# A Bad Back Needs Help

## *Case Presentation*

*History of Present Illness*

A 61-year-old man was admitted to the hospital with a 2 month complaint of atraumatic back pain, worsening over the previous two weeks. The patient described his pain as sharp, 10/10 in intensity, radiating to his ribs, right hip, and right groin, and aggravated by coughing, weight lifting, and movement. His pain was worse in the supine and prone positions, with some relief provided by sitting, and relieved with high doses of pain medications, topical lidocaine, menthol-containing skin ointments, and chiropractic adjustments. Over the 2 days prior to admission, the patient became increasingly desperate as a result of his pain, and drank several pints of vodka.

*Past Medical and Social History*

The patient is a retired machinist whose medical history includes fibromyalgia and binge drinking. He smokes three-quarters of a pack of cigarettes per day.

*Physical Examination*

Physical examination showed normal vital signs and there was pain to palpation over the thoracic spine but no pinpoint tenderness or vertebral abnormalities. Back extension was limited, although flexion was 100 degrees. Lateral flexion was limited by pain equally bilaterally. Neurological examination was normal.

*Laboratory Evaluation*

Admission laboratory values included complete blood count, showing a normal white blood cell count but a normocytic, normochronic anemia, with a hemoglobin of 8.4 mg/dL and an elevated platelet count of 454,00 cells/μL. Serum chemistries showed an elevated glucose of 295 mg/dL and modest hypokalemia of 3.4 mmol/L. Liver enzymes were all modestly elevated. Urine analysis showed glycosuria of 150-200 mg/dL and microscopy showed 13 red blood cells per high-power field. Cultures of blood and urine were negative. Material obtained for sputum specimen was deemed inadequate for evaluation.

*Radiographic Evaluation*

Admission chest radiography (Figure 1, lateral projection) and thoracic spine magnetic resonance imaging (Figure 2) was performed.

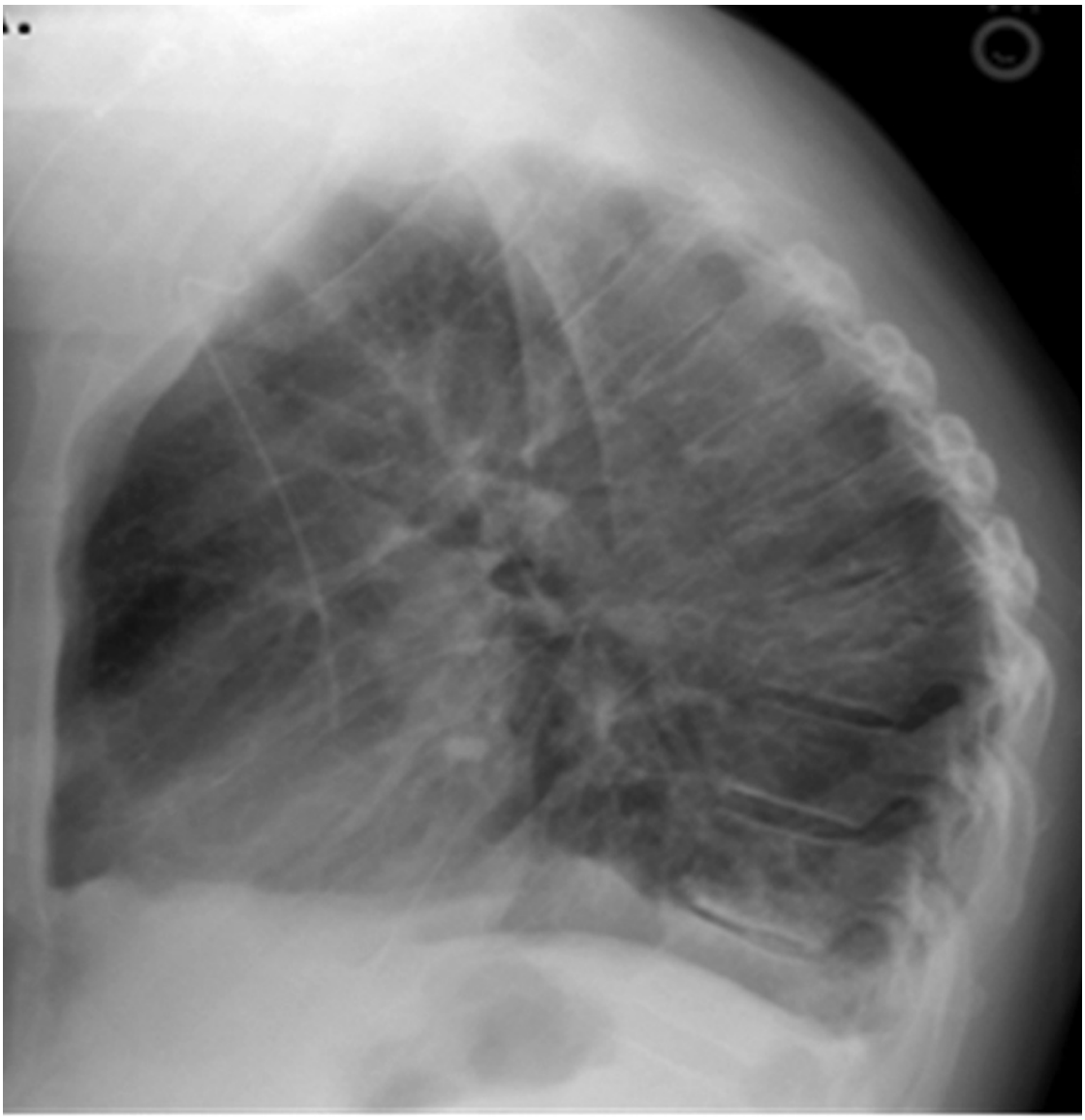

**Figure 1**: Lateral projection from a frontal and lateral chest radiographic examination shows compression fractures involving the mid-thoracic spine.

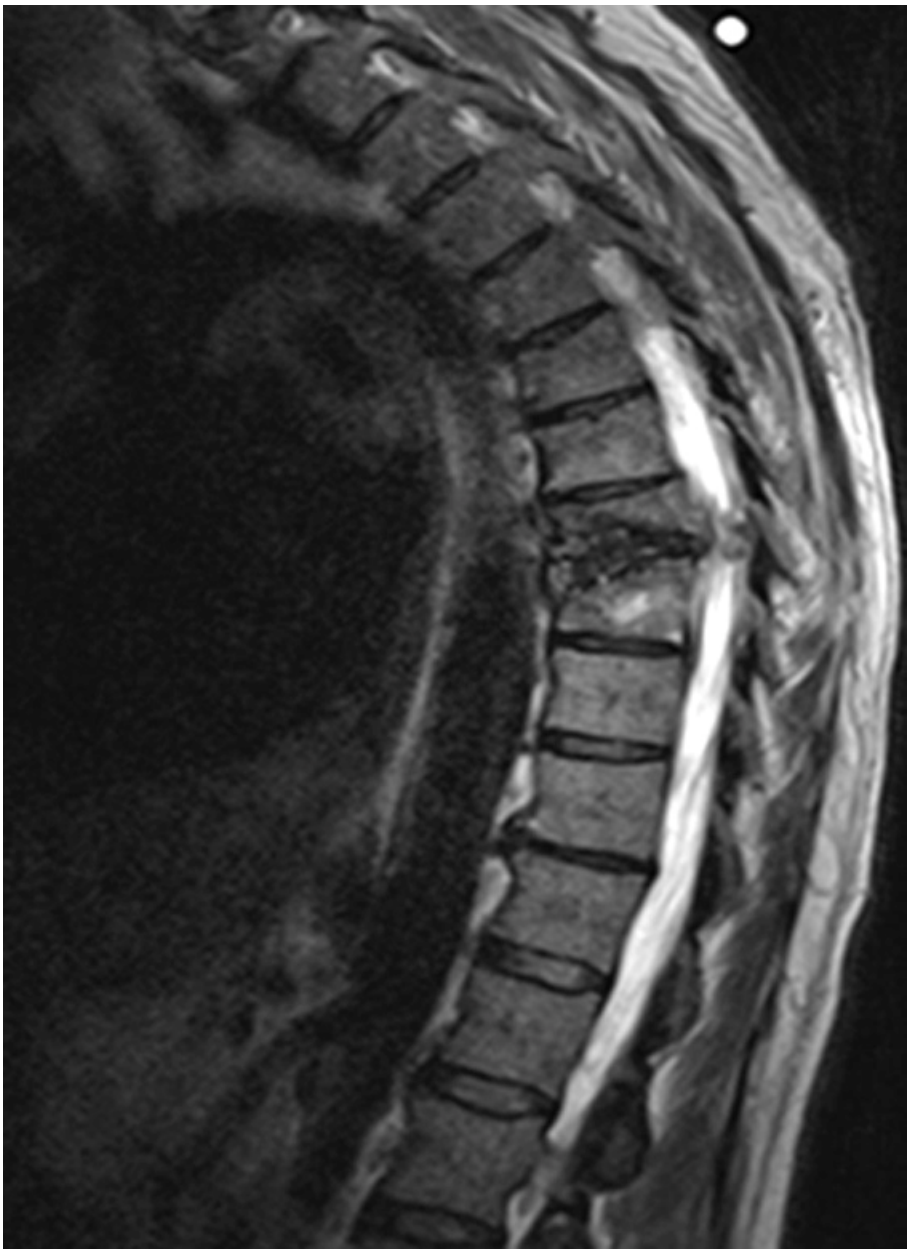

**Figure 2**: Thoracic spine sagittal T2-weighted magnetic resonance imaging shows loss of normal height of approximately one-third of the normal vertebral body height at T8-T9.

The patient was taken to the operating room for drainage of a paraspinal abscess, and biopsies and cultures from material obtained at the T8-T9 levels were performed- these cultures were negative. Nearly one month later, the spine was stabilized with rods and screws and the biopsies and cultures were repeated. These cultures eventually grew *Mycobacterium tuberculosis* and anti-tuberculous therapy was initiated.

## *Questions and Discussion*

Which of the following drug regimens would be appropriate therapy for this patient?

1. Isoniazid 300 mg, rifampin 600 mg daily.
2. Isoniazid 600 mg, rifampin 1200 mg three times per week.
3. Isoniazid 300 mg, rifampin 600 mg, and ethambutol 1200 mg daily.
4. Isoniazid 300 mg, rifampin 600 mg, ethambutol 1200 mg, and pyrazinamide 1500 mg daily

Tuberculous spondylitis, also known as *Pott's disease*, results from hematogenous spread of tuberculosis from an extraspinal source (1). The infection typically involves the anterior aspect of the vertebral body, beginning within the subchondral plate, and spreads within the subligamentous space to involve an adjacent vertebral body. In adults, because the intervertebral disc is relatively avascular, the intervening disc space is typically secondarily involved by infection, resulting in discitis in addition to osteomyelitis. In contrast, in children, the intervertebral disc space is relatively vascular and may be the primary site of infection. Disc space involvement in patients with tuberculous spondylitis typically occurs late in the disease course, in contrast to pyogenic discitis and osteomyelitis. As the vertebral body becomes progressively destroyed, loss of vertebral height ensues, producing the development of the kyphosis, or gibbus deformity, typical of this disorder. Tuberculous spondylitis typically involves several vertebral body levels and relatively spares the discs spaces and posterior elements, in contrast to pyogenic discitis and osteomyelitis. Spread of infection into the adjacent psoas muscles is common, often producing fluid collections that are detectable on cross sectional imaging. Calcification may develop within these collections and is pathognomonic of tuberculous infection.

The indolent nature of tuberculous osteomyelitis and septic arthritis often leads to delayed or overlooked diagnoses. The most common symptom of tuberculous spondylitis is local pain, becoming increasingly over weeks to months, and occasionally associated with muscle spasm and rigidity. Constitutional symptoms, fever, and weight loss are present in less than 40% of patients (1). The most important potential complication of tuberculous spondylitis is spinal cord compression during the active phase of the infection, resulting in paraplegia. In countries where the incidence of tuberculosis is low, the diagnosis of tuberculous spondylitis is often significantly delayed due to a low index of suspicion (2). Unfortunately, the presentation of tuberculous spondylitis also tends to be late in highly endemic areas as a result of poor access to medical care and/or poverty; in this setting, 40-70% of patients with tuberculous spondylitis have symptoms and signs of spinal cord compression at the time of diagnosis.

The American Thoracic Society, Centers for Disease Control, and Infectious Disease Society of America recommends 4 drug therapy for initial treatment of tuberculous spondylitis (1). Therefore in the question above, response #4 is correct. Treatment for

tuberculous spondylitis for a minimum of 6 months is recommended, but usually 12-18 months is typical, with even longer treatment for slowly responding patients.

This patient responded well to therapy, although his wife, a naturopath, felt he was taking too much medication. After several months of therapy, drug sensitivity results became available, showing that the organism in this patient was resistant to isoniazid at 0.2 micrograms/ml, but sensitive at 1.0 microgram/ml.
What should be done next?

1. Stop the isoniazid
2. Continue the present regimen
3. Add a fluoroquinolone
4. Add an aminoglyoside
5. Add linezolid

Some experts favor continuing isoniazid in the setting of "low-level" isoniazid resistance, i.e., resistant to a concentration of 0.2 micrograms/mol but sensitive to 1.0 micrograms/mL (2). Others favor addition of fluoroquinolone to this regimen for the duration of therapy (3). Regardless, close observation, usually with directly observed therapy, is probably prudent. Therefore, either answers #2 or #3 is correct. The patient was continued on his present regimen and continues to make slow clinical progress.

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