressively against any twisting, bending, or extension motions. Patients are usually most comfortable lying down, and they may have moderate to severe symptoms when standing upright or walking.

In advanced disease, focal kyphosis can be seen on physical examination, usually in the midthoracic to thoracolumbar spine. The sharp angulation results in prominence of the spinous processes at the level of the vertebral collapse, forcing the patient to stoop or lean forward. In severe kyphosis, the patient may not be able to stand without leaning with the hands on a walker or desk.

Neurologic symptoms

Neurologic symptoms of spinal involvement may be subtle at first, but will progress over time. Early symptoms may include numbness and tingling in the lower extremities, numbness or paresthesia in a belt-like distribution around the chest wall, or a subjective sense of weakness with activity. As the disease progresses, symptoms become more pronounced.

The level of spinal cord involvement determines the level of impairment. When cervical tuberculosis progresses and causes cord or root compression, the earliest signs are weakness, pain, and numbness of the upper and lower extremities. Progressive deformity or abscess gradually increases pressure on the cord, and symptoms eventually progress to full-blown quadriplegia.

If the thoracic or lumbar spine is involved, upper-extremity function remains normal while lower-extremity symptoms progress over time. As cord compression becomes more severe, subjective symptoms give way to objective findings of motor weakness, hyperreflexia, and reflex abnormalities, including a positive Babinski reflex (toes curl upward) and sustained clonus.

If a lumbar abscess or vertebral collapse results in compression of a single nerve root, symptoms may mimic those of a herniated disc. These patients experience radicular pain radiating over the distribution of the affected nerve root, and they may experience weakness in a specific motor distribution. Unlike a true herniated nucleus pulposus, in which symptoms typically increase with activity and decrease with rest, the radicular pain secondary to tuberculosis infection tends to be intractable and constant.

Patients with cauda equina compression due to lumbar infection have weakness, numbness, and pain, but have decreased or absent reflexes among the affected muscle groups rather than the hyperreflexia seen with spinal cord compression.

Patients with meningeal involvement may present with headache, nausea, vomiting, photophobia, or impaired consciousness, but occasionally present with acute hydrocephalus, seizures, cranial nerve palsies, or—in rare cases—cavernous sinus thrombosis.

DIAGNOSTIC IMAGING

Plain radiography

Plain anterior-posterior and lateral radiographs should be the first imaging studies ordered in any patient with chronic, progressive back pain.

In patients with tuberculous spondylitis, radiographic findings depend on the extent and duration of infection.

While pyogenic infections typically destroy the intervertebral disc before generating a pronounced osteolytic reaction in the adjacent vertebrae, granulomatous infection may produce a much more indolent and subtle diagnostic picture.

Initial radiographs may be entirely normal in tuberculous disease, but over time, disc space narrowing and end-plate reaction both become prominent. In contrast to pyogenic infection, tuberculous infection may spare the disc space entirely in up to 50% of cases, producing central body involvement that is difficult to distinguish from a tumor.[18] In these cases, central rarefaction of the vertebral body inevitably progresses to vertebral collapse and kyphosis.[19,20]

Plain radiographs are the first imaging studies for any patient with chronic, progressive back pain

Magnetic resonance imaging

Magnetic resonance imaging (MRI) is the diagnostic study of choice after plain radiography.

MRI demonstrates the relative sparing of the disc space (**FIGURE 2**) and, at the same time, involvement of the vertebral bodies on either side of the disc, a rare finding in malignant disease. Dissection of the anterior soft tissues, with abscess formation and collection and expansion of granulation tissue adjacent to the vertebral body, is highly suggestive of tuberculosis.

Epidural abscesses, compression of the nerve root, or compression of the spinal cord are also best demonstrated with MRI studies.[21–23]

CONFIRMING THE DIAGNOSIS

Whenever spinal tuberculosis is suspected, a primary workup for systemic infection is required.

Chest radiographs may show apical lesions or a Ghon complex characteristic of pul-

monary tuberculosis.

The purified protein derivative (PPD) test is sensitive for disease exposure but does not indicate active disease or reveal the degree of infectiousness. The PPD test may also be mildly positive if the patient has ever received BCG vaccine.

A definitive diagnosis is made when acid-fast tuberculous bacilli are cultured from sputum, urine, or biopsy material. While the acid-fast bacilli stain may show acid-fast organisms in the initial clinical specimen, these may not be present in every case. Tuberculous bacilli grow slowly in culture, and confirmation may not be available for 6 to 8 weeks.[24]

Polymerase chain reaction testing is exquisitely specific for tuberculous bacillus, and provides rapid confirmation of a positive culture. It is so specific, however, that it may overlook other species of *Mycobacteria*, and is only approved for use with pulmonary specimens.[25,26]

Needle biopsy

Surgery itself does not eradicate the infection, so drug therapy is key

If the clinical suspicion of spinal tuberculosis is high and if radiographic studies show a destructive lesion warranting surgical treatment, then open debridement of the lesion will provide ample material for culture and diagnosis. However, if the process is caught earlier in its development, there may be no indication for surgical intervention. In this case, needle biopsy guided by computed tomography or MRI may provide diagnostic material.

Guided by imaging, a fine needle can be introduced into the abscess cavity through the posterior muscular wall. If a fluid abscess is encountered, material can be drawn through the fine needle without difficulty. If granulation tissue is encountered, a trocar may be needed to obtain an adequate tissue specimen.

If fine needle or trocar biopsy is not adequate, percutaneous biopsy may obtain enough tissue for histologic diagnosis and culture. In the operating room under fluoroscopic guidance, a large trocar can be introduced into the collapsed vertebral body to obtain a bone specimen, or into the disc space to obtain disc material and granulation tissue for culture and diagnosis.

TUBERCULOUS MENINGITIS

A common presentation in the developing world, tuberculous meningitis is distinctly uncommon in developed nations with a low incidence of tuberculosis.[9] Recent increases in immigration and in the numbers of elderly and immunocompromised patients have, however, resulted in a measurable increase in the incidence and mortality associated with tuberculous meningitis.

If tuberculous meningitis is not diagnosed promptly, the risk of significant morbidity and mortality is higher, but the diagnosis is likely to be made quickly only if the physician has a high index of suspicion. Despite modern therapies, the mortality rate still ranges from 9.5% to 55.6%, and neurologic sequelae from 27% to 42%.[4,12,27]

DRUG THERAPY

Multidrug therapy remains the cornerstone of tuberculosis treatment, irrespective of skeletal involvement. For patients with spinal infection, the goals of treatment are to eradicate the disease and to prevent or correct neurologic deficits and spinal deformities.

First-line drugs

Patients with early disease, no neurologic deficit, and little or no kyphosis can be treated with a multidrug combination alone. Isoniazid remains the most widely used and effective antituberculous agent worldwide.[28] The combination of isoniazid, rifampin, ethambutol, and pyrazinamide can overcome organisms resistant to isoniazid alone or to other combinations of antibacterial drugs. Streptomycin is sometimes used instead of ethambutol.

Pyridoxine (vitamin B) should be given concurrently to reduce the risk of peripheral neuritis, which may occur in 2% of patients taking isoniazid.[29]

Second-line drugs

While the first-line agents isoniazid, rifampin, pyrazinamide, and ethambutol are sufficient for most patients presenting with active tuberculosis, patients in larger city centers, those immigrating from Mexico or the Pacific rim,

and those who have not complied with previous therapy may present with tuberculosis strains that are resistant to multiple drugs and require second-line agents for successful treatment. These are selected on the basis of regional patterns of sensitivity, side effect profiles, and the mechanism of action.

Second-line drugs such as cycloserine and the quinolones may also be used in patients who do not tolerate one of the first-line agents. Although multidrug treatment is now the standard approach to initial care, once definitive sensitivities are established, some of the drugs may be discontinued.

In severely ill patients with septicemia, drug therapy is with a combination of isoniazid, rifampin, streptomycin, and pyrazinamide. In the intensive care setting, additional drugs may include amikacin and quinolones.

Supportive care is crucial in stabilizing the patient until chemotherapy provides benefit. Steroids are occasionally used in patients presenting with pericarditis or meningitis.

Antibiotic therapy is typically maintained for 6 to 9 months, whether there is skeletal involvement or not. Patients who do not complete a full course of therapy are at risk for relapse and drug-resistant infection.

THE ROLE OF SURGERY

While new drugs are effective for most cases of spinal tuberculosis, surgical intervention is necessary in advanced cases with extensive bony destruction, abscess formation, or neurologic compromise.[30,31]

The goal of surgery is to prevent or correct neurologic deficits and spinal deformities. Surgery also facilitates successful chemotherapy, since the abscess cavity provides an avascular environment that protects bacilli from systemic antibiotics.

When surgery is needed, results are best earlier in the disease process, before scarring and fibrosis develop. Later, dense scarring causes adhesions to the great vessels or vital structures, making dissection and surgical exposure dangerous. The clinical response to surgery is also faster and more complete in patients with active disease compared with those with chronic disease and deformity.

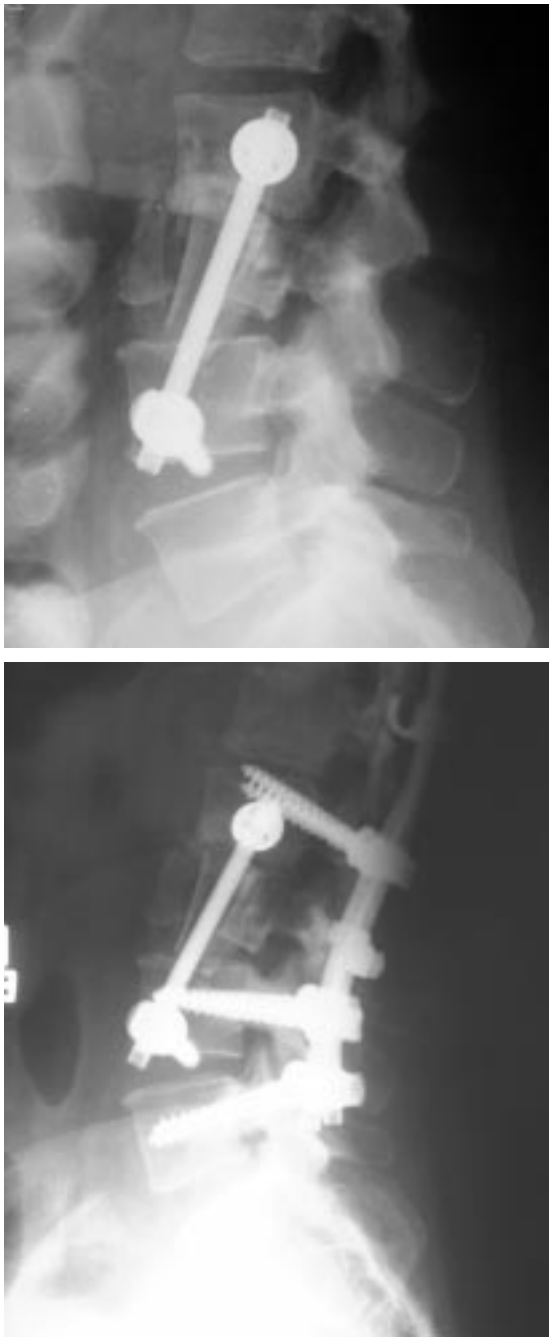

FIGURE 4. TOP, vertebrectomy and abscess drainage was necessary before chemotherapy could be successful. Excision of the infected vertebral body decompresses the cauda equina, but leaves a gap that must be filled with bone. **BOTTOM**, posterior instrumentation with longitudinal rods, combined with fixation hooks and screws, provides enough initial rigidity to permit the patient to get up and out of bed for rehabilitation.

Classic approach

The classic surgical approach to spinal tuberculosis is by anterior exposure of the spine, an operation first described by Hodgson and Stock in 1956.[32] The anterior approach—either through the chest wall or via a retroperitoneal approach to the thoracolumbar or lumbar spine—allows the surgeon to remove all infected and devitalized bone and any material created by the infection. Complete removal of diseased bone and disc is necessary to provide a solid substrate for reconstruction (FIGURE 4).

Debridement removes the entire vertebral body, along with the posterior longitudinal ligament and any epidural abscess or granulation material compressing the neural elements.

Spinal alignment is manually corrected, and a strut is placed between the remaining healthy vertebral bodies to maintain normal alignment and stability. In the past, the appropriate strut was always harvested from the patient's own iliac crest, but more recent experience has shown that allograft bone can also be used successfully.

After anterior strut graft reconstruction, a second operation is usually needed to stabilize the spine and allow early ambulation and activity. Posterior instrumentation and fusion, following the anterior decompression and reconstruction, prevent recurrent kyphosis and protect the anterior construct from collapse (FIGURE 4). The staged anterior and posterior procedures consistently restore neurologic function when deficits are incomplete, and prevent or correct kyphosis in most patients.[33]

Once the second procedure is completed, the patient can be up and out of bed immediately for ambulation and rehabilitation. Prolonged bed rest is strongly discouraged.

Surgery alone neither eradicates local disease nor treats the systemic infection. Drug therapy remains the cornerstone of successful therapy.

Complications of surgery

Complications of surgical treatment of spinal tuberculosis are similar to those associated with surgical debridement and reconstruction for other forms of infection or tumor. Depending on the patient's health and age, complications may be frequent, with an overall mortality rate of roughly 3%.

Patients with chronic disease or deformities that have become severe are at greater risk of neurologic injury, hemorrhage, or visceral injury during surgery. The increase in surgical complexity and potential morbidity associated with chronic, advanced disease is another good reason for earlier surgical intervention in patients with extensive bony involvement or impending neurologic compression.

CASE CONTINUED

Despite aggressive antibiotic and supportive treatment, our patient's vision never returned. The abscesses and spinal infection were cured, and her back pain and deformity were relieved by the spinal reconstruction. Still, her overall function and quality of life have been severely curtailed by this ancient but still dangerous disease.

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