27 Tuberculous Spinal Infection

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INTRODUCTION

Tuberculosis (TB) is classically considered an infectious disease of developing countries; however, it has made a resurgence in other areas of the world with the increased use of immunosuppressive drugs, increased immigration, and the relatively recent appearance of HIV. The musculoskeletal system is the most common extrapulmonary site of TB infection, with spinal involvement seen in 50% of skeletal cases. Tuberculous spondylitis refers to vertebral body involvement with TB. When compared with pyogenic infections of the spine, tuberculous spondylitis has a distinct pattern of spinal involvement on imaging, as well as a unique pattern of progression that warrants its own description.

Within endemic countries, tuberculous spondylitis typically affects children and young adults during primary lung infection; however, in Western countries adults are more commonly affected after reactivation of latent disease. As a result, the clinical diagnosis of early spinal TB can be difficult in patients without a known history of pulmonary TB. Less than half of these patients will have simultaneous pulmonary infection. Furthermore, the classic constitutional symptoms of TB (e.g., fever, night sweats, weight loss) are present in less than 40% of spinal cases and may not become clinically apparent for months after initial spinal involvement. The usual duration of illness ranges from 4 to 11 months. In some instances the diagnosis may be delayed by more than a year.

Most patients will seek medical care only after developing severe pain or neurologic complications. Radiologic examinations are one of the first and most important steps in establishing the diagnosis of tuberculous spondylitis. The goal of early diagnosis is avoiding significant morbidity associated with spinal instability, which may occur with delayed treatment. After suspicious imaging abnormalities are identified in the spine, percutaneous image-guided bone or soft tissue biopsies can be performed. Acid-fast staining or polymerase chain reaction can be used to quickly identify the organism when specifically suspected. Notably, TB is notoriously difficult to isolate on cultures, averaging 4 to 6 weeks to obtain results, with a sensitivity of 80%. As a result, microbiology data are often negative. Histopathology of the bone biopsy may show nonspecific granulomatous changes suggesting TB. The cryptic clinical and microbiology features of this disease accentuate the need for an accurate imaging diagnosis.

Lastly, spinal TB is far from a new disease. DNA analysis has identified TB strains from bone biopsies of ancient Egyptian mummies, making it one of the oldest known communicable diseases. However, newer multidrug resistant TB strains have been discovered, necessitating prolonged and aggressive treatment regimens.

IMAGING EVOLUTION: OVERVIEW

Tuberculous spinal infections are most often encountered in the lower thoracic or upper lumbar spine, although any spinal segment may be involved. TB infection is spread hematogenously either arterially or by Batson venous plexus, with the primary exposure site occurring in the lungs or genitourinary system. Similar to pyogenic infections, the anterior vertebral end plate is typically the first site of involvement in the spine, followed by involvement of the central vertebral body. Involvement of the posterior elements is rare (Fig. 27.1).

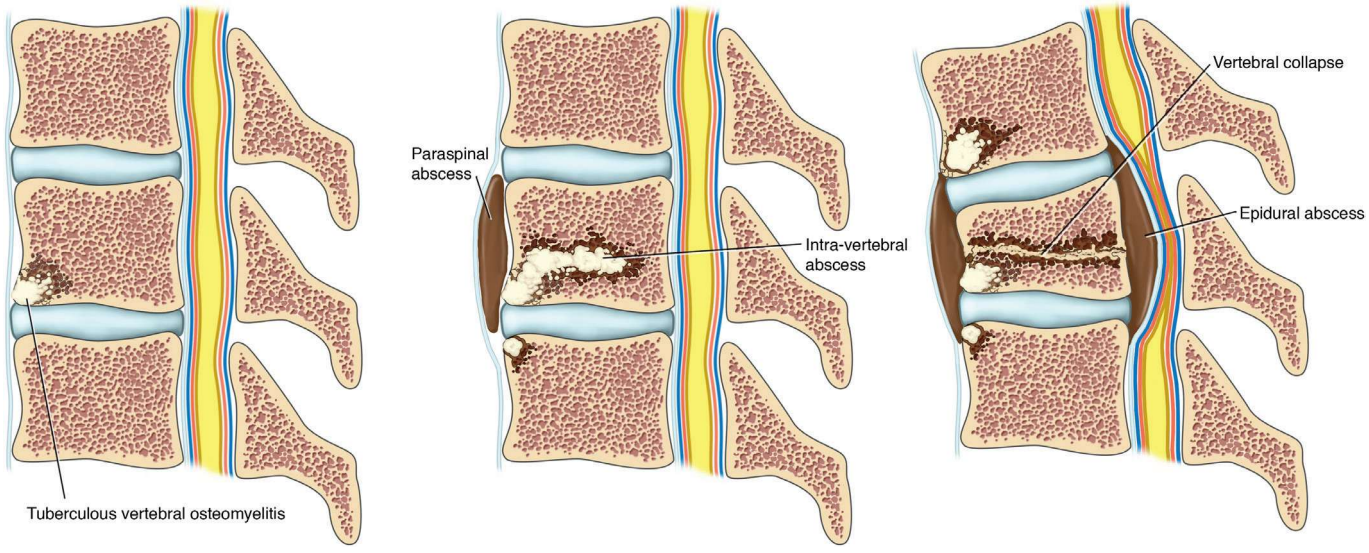

Figure 27.1. Evolution of tuberculous spondylitis. Initial vertebral body involvement may be indistinguishable from early pyogenic spondylitis; however, the disc space is uniquely spared in the early and intermediate phases of tuberculous spondylitis, in contrast to pyogenic bacterial infections. Bone destruction with peripherally enhancing intraosseous abscesses and thin-walled subligamentous paraspinal abscesses demonstrating limited surrounding inflammatory phlegmonous changes is commonly encountered prior to involvement of the disc space. Vertebral collapse is a common late-stage complication.

After the vertebral body is involved, the infection will spread along the undersurface of the longitudinal ligaments to involve multiple adjacent vertebrae and into the epidural/paraspinal soft tissues to form abscesses. Disc destruction is typically not present in tuberculous spondylitis, which is thought to be due TB's lack of proteolytic enzymes, which are present in most pyogenic bacterial infections.

Vertebral body collapse is much more common with TB than with pyogenic infections, creating the classic "gibbus deformity" of short segment kyphosis (Fig. 27.2). This classical late stage

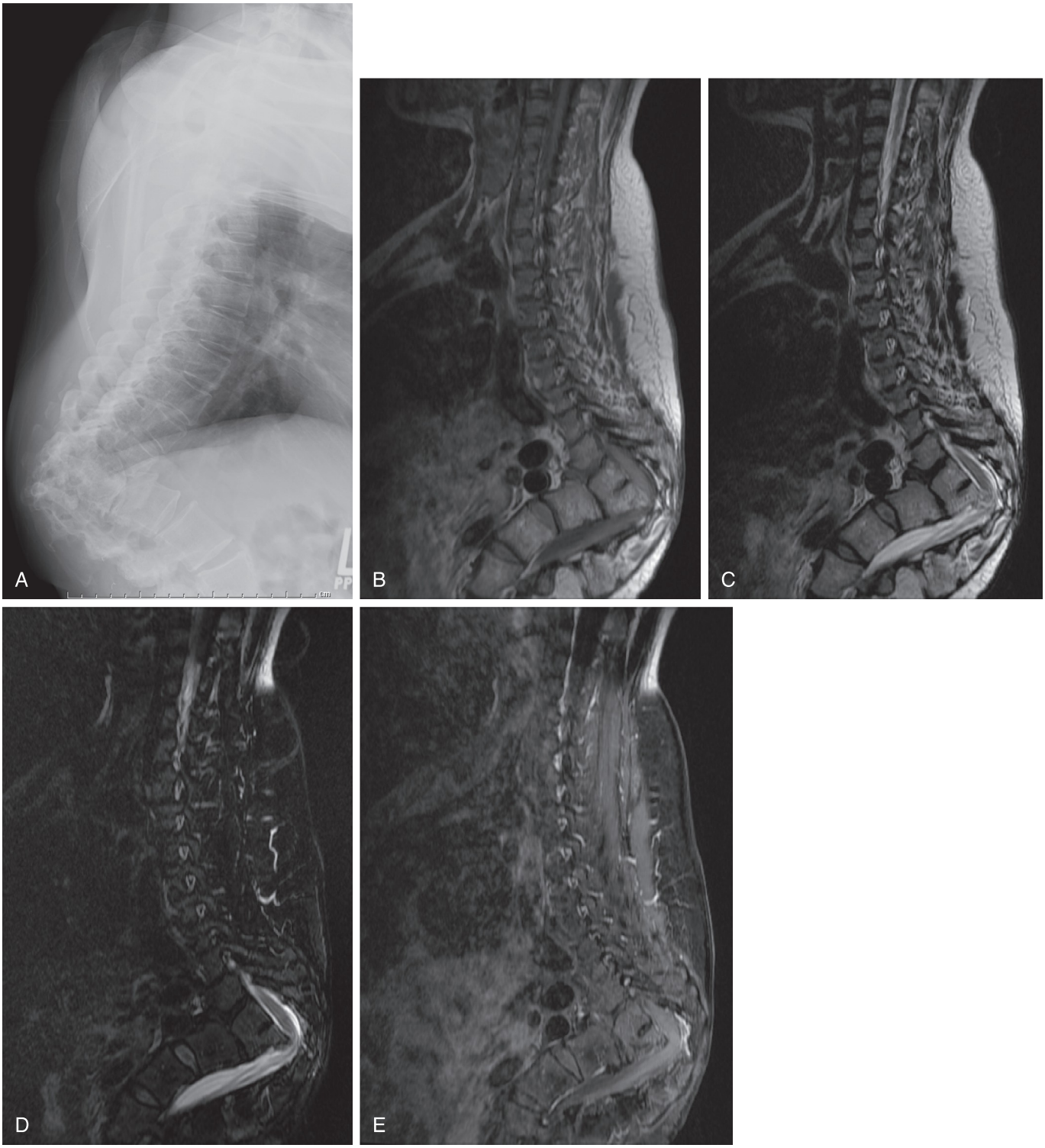

Figure 27.2. Gibbus deformity of spinal tuberculosis. Lateral radiograph of the spine (A) demonstrates severe focal kyphosis of the upper lumbar spine. Sagittal T1 (B), T2 (C), STIR (D), and postcontrast T1 (E) images of the thoracolumbar spine demonstrate severe focal kyphosis with vertebral collapse. No bone marrow edema or paraspinal abscess is identified, which is consistent with remote chronic disease changes.

appearance of severe focal kyphosis was first described by English surgeon Sir Percivall Pott in 1779 (hence the use of the term Pott disease). Neurologic complaints are much more common at this stage of the disease because the kyphosis can result in compression of the anterior spinal cord.

IMAGING EVOLUTION: IN GREATER DEPTH

Although magnetic resonance imaging (MRI) is the "gold standard" for infectious spondylitis imaging, radiographs and computed tomography (CT) may be the only modalities available in endemic regions and early disease may go undiagnosed in these instances. Many of the findings on radiographs and CT are similar, with the main advantage of CT being earlier recognition of paraspinal abnormalities and superior evaluation for calcifications that may be seen within tuberculous abscesses. The earliest finding on radiographs and CT is rarefaction and irregularity of the vertebral end plates. Late-stage findings include vertebral body collapse, osseous fragmentation, and disc height loss.

With MRI, the earliest finding of anterior end plate edema is often indistinguishable from other infectious and inflammatory spondyloarthropathies. However, as tuberculous vertebral osteomyelitis progresses, focal vertebral bone marrow edema and contrast enhancement will often develop into peripherally enhancing intraosseous abscesses with sparing of the intervertebral disc, in comparison to the more diffuse homogeneous enhancement of pyogenic osteomyelitis with early involvement of the adjacent intervertebral disc. Bone destruction will continue to be the predominant finding in tuberculous spondylitis, with eventual vertebral body collapse. Often more than one vertebral level will be involved at presentation.

Although discitis may occur in advanced disease (reported in up to 50%–70% of cases), one major study found disc height to be preserved in more than half of tuberculous spondylitis cases. Although infection may spread through the disc to involve the contiguous vertebral body (as typically seen in pyogenic infections), the unique pattern of multilevel vertebral osteomyelitis seen with tuberculous spondylitis is more likely to occur by subligamentous spread, best seen on postcontrast imaging (Fig. 27.3). MRI of

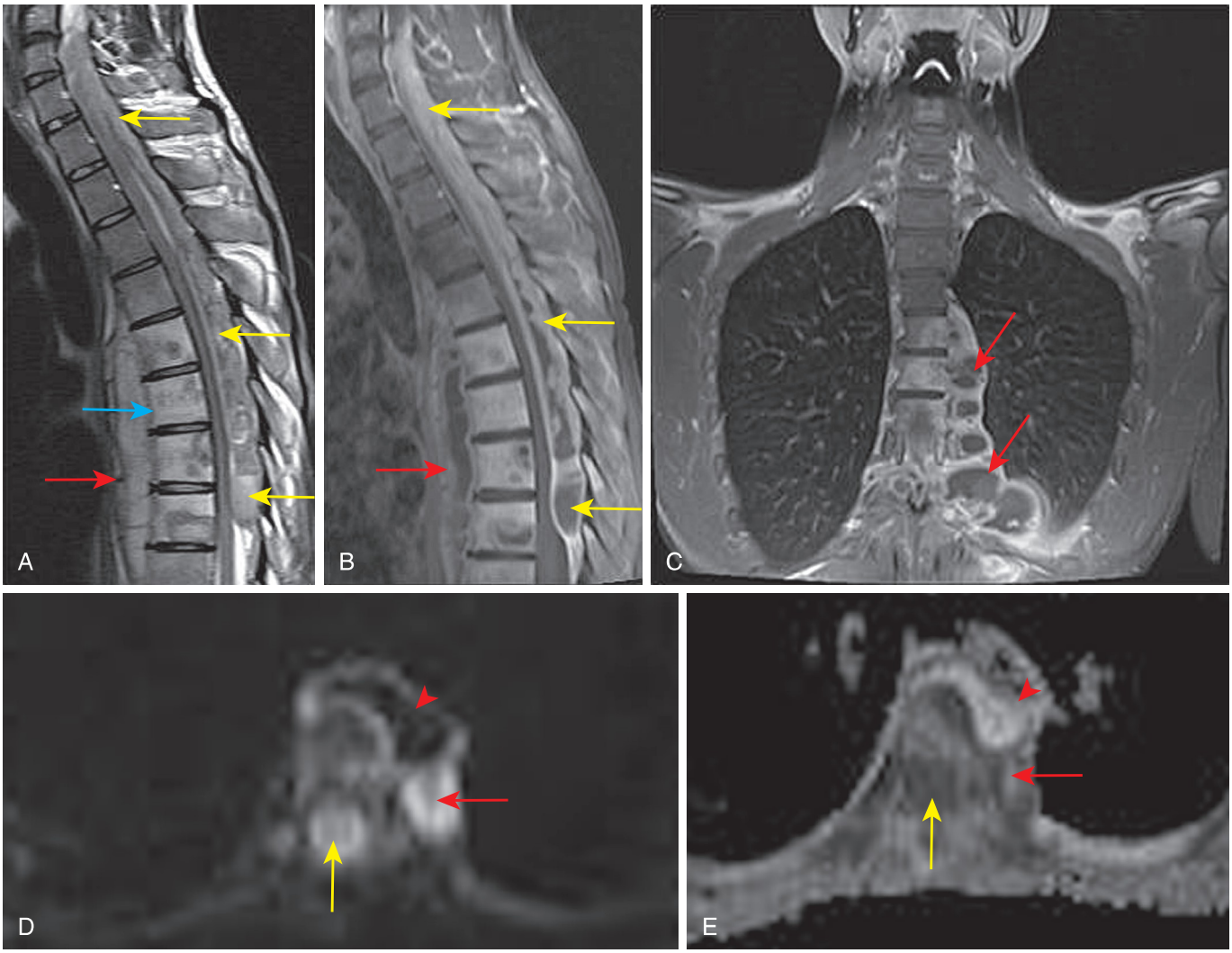

Figure 27.3. Tuberculous spondylitis with epidural and paraspinal abscesses. Sagittal T2 image (A) shows abnormal marrow signal from T4 to T9 *(blue arrow)* with abnormal fluid collections in the epidural *(yellow arrows)* and prevertebral *(red arrows)* spaces. These collections are better seen on postcontrast sagittal (B) and coronal (C) images. Note the sparing of the intervertebral discs. DWI (D) shows areas of hypointensity and hyperintensity with corresponding high and low ADC values (E). The collection with high ADC value (*red arrowhead* in D and E) is a "cold abscess." *(From Moritani T, Kim J, Capizzano AA, et al. Pyogenic and non-pyogenic spinal infections: emphasis on diffusion-weighted imaging for the detection of abscesses and pus collections.* Br J Radiol. *2014;87[1041]:20140011.)*

the entire spine should be performed in these cases to exclude distant skip lesions.

Abscesses are also very common in tuberculous spondylitis, occurring after extension into the surrounding soft tissues. Abscesses can grow to be quite large without causing significant pain or surrounding inflammatory change, often termed "cold abscesses." Cold abscesses can be distinguished from pyogenic abscesses by the lack of diffusion restriction on diffusion-weighted imaging (DWI) (see Fig. 27.3). Calcifications within the abscesses are a chronic finding and considered pathognomonic for TB.

In a study that described the early MRI findings of spinal TB, osteomyelitis was the earliest imaging finding, occurring on average 3 months after symptom onset, followed by abscess formation (5 months after symptom onset), and finally discitis (7 months after abscess formation). The overall progression of tuberculous spondylitis may, in part, occur at different rates depending on the patient's ability to mount an immune response.

Notably, a normal appearance of the lungs should not influence interpretation of the spinal findings because spinal TB in adults is typically the result of reactivation of latent disease.

MIMICS AND DIFFERENTIAL DIAGNOSIS

1. *Pyogenic Spondylitis:* As discussed in the previous chapter and earlier in this chapter, early hematogenous pyogenic infections classically involve the anterior vertebral body end plates, similar to TB. Unlike TB, pyogenic osteomyelitis is more likely to demonstrate diffuse homogenous enhancement on MRI and to demonstrate disc destruction. Subligamentous spread, skip lesions, and vertebral body collapse are less frequently encountered. (See dedicated Spondylodiscitis chapter for case examples.)
2. *Brucellar Spondylitis:* Brucellosis is a gram-negative coccobacillus that is encountered most often in developing nations, commonly from ingestion of unpasteurized milk. The organism has a predilection for the musculoskeletal system, particularly the spine, where both focal and diffuse forms exist. Brucellar spondylitis is often difficult to distinguish from its tuberculous counterpart. Gibbus deformities and paraspinal calcifications are less commonly seen in brucellosis; however, serologic tests should be performed for both organisms for definitive diagnosis (Fig. 27.4).

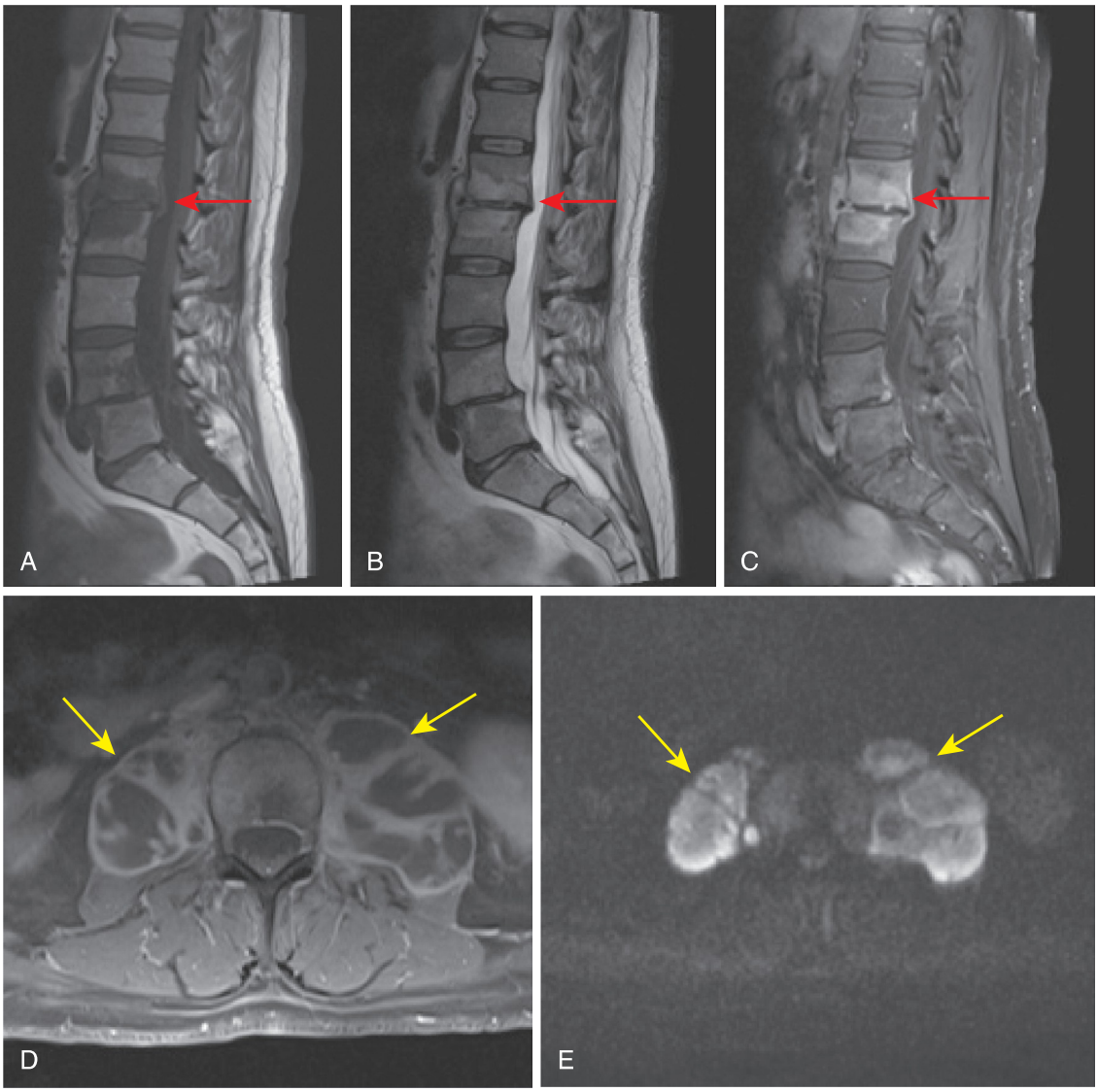

Figure 27.4. A 62-year-old recent immigrant from Mexico. Sagittal T1 (A), T2 (B), and fat-saturated postcontrast T1 (C) images show abnormal vertebral body marrow signal abnormality at L1-L2 *(red arrows)*. There is a small epidural phlegmon and prespinal soft tissue enhancement at L1-L2. Axial postcontrast (D) and DWI (E) images show large, rim-enhancing fluid collections with diffusion restriction in both psoas muscles consistent with large abscesses *(yellow arrows)*. Sampling of this fluid revealed *Brucella* as the causative organism.

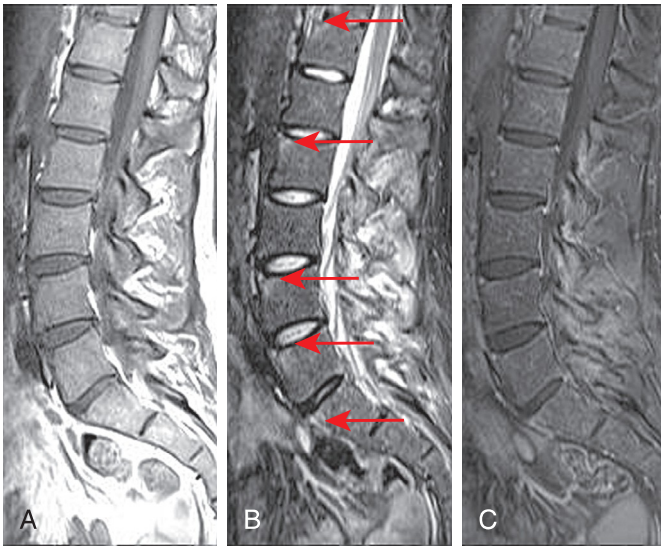

Figure 27.5. Sagittal T1 (A), STIR (B), and T1 fat-saturated postcontrast (C) images in a young patient with back pain demonstrate edema of the anterior superior end plates of T12, L2, L4, L5, and S1 (*red arrows;* best seen on STIR) without appreciable enhancement. In addition, the patient was found to have bilateral sacroiliitis (not shown). Findings represent early inflammation of ankylosing spondylitis.

3. *Ankylosing Spondylitis:* Early in its disease course, active inflammation and erosions at the corners of the end plates (Romanus lesions) may be indistinguishable from infection. A late manifestation of ankylosing spondylitis is pseudoarthrosis. This occurs after a traumatic event on the fused spine, creating a three-column stress fracture that extends through the posterior elements. The fracture line provides a point of mobility in an otherwise fused spine, and surrounding inflammation results (Andersson lesion). This should be distinguishable from infection, given the clinical history, lack of widespread edema/enhancement, and involvement of the posterior elements (Fig. 27.5).
4. *Metastatic Disease:* Metastatic disease classically demonstrates T1 hypointense marrow signal of multiple vertebral bodies with potential for collapse, similar to TB. Metastatic disease more frequently involves the posterior vertebral body and posterior end plates and should not have epidural or paraspinal abscesses/fluid collections, although it can exhibit solidly enhancing epidural or paraspinal disease.

SUGGESTED READING

Al-Nakshabandi NA. The spectrum of imaging findings of brucellosis: a pictorial essay. *Can Assoc Radiol J*. 2012;63(1):5–11.
Burrill J, et al. Tuberculosis: a radiologic review 1. *Radiographics*. 2007;27(5):1255–1273.
Chang M-C, et al. Tuberculous spondylitis and pyogenic spondylitis: comparative magnetic resonance imaging features. *Spine*. 2006;31(7):782–788.
Chen C-H, et al. Early diagnosis of spinal tuberculosis. *J Formos Med Assoc*. 2016;115(10):825–836.
Colmenero JD, et al. Establishing the diagnosis of tuberculous vertebral osteomyelitis. *Eur Spine J*. 2013;22(4):579–586.
Currie S, et al. MRI characteristics of tuberculous spondylitis. *Clin Radiol*. 2011;66(8):778–787.
Desai SS. Early diagnosis of spinal tuberculosis by MRI. *Bone Joint J*. 1994;76(6):863–869.
Garg RK, Dilip Singh Somvanshi. Spinal tuberculosis: a review. *J Spinal Cord Med*. 2011;34(5):440–454.
Gouliamos AD, et al. MR imaging of tuberculous vertebral osteomyelitis: pictorial review. *Eur Radiol*. 2001;11(4):575–579.
Hong SH, Choi JY, Lee JW, et al. MR Imaging assessment of the spine: infection or an imitation? *Radiographics*. 2009;29(2):599–612.
Lee KY. Comparison of pyogenic spondylitis and tuberculous spondylitis. *Asian Spine J*. 2014;8(2):216–223.
Momjian R, Mina G. Atypical imaging features of tuberculous spondylitis: case report with literature review. *J Radiol Case Rep*. 2014;8(11):1.
Moritani T, Kim J, Capizzano AA, et al. Pyogenic and non-pyogenic spinal infections: emphasis on diffusion-weighted imaging for the detection of abscesses and pus collections. *Br J Radiol*. 2014;87(1041):20140011.
Taylor GM, Murphy E, Hopkins R, et al. First report of Mycobacterium bovis DNA in human remains from the Iron Age. *Microbiology*. 2007;153(4):1243–1249.