Imaging of Spine Infection

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KEYWORDS
- Spine infection
- Pyogenic spondylodiscitis
- Pyogenic
- Granulomatous
- Epidural abscess
- Facetitis
- Pyomyositis

KEY POINTS
- Spinal infections are increasing, and the radiologist plays a critical role in both image interpretation and image-guided sampling.
- Magnetic resonance imaging is the modality of choice for imaging spinal infection.
- Pyogenic spondylodiscitis is typically centered about a disc space and when possible, should be differentiated on imaging from Modic type 1 endplate changes.
- Spinal epidural abscesses and the rare subdural abscesses are emergencies.
- Tuberculous spondylitis can be suggested over pyogenic spondylodiscitis on the basis of several characteristic imaging features.

This article reviews the imaging and relevant clinical details of infection of the extradural spine. Radiologists play an important role in diagnosing spinal infection. Due to the aging and more globally mobile population, the increase in predisposing comorbid factors such as diabetes and intravenous drug use, the increase in spinal instrumentation, elevated awareness, and the increased use and diagnostic performance of advanced imaging, spine infections are increasing in incidence and in frequency of diagnosis. They are clinically important despite their relative rarity, because they may be life-threatening, and because early diagnosis leads to improved outcomes. The focus is on pyogenic spondylodiscitis, the most common type of spine infection encountered in routine clinical practice. The also typically pyogenic conditions of epidural abscess and subdural abscess, facet joint infection, and pyomyositis are discussed. Nonpyogenic, granulomatous infections are also addressed. Magnetic resonance imaging (MRI) is emphasized, as it is generally the most sensitive and specific diagnostic modality for these entities. The radiologist’s role in performing minimally invasive sampling procedures is highlighted. With the exception of subdural (intradural) abscess, infections and inflammatory conditions of the spinal cord, spinal nerve roots, and spinal meninges are not considered. The extremely rare parasitic spinal infections are also beyond the scope of this article. The imaging of these conditions has been reviewed elsewhere, including recently in this journal.

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PYOGENIC SPONDYLODISCITIS

Background

Pyogenic spondylodiscitis is a bacterial infection of the bony spinal column, the intervertebral discs, and/or the ligaments of the extradural spine. The most common cause of pyogenic spondylodiscitis is hematogenous spread of infection from a remote site. This is typically via the arterial route, although the paravertebral venous plexus has also been implicated. Urinary tract infections are the most frequent culprit. Other etiologies include direct inoculation, such as in the postoperative setting, discography, and therapeutic spinal injections, and contiguous spread from adjacent infected sites. The infection is usually unimicrobial. Staphylococcus aureus is the most frequent causative organism, accounting for at least one-third of cases in multiple studies. Diabetic patients in their 50s and 60s are frequently affected, and...
the disease is more common in men. Additional predisposing factors include chronic disease, such as renal failure and cirrhosis, other immunosuppressed states, and intravenous drug use.

Clinically, patients with pyogenic spondylodiscitis often pose a diagnostic dilemma, because the presentation may be subtle, insidious, and nonspecific. The time course can range from acute to chronic. Typically the patients present with focal back pain, often with associated muscle spasm and tenderness to palpation. There may be limited range of motion, neurologic deficit, weight loss, and malaise. Fever is relatively common but inconsistently present. The symptoms are often progressive, as the disease is frequently diagnosed relatively late in its course, even up to several months after symptom onset. Laboratory evaluation is not reliably sensitive, although elevated erythrocyte sedimentation rate (ESR) and elevated C-reactive protein (CRP) are often present. Leukocytosis is less commonly observed.7–9

Anatomically, the lumbar spine is most frequently affected, while the cervical spine is the least common subsite. The vertebral bodies and the intervertebral discs in the adult have a tenuous vascular supply, as both the rich network of intrasosseous anastomoses and the profuse capillary network at the vertebral margins of the discs involute by adolescence. The most widely accepted pathophysiologic hypothesis is that an infected embolus causes infarction and subsequent infection in the vertebral body metaphysis. Infection typically begins in the anterior aspect of the vertebral bodies. It can spread to the remainder of the body, the opposite endplate, and particularly early, the adjacent disc. Spread into the paraspinal and/or epidural spaces is common. Infection can also extend more deeply into the subdural or subarachnoid spaces. In children, the still highly vascular disc is often the primary site of infection, as the robust intrasosseous arterial anastomoses protect the bone from substantial involvement.

**Imaging Evaluation**

Once a decision has been made to pursue spinal imaging in a patient with back pain, radiographs should be the first imaging modality obtained. However, radiographs are notoriously insensitive for discitis–osteomyelitis, particularly early in the course of the disease. They may in fact remain normal for several weeks after infection. Thus, findings such as endplate irregularity/erosion, loss of disc space height, and paraspinal bulging or loss of soft tissue planes typically lag behind the clinical evolution. But since the disease is often not diagnosed early, with at times months of symptoms before diagnosis, radiographs can be useful and are often positive (Fig. 1A, B). In a recent systematic review, radiographs demonstrated abnormal findings in 89% of cases.7 Chronic phase findings include endplate sclerosis and ankylosis across an affected interspace.

MRI is the most useful modality for imaging spine infection, and the findings of pyogenic spondylodiscitis have been well described and reviewed in the literature (see Figs. 1C–G; 2A–F, H–L, 3C, D; Box 1). A screening study when clinical suspicion exists should evaluate the entire neuroaxis. Regarding MRI protocol, fat-suppressed T2-weighted sequences are useful, and postgadolinium T1-weighted imaging should include fat suppression to increase conspicuity of the findings.10 In typical disease, infection begins in the anterior aspect of a vertebral body in the metaphyseal region and then spreads, often to involve the intervertebral disc and adjacent vertebral body. Bone marrow involvement manifests as an edema-type abnormality (T1 hypo-, T2 hyperintensity and contrast enhancement), and is classically most marked along the endplates at the infected level. Marrow T1 hypointensity and enhancement have each been found by some authors to be more common than marrow T2 hyperintensity.11 Endplate erosions, and at later stages, vertebral body destruction, may be present. Disc involvement is evidenced by T2 hyperintensity, disc space height loss, loss of the normal T2 hypointense intranuclear cleft, and enhancement. The enhancement pattern of the involved disc is highly variable. Involved paraspinal soft tissues may show an enhancing edema-type abnormality (see Fig. 1G, 2E, F and K, L) or frank abscess (T1 hypo-, T2 hyperintense fluid collection with peripheral contrast enhancement). Overall, findings that have been shown to have particularly high sensitivity for the diagnosis of pyogenic spondylodiscitis include the presence of paraspinal or epidural inflammation, vertebral body T1 hypointensity, disc space T2 hyperintensity, and disc space enhancement. In contrast, findings with relatively low sensitivity include disc T1 hypointensity and height loss.12 Radiologists should scrutinize cases of pyogenic spondylodiscitis for the presence of an epidural abscess. Epidural abscesses have relatively high associated morbidity and mortality, particularly when treatment is delayed. Note that this subsite of infection may also be involved primarily, as extension of infection from other sites (such as facets, paraspinal regions, or the retroperitoneum), or in cases of granulomatous infection. Epidural abscesses, including the MRI...
findings are discussed in greater detail in a subsequent section.

The MRI-based diagnosis of pyogenic spondylodiscitis is not always straightforward. Indeed, pyogenic spondylodiscitis may have atypical appearances, including: lack of expected signal abnormalities and endplate erosive changes early in its course (see Fig. 2), involvement of a single vertebral body, involvement of 1 vertebral body and 1 disc, and involvement of 2 adjacent bodies without the intervening disc. Occasional, vertebral osteomyelitis may present as solitary or multiple discrete, enhancing bony spinal lesions without suspicious abnormalities of the intervertebral discs, mimicking metastatic disease. In addition, when clinical information is not helpful or not available to the radiologist, confident diagnostic interpretation of equivocal cases can be difficult.

**Fig. 1.** Pyogenic spondylodiscitis. An 88-year-old man, with 1 month of back pain, presented with fever and *Escherichia coli* bacteremia. Anteroposterior (A) and lateral (B) views from standing radiographs demonstrate nonspecific loss of disc space height at T12-L1 (arrows). Radiographs are relatively insensitive. Even when findings are present, such as in this case, they are nonspecific. (C) T2-weighted sagittal MRI demonstrates T2 hyperintensity in the narrowed T12-L1 disc space. Note the relative lack of obvious T2 hyperintensity in the adjacent vertebral marrow. (D) T2-weighted, fat-saturated sagittal MRI demonstrates to much better advantage the abnormal T2 hyperintensity in the T12 and L1 vertebral bodies. (E) T1-weighted sagittal MRI demonstrates T1 hypointensity in the T12 and L1 vertebral bodies, centered about the T12-L1 interspace, with some sparing along the opposite endplates. Such marrow T1 hypointensity is a highly constant finding in spondylodiscitis. (F) Postgadolinium, fat-saturated T1-weighted sagittal MRI demonstrates avid enhancement corresponding to the abnormal vertebral marrow signal. Minimal disc space enhancement and mild ventral epidural (white arrow) and anterior paraspinal enhancement (black arrow) are evident. (G) Postgadolinium, fat-saturated T1-weighted axial MRI at the inferior T12 vertebral body level confirms the vertebral body (black triangle), ventral epidural (white arrow), and paraspinal contrast enhancement (black arrows). In this case, the epidural and paraspinal enhancement represents inflammation/phlegmon and/or venous engorgement in these spaces, without discrete abscess formation.
Although MRI is the modality of choice for imaging of pyogenic spondylodiscitis, it is not without shortcomings. MRI is not recommended for routine follow-up. It may lag behind the clinical picture, both at the onset of disease and as the disease improves (see Fig. 2). Several studies demonstrate that the MRI findings can worsen despite clinical improvement. Abnormal MRI findings, particularly of the involved bone and disc, and less so of the paraspinal soft tissues, may persist during the initial 4 to 8 weeks of antibiotic therapy and even months after clinical cure has been achieved. Therefore, MRI is not particularly useful in following patients who are clinically improving on antibiotic therapy. One study suggests that MRI at 4 to 8 weeks may be helpful in patients who are not demonstrating clinical or inflammatory biomarker evidence of improvement. One sign that has been suggested to correlate with healing at MRI follow-up is focal development of marrow T1 hyperintensity (see Fig. 2M). This is thought to correspond with fatty
replacement of healed, previously abnormal bone marrow.\textsuperscript{14,15}

MRI is also challenging in the setting of postoperative infection. Postoperative discitis and/or osteomyelitis is a relatively uncommon complication after lumbar spine surgery. MRI is less reliable in this setting, as even the normal postoperative spine may demonstrate disc or endplate signal changes/enhancement. MRI cannot reliably differentiate between pathology and expected postoperative evolution until at least 6 months after surgery.\textsuperscript{22} One finding that may suggest postdiscectomy change rather than infection is 2 parallel thin bands of enhancement in the disc space; this is in contrast to the more amorphous enhancement generally seen with infection.\textsuperscript{22} Paravertebral enhancement supports the diagnosis of infection, while the absence of enhancing, edema-type subendplate changes or disc space enhancement makes infection unlikely.\textsuperscript{23}

Computed tomography (CT) is more sensitive than radiography, owing to its superior anatomic resolution. It can demonstrate many of the same findings as radiographs and even MRI, particularly when intravenous contrast is used to better evaluate the soft tissues. It is particularly useful when MRI is contraindicated, not available, or equivocal. In the author's experience, it can be especially helpful in confirming the suspicion of advanced degenerative (age-related) disc changes in patients who have been referred for biopsy to exclude infection (Fig. 4). Often, careful review of the CT (and any prior available imaging) may reveal evidence of advanced age-related disc changes, such as disc space vacuum phenomenon, which may obviate the need for biopsy. Image-guided biopsy will be discussed further, but it should be noted here that performing such a biopsy is not without risk. There are the inherent risks and costs of an invasive procedure, and perhaps more importantly, the imperfect sensitivity of biopsy sampling, which, if negative, obligates the clinician to at least consider ordering a repeat biopsy.

Despite relatively high sensitivity and specificity, nuclear medicine imaging is used only in select situations. This is primarily because of its limited spatial resolution and long examination time, as well as the availability, high spatial resolution, and excellent diagnostic performance of MRI. The nuclear study of choice in the author's center is sequential \textsuperscript{99m}technetium-methylene diphosphonate and \textsuperscript{67}gallium-citrate scintigraphy. Uptake that is greater and/or anatomically discordant on the gallium (inflammation detecting) than the technetium (metabolism detecting) portion of the study is the most accurate finding for spondylodiscitis. Select indications in the author's center include patients in whom MRI is contraindicated; MRI and CT are equivocal; multifocal infectious disease is suspected; or the clinical suspicion is high but the MRI is negative. Short-term follow-up MRI may also be helpful in the latter situation.

![Fig. 2](image-url)

**Fig. 2.** MRI pitfalls in pyogenic spondylodiscitis: (1) initial findings may be subtle, especially when early and/or at edge of film if only 1 segment of spine is imaged, and (2) evolution of MRI findings is often discordant with clinical improvement. A 57-year-old man presented for lumbar spine MRI, for the indication of acute lumbar back pain. Initial lumbar spine MRI (A) postgadolinium, fat-saturated sagittal T1-weighted MR image was interpreted as negative. In retrospect, on the corner of the image is subtle thickening and enhancement in the ventral epidural space at T11-12 (white arrow) and less so subjacent to the T12 superior endplate (black arrow). 4 days later, thoracic spine MRI was obtained: sagittal T2-weighted (B), postgadolinium, fat-saturated T1-weighted sagittal (C, D) and axial (E) inferior T11 endplate, (F) superior T12 endplate MR images. The ventral epidural thickening and enhancement compatible with phlegmon is clearly demonstrated and has progressed (white arrows in C, E, F). Disc space T2 hyperintensity and enhancement are evident (block white arrows in B and D). Paraspinal inflammation is present (black arrows in E, F). A fluoroscopically guided disc space biopsy via discography approach was performed (G), yielding Morganella morganii, a gram-negative rod. Despite considerable clinical improvement with medical therapy, including improved symptoms and normalization of inflammatory markers, follow-up MRI 5 weeks later generally demonstrates progression of findings: sagittal T2-weighted (H), postgadolinium, fat-saturated T1-weighted sagittal (I, J) and axial (K) inferior T11 endplate, (L) superior T12 endplate MR images. There is interval increase in loss of disc space height and endplate erosion, T2 hyperintensity (H) and enhancement (I, J) of the disc space and adjacent endplates (block white arrows in H-J), enhancement of the vertebral bodies (black arrowheads in K, L) and paraspinal inflammation (black arrows in K, L). However, epidural thickening and enhancement has improved (white arrows in I, K, L). Some studies suggest soft tissue findings tend to improve before disc space and bony findings, but MRI is generally not useful to follow-up patients who are clinically improving. Sagittal T1-weighted MR image from a follow-up MRI performed 9 months later (M) demonstrates marrow T1 hyperintensity about the interspace (white arrows), corresponding with healed, fatty-replaced marrow. Note the otherwise diffuse marrow T1 hypointensity, which is related to the patient's known non-Hodgkin lymphoma and/or its treatment.
Differential Diagnosis

The most common entity on the imaging differential diagnosis for pyogenic spondylodiscitis is degenerative or age-related disc change, more specifically, the Modic type 1, active endplate change (see Fig. 4). This distinction can be particularly challenging when clinical information is not supportive, as in an afebrile patient. Modic type 1 changes are characterized by edema-type (T1 hypo-, T2 hyperintense) signal abnormality along the vertebral endplates adjacent to a degenerating disc, with T2 hyperintense, fluid filled peripherally enhancing disc space, enhancing edema within the L4 and L5 vertebral bodies, and ventral epidural and paraspinal phlegmon/inflammation. Fluoroscopically guided aspiration of the disc fluid (not shown) was negative but patient had been on antibiotics. Given the imaging findings, clinical features (including elevated ESR and CRP), and risk factors (including morbid obesity and active bacterial external otitis), she was clinically presumed to have and treated for pyogenic spondylodiscitis. (Courtesy of K Schwartz, MD.)

**Box 1**

**Pyogenic spondylodiscitis: classic imaging findings**

- Disc space: T2 hyperintensity, enhancement, height loss
- Adjacent vertebral bodies: endplate destruction, T1 hypo-, T2 hyperintensity, enhancement
- Paraspinal soft tissues: ill-defined inflammation/swelling, abscess
- Epidural space: reactive enhancement/venous plexus distention, phlegmon, abscess

Fig. 3. Disappearing vacuum sign in pyogenic spondylodiscitis. A 46-year-old woman presented for lumbar spine radiographs for the indication of “back pain after a fall.” Lateral radiograph (A) demonstrates a disc space vacuum phenomenon at L4-5. 3 weeks later, she presented with worsening back pain. Lateral radiograph (B) demonstrates loss of the vacuum sign, as well as endplate irregularity and apparent disc space widening at L4-5 suspicious for spondylodiscitis. Sagittal fat-saturated T2 (C) and T1 postgadolinium (D) MRI demonstrate findings of spondylodiscitis, with T2 hyperintense, fluid filled peripherally enhancing disc space, enhancing edema within the L4 and L5 vertebral bodies, and ventral epidural and paraspinal phlegmon/inflammation. Fluoroscopically guided aspiration of the disc fluid (not shown) was negative but patient had been on antibiotics. Given the imaging findings, clinical features (including elevated ESR and CRP), and risk factors (including morbid obesity and active bacterial external otitis), she was clinically presumed to have and treated for pyogenic spondylodiscitis. (Courtesy of K Schwartz, MD.)
spondylodiscitis (see Box 1; Table 1). Often, the degenerating disc associated with Modic type 1 changes demonstrates T2 hypointensity/lack of T2 hyperintensity, in contrast to the hyperintensity typically seen in discitis. This is not always the case, however, as a severely degenerated disc may be T2 hyperintense, and even signal isointense to fluid. Other useful MRI features that can suggest Modic type 1 changes over infection are: stability over time, lack of paraspinal or epidural involvement, lack of disc or endplate enhancement, lack of endplate destructive changes, and presence of a degenerative disc space vacuum sign. The vacuum sign is unlikely to be present in an infection; rare exceptions include an infection very early in its course, in case-reportable infections with gas-forming bacteria, or an infection due to fistula with the gastrointestinal tract. Occasionally
disappearance of a previously visualized vacuum sign may be a clue to the presence of discitis (Fig. 3). Both the vacuum sign and the lack of destructive changes are often better appreciated with CT than MRI (see Fig. 4). In the author’s center, CT is thus used liberally to further evaluate questionable discitis–osteomyelitis seen on MRI, especially when the MRI findings are not strongly suggestive, and a biopsy has been requested. Nuclear medicine imaging can also be helpful in such cases. However, active degenerative age-related disc change cannot always be confidently distinguished from infection by imaging. When the clinical impression, laboratory findings, and imaging do not allow exclusion of infection, biopsy may be performed.

Additional important differential considerations include granulomatous spine infections, which are the subject of a following section. Neuropathic arthropathy of the spine (Charcot spine) may also simulate disc space infection. Features of this destructive entity that are typically not present in pyogenic infection are: disc space vacuum phenomenon, facet joint involvement, exuberant bony debris about the arthropathic joint, joint disorganization with spondylolisthesis or dislocation, T2 hyperintensity and enhancement diffusely involving the vertebral body (rather than just adjacent to the endplates), and rim enhancement of the involved disc. Acute Schmorl nodes may mimic spondylodiscitis, particularly as they may enhance and cause signal change and enhancement in the adjacent bone. However, such acute nodes are typically characterized by a concentric ring of edema-type signal around the node, involvement of only the endplate with the herniated node, and lack of diffuse abnormal signal changes within the disc.

### Table 1

<table>
<thead>
<tr>
<th>Favoring Spondylodiscitis</th>
<th>Favoring Modic Type 1 Endplate Changes</th>
<th>Pitfall/Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disc space signal</td>
<td>T2 hyperintensity</td>
<td>T2 hypointensity or lack of T2 hyperintensity</td>
</tr>
<tr>
<td>Disc space enhancement</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Disc space vacuum sign</td>
<td>Absent, only minimal, or “disappearing”</td>
<td>Often present</td>
</tr>
<tr>
<td>Vertebral body endplates</td>
<td>Endplate destruction</td>
<td>Lack of endplate destruction</td>
</tr>
<tr>
<td>Paraspinal, epidural spaces</td>
<td>Inflammation and/or abscess</td>
<td>Absent</td>
</tr>
<tr>
<td>Location</td>
<td>Anteriorly eccentric</td>
<td>Laterally eccentric: at point of biomechanical stress (eg, inner aspect of curve)</td>
</tr>
<tr>
<td>Fever, elevated inflammatory markers</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Short-term follow-up</td>
<td>Progression</td>
<td>Stability</td>
</tr>
<tr>
<td></td>
<td></td>
<td>If remote comparison is available, even Modic 1 can show significant progression</td>
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</tbody>
</table>

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idiopathic skeletal hyperostosis) may mimic pyogenic spondylodiscitis. The identification of fracture line(s) in the posterior elements can be helpful to suggest the former over infection. Other entities that less commonly pose a diagnostic challenge include dialysis-associated spondyloarthropathy and tumors with propensity to cross the disc space, such as chordoma and myeloma.

**Treatment**

Antibiotic therapy is the mainstay of clinical management, but ideally is withheld or discontinued until after a sampling procedure has been performed. Surgery is performed relatively commonly in patients who suffer complications, such as neural element compression, spinal deformity, or instability related to collapse of the discs or vertebral bodies. As mentioned previously, the clinical and imaging evolution may be discordant. The most important but not perfectly predictive feature indicating healing of discitis–osteomyelitis is decreasing clinical symptomatology, particularly pain and if initially present, fever. Laboratory markers such as improving leukocytosis and ESR can also be helpful. Persistently elevated or increasing ESR and CRP at week 4 of antibiotic therapy suggest a high rate of treatment failure. Relapse risk is significant, and was found to occur in approximately one-third of patients in a recent systematic review, although lower rates are reported elsewhere in the literature. The mortality rate is low, 6% in a recent systematic review.

**SPINAL EPIDURAL ABSCESSION**

**Background**

Although epidural abscesses may occur due to direct extension, such as from spondylodiscitis or facet joint infection, they are more commonly primary, related to either hematogenous spread or iatrogenic inoculation from an invasive spinal procedure. An underlying infectious process is the most common comorbidity. Predisposing immunosuppressive conditions may be present, the most common being diabetes. As in pyogenic spondylodiscitis, men are more commonly affected; *S. aureus* is the most common pathogen (~70% of cases).

The clinical presentation of patients with spinal epidural abscess is variable. Middle-aged to elderly adults are most commonly afflicted. Back pain is the most common symptom and is often severe. Fever and neurologic deficits are also common, and other features may be present, such as limited range of motion and tenderness. Elevations of ESR and CRP are more common than leukocytosis.

Spinal epidural abscesses are most common in the thoracic spine, but can occur in any spinal segment and can even involve the length of the spine. Some investigators have found that they more commonly complicate spondylodiscitis in the cervical than in the thoracic or lumbar spine. The abscesses can be located either in the anterior or posterior epidural space. The cause of neurologic deficits can be direct mass effect or a vascular mechanism via thrombosis or thrombophlebitis.

**Imaging Evaluation**

Some sources recommend emergent MRI of the entire spinal axis when spinal epidural abscess is clinically suspected. MR images reveal a T1 hypo-, T2 hyperintense mass in the epidural space (Figs. 5 and 7). On postgadolinium T1-weighted imaging, this may enhance either homogeneously or heterogeneously in the stage of epidural plexus engorgement/phlegmon (see Fig. 2A, C, E, F) or peripherally with central fluid signal in the mature stage of a centrally pus-filled abscess (see Figs. 5C, E and 7E). Compared with neoplasms, more acute processes such as epidural abscess and hematoma more commonly violate the midline septum of the ventral epidural space. There may be additional engorgement of the epidural plexus or basivertebral veins and/or prominent dural contrast enhancement. Thecal sac and even spinal cord (see Fig. 5D) or cauda equina (see Fig. 7D) compression are common. Diffusion weighted imaging may show the expected restricted diffusion within a spinal epidural abscess.

On follow-up imaging, changes in an abscess do appear to correlate with clinical improvement or deterioration. However, findings of associated spondylodiscitis, when present, evolve in the unpredictable manner described previously.

**Differential Diagnosis**

When associated with spondylodiscitis or facetitis, epidural abscess is not usually a diagnostic challenge on imaging. When an epidural abscess is primary, differential considerations include malignancy, particularly metastasis, and epidural hematoma. Clinical history is often most useful in distinguishing among such entities.

**Treatment**

The classic treatment of choice is emergent surgical decompression and abscess drainage, followed by antibiotic therapy. Select patients are managed medically. Antibiotic therapy may be delayed in the neurologically stable patient until emergent surgery with sampling has been
MRI findings may influence surgical approach, as a more phlegmonous collection may require a widespread decompressive approach with laminectomy, whereas a pus-filled abscess may be treated by limited laminotomies and catheter irrigation. Even in some modern series, the mortality is relatively high (~10%–20%).

**SPINAL SUBDURAL ABSCESS**

Primary subdural (intradural) abscess of the spine is an extremely rare, case-reportable condition. The most common location is the lumbar region. Risk factors and clinical features are similar to epidural abscess. *S. aureus* is the most common organism. Treatment is typically emergent surgical drainage with subsequent antibiotic therapy. The subdural location, deep to the epidural space, can be readily identified on MRI (see Fig. 6).

**FACET JOINT INFECTION**

**Background**

Facet joint infection (facetitis) is an uncommon condition, but like pyogenic spondylodiscitis, it is not rare. It is being increasingly recognized and
Fig. 6. Subdural abscess. An 82-year-old woman presented with acute severe back pain from the neck to the sacrum in the setting of a \textit{S} \textit{aureus} urinary tract infection. Sagittal T2-weighted (A), T1-weighted (B), postgadolinium fat-saturated T1 weighted (C–E), and axial (mid-thoracic level) postgadolinium fat-saturated T1-weighted (F) and T2-weighted (G) MR images. A large, extensive T2 hyper- (A, G), T1 hypointense (B), peripherally enhancing (C–F) fluid collection is located dorsally in the subdural (intradural) space (small black/white arrows). It contacts and ventrally displaces the spinal cord, and can be clearly delineated from the tiny, triangular, and more superficial dorsal epidural fat (white arrowheads in F, G). Its extent is from approximately T2 (block white arrow in D) to the sacrum (block white arrow in E). This was surgically evacuated (H), and \textit{S} \textit{aureus} was cultured. The patient's clinical status after 8 weeks of antibiotic therapy was excellent. (Courtesy of N Campeau, MD, A Nassr, MD.)
may be increasing in incidence. It may result from hematogenous dissemination of a pathogen, most commonly \textit{S} aureus, or be seen in patients without known predisposing recent infection. Sources include cutaneous, respiratory, urinary, and catheter-related infections. The condition is thought to occur most commonly due to spread from either an adjacent site of infection or hematogenous seeding from a remote site. Primary paraspinal pyomyositis is a rare, serious infection of the paraspinal musculature. Primary pyomyositis much more commonly involves the large muscles of the body, especially the thigh muscles. It is usually seen in tropical regions, where it is endemic, but it is being increasingly recognized in temperate zones. Any age group can be affected, but the classic tropical type is most common in children and young adults following muscle trauma. \textit{S} aureus is the pathogen in the majority of reported cases.

**PYOMYOSITIS**

**Background**

Infections of the paraspinal muscles are most commonly due to spread from either an adjacent site of infection or hematogenous seeding from a remote site. Primary paraspinal pyomyositis is a rare, serious infection of the paraspinal musculature. Primary pyomyositis much more commonly involves the large muscles of the body, especially the thigh muscles. It is usually seen in tropical regions, where it is endemic, but it is being increasingly recognized in temperate zones. Any age group can be affected, but the classic tropical type is most common in children and young adults following muscle trauma. \textit{S} aureus is the pathogen in the majority of reported cases.

Patients present with fever, pain, and swelling of the involved region. Although inflammatory markers are typically elevated, blood cultures are frequently negative. Early recognition is critical, but delayed diagnosis is common. The condition is often initially misdiagnosed as a variety of conditions, such as hematoma, muscle strain/spasm/rupture, venous thrombosis, thrombophlebitis, cellulitis, osteomyelitis, septic arthritis, or tumor. Three classic stages are described: stage 1, diffuse muscle infection; stage 2, intramuscular abscess; and stage 3, myonecrosis with sepsis. The condition is thought to occur most commonly due to a damaged muscle’s compromised resistance to infection during concurrent transient bacteremia. However, a variety of predisposing factors causing immune-compromise may also be involved, particularly in older patients presenting in temperate zones.

**Imaging Evaluation**

Radiographs have limited utility. Although they can help exclude a primary bone lesion, even when findings such as soft tissue prominence or loss of fat planes are demonstrated, they are nonspecific. Advanced cross-sectional imaging may demonstrate the replacement of muscle by fluid and inflammatory cells. On CT, hypoattenuation and swelling of the affected muscle group may be seen. Intramuscular accumulation of fluid can be seen in the abscess stage. Ultrasound may also be used to demonstrate the infected muscle or intramuscular abscess, particularly in children. MRI is the most sensitive and specific test (see Fig. 8). Findings in the early, invasive stage of
infection are not as specific as when a frank abscess is demonstrated. This invasive stage is characterized primarily by T2 hyperintensity in the affected muscle. The abscess stage demonstrates a peripherally enhancing, centrally T2 hyperintense fluid collection, with variable central T1 signal intensity and mass effect (see Fig. 8). A peripheral T1 hypointense, T2 hyperintense rim may be present. Lumbar paraspinal lesions can be so large that they cause mass effect on retroperitoneal structures. Spread of inflammation/infection may occur from (see Fig. 8A, C) or to adjacent soft tissue, bone, or joints. Even associated epidural abscess formation has been reported. When untreated or unrecognized, the disease progresses to its final stage of myonecrosis with sepsis; mass effect resolves and the central T2 hyperintensity decreases.

**Treatment**

Treatment includes aggressive antibiotic therapy. Surgical or image-guided drainage is often required when the disease reaches the mature, suppurative abscess stage. The prognosis is good, unless cases are untreated or diagnosed late, in which case mortality can increase to approximately 15%.  

**GRANULOMATOUS SPINAL INFECTION**

**Tuberculous Spondylitis**

**Background**

Tuberculous spondylitis (Pott disease) is the most common nonpyogenic, granulomatous infection of the spine, and the most common overall cause of spine infection in the world. Tuberculosis...
remains endemic in certain regions, and has been recently resurgent in developed countries due to increasing immigration of people from endemic areas, development of drug-resistant strains, and the human immunodeficiency virus (HIV) pandemic. Among patients with tuberculosis, tuberculous spondylitis is much more common in HIV-positive than HIV-negative patients.

Clinically, the classic presentation of tuberculous spondylitis is back pain, kyphotic deformity, and at times a cold abscess (a slowly forming abscess with little associated inflammation). However, tuberculosis of the spine tends to have a more indolent and less painful course compared with pyogenic spondylodiscitis, with latency as long as several years. Systemic symptoms such as weight loss, fever, and malaise may precede the spinal infection.

Anatomically, tuberculous spondylitis is most common in the thoracic spine, particularly at the thoracolumbar junction. It classically begins in the anterior aspect of the vertebral body, from where it can spread in subligamentous fashion anteriorly in the paraspinal region or posteriorly in the epidural space. It is also prone to exhibit intradural involvement, including intradural abscesses and spinal cord myelitis; such spinal tuberculous meningitis will not be further considered here. *Mycobacterium tuberculosis* may seed the spine by hematogenous dissemination, typically from a clinically quiescent primary pulmonary focus. Of note, chest radiographs...

**Fig. 8.** Pyomyositis, with paraspinal abscesses, likely due to adjacent facetitis. 67-year-old man presented with methicillin resistant *S aureus* bacteremia and a screening spine MRI to search for a source was requested. Sagittal fat-saturated T2-weighted (A), T1-weighted (B), postgadolinium, fat-saturated T1-weighted (C), and axial T2-weighted (D), and postgadolinium fat-saturated T1-weighted (E) MR images. A nearly 3 cm T2 hyperintense (A, D), T1 hypointense (B), and peripherally enhancing (C, E), fluid collection (white arrows) is present in the left upper cervical paraspinal muscles. A second smaller, similar collection is present more posterolaterally (black arrows in D, E). Mild associated inflammation/poorly organized infection manifests as ill-defined T2 hyperintensity (A, D) and enhancement (C, E) about the abscesses. The abscesses likely arises from the infected appearing left C5-6 facet joint, which is distended with fluid and exhibits facet/peri-facet edema and enhancement (white arrowheads in A, C). Ultrasound-guided drainage of the paraspinal abscess (F) yielded *S aureus*. (Courtesy of G Miller, MD.)
commonly do not demonstrate evidence of pulmonary tuberculosis in patients with tuberculous spondylitis.\textsuperscript{58}

\textbf{Imaging evaluation}

As for other spinal infections, MRI is the modality of choice in tuberculous spondylitis. The classic imaging appearance of tuberculous spondylitis is similar to pyogenic spondylodiscitis, with involvement of one or more adjacent vertebral levels, destruction of the interspace(s), and adjacent paraspinal soft tissue mass.\textsuperscript{55} However, disc space involvement is often not present, not as severe, and/or more delayed when compared with bacterial spondylodiscitis. This atypical pattern of tuberculous spondylitis is reported by some authors to be quite common and increasing in frequency; in 1 large study, more than 50\% of tuberculous spondylitis patients had only isolated vertebral lesions, mimicking metastases, without disc involvement.\textsuperscript{59} Anecdotally, this is the most common pattern seen in the author’s practice (Figs. 9 and 10). Sparing of the disc space has been ascribed to the fact that \textit{Mycobacterium} lacks

\begin{figure}[h]
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\caption{Tuberculous spondylitis, with paraspinal abscesses and epidural phlegmon. A 53-year-old woman presented with several weeks of back pain and low-grade fevers. Postgadolinium, fat-saturated T1-weighted sagittal (A, B), and sagittal (C) and axial T2-weighted (D) MR images. There are multiple foci of abnormal marrow enhancement (A, B) and T2 hyperintensity (C) throughout the visualized spine. Prominent ventral paraspinal infection in the thoracic region (small white arrows in A) extends across multilevel levels along the anterior longitudinal ligament with relative preservation of disc spaces. A large, discrete peripherally enhancing, centrally T1 hypointense (B), T2 hyperintense (C, D) ventral paraspinal fluid collection is present (large white arrow in B–D), compatible with a paraspinal abscess. Subligamentous ventral epidural phlegmon along the posterior longitudinal ligament manifests as homogeneously enhancing epidural soft tissue (arrowhead in A). Percutaneous CT-guided sampling (E) yielded granulomatous inflammation on pathology, and \textit{M tuberculosis} on microbiology. (Courtesy of J Wald, MD, J Morris, MD, G Miller, MD.)}
\end{figure}
Fig. 10. Tuberculous spondylitis, with predominantly multilevel bony involvement, including of posterior elements. A 42 year-old man was referred for possible metastatic disease. Sagittal T1- (A) and fat-saturated T2- (B), and axial T2-weighted (C–E) MR images. There are multiple bony spinal lesions, including in the vertebral bodies (A, B). A prominent lesion is present in the L3 spinous process (small white arrow in B, C). At L5, a dorsal vertebral body lesion extends into the ventral epidural space and partially effaced the thecal sac (large white arrow in B and D). At L4, extension is seen into the left psoas muscle (arrowhead in E). Image from percutaneous CT-guided sampling (F) confirms the lytic nature of the L3 spinous process lesion. The biopsy yielded caseating granulomatous inflammation on pathology, and M tuberculosis on microbiology. (Courtesy of P McGough, MD, T Maus, MD.)
proteolytic enzymes. From this location, the infection may spread in subligamentous fashion across 1 or more levels beneath the anterior longitudinal ligament (see Fig. 9A). Spread in the epidural space may also occur (see Figs. 9A and 10B, D) but is less common than in the anterior paravertebral regions (see Fig. 9A–D).50 Contrast-enhanced studies often demonstrate thin, smooth enhancing walls of the paravertebral collections. The subligamentous spread may be much more extensive than the vertebral involvement (see Fig. 9A), and can lead to skip lesions of involved bones/discs with intervening normal levels.61 In addition to subligamentous spread, spread to adjacent soft tissue is also common, particularly the anterolateral paraspinal soft tissues (see Fig. 10E). A tuberculous abscess of the psoas muscle occurs in approximately 5% of cases and may contain calcifications, best seen on CT.62 CT can also be useful in demonstrating endplate erosive changes and bony lytic lesions. The posterior elements (see Fig. 10B, C) are more commonly involved than in pyogenic disease. A classic finding is gibbus deformity, due to preferential anterior column involvement causing collapse of a partially destroyed vertebral column. Similar to other spinal infections, radiographic findings tend to lag behind the pathologic changes of tuberculous spondylitis.60

In addition to disc space sparing, atypical appearances of tuberculosis spondylitis include: single vertebral level disease (vertebra plana, ivory vertebra, neural arch involvement, or panvertebral involvement)55 and multilevel disease (contiguous or noncontiguous).55,63 Although considered atypical, several of these and some of the more typical features can suggest tuberculous spondylitis over pyogenic spondylodiscitis.

Differential diagnosis
As described previously, tuberculosis spondylitis can appear identical to typical pyogenic spondylodiscitis when the disc space is involved. Imaging features that favor tuberculosis spondylitis include: well-defined paraspinal signal abnormality; large (larger) inflammatory collections, including large paraspinal cold abscesses; thin, smooth abscess walls; sparing of the disc; subligamentous spread to 3 or more levels; multiple vertebral or entire-body involvement; skip lesions; and posterior element involvement (Box 2, Table 2).13,60,64–66

An additional granulomatous disease that can mimic the appearance of tuberculosis spondylitis is brucellar spondylitis, which will be discussed briefly. Solitary tumor or metastases may be misdiagnosed when tuberculosis spondylitis demonstrates uni- or multilevel bony involvement with disc space sparing.

### Box 2

**Imaging clues: tuberculous spondylitis**

**Classic:**
- Similar to pyogenic spondylodiscitis
- Disc space involvement less severe
- Large paraspinal abscess, smooth wall, ± calcifications
- Subligamentous spread

**Atypical:**
- Disc sparing, with either single or multilevel bony involvement only
- Multilevel involvement, contiguous or skip lesions
- Vertebra plana
- Posterior element involvement
- Panvertebral involvement

### Brucellar Spondylitis

**Background**

Brucellosis is a zoonosis endemic in rural areas of Saudi Arabia and the Mediterranean basin. Those most at risk are farm workers, slaughterhouse workers, and veterinary personnel. It is caused by a gram-negative bacillus, and usually acquired by ingestion of raw meat or unpasteurized dairy products. The most common animals to harbor brucellosis are sheep, cattle, goats, pigs, and dogs.61,67

Some studies suggest high-grade fever is seen more commonly than with pyogenic spondylodiscitis or tuberculous spondylitis.65 Blood cultures and serologic testing typically allow diagnosis, whereas percutaneous sampling of the spine is of little utility.58 Treatment is medical therapy with antibiotics, with a typical duration of 3 to 6 months, but recurrences are not uncommon.53

**Imaging evaluation, differential diagnosis**

Brucellosis most commonly affects the lumbar and lumbosacral regions, unlike tuberculosis spondylitis, which is more common in the thoracic spine. The MRI findings of brucellosis may be indistinguishable from those of tuberculous spondylitis. However, one study suggests that brucellosis...
tends to appear less aggressive than tuberculous spondylitis. Specific features that favor brucellosis over tuberculous spondylitis include: relatively intact vertebral body and even disc despite signal abnormality/enhancement, spared posterior elements, lack of paraspinal abscess, and lack of associated spinal deformity. A relatively low rate of abscess formation has been confirmed by other investigators.

70 The differentiating features among pyogenic, tuberculous, and brucellar spine infections are highlighted in Table 2.

**Fungal Spondylitis**

Fungal spondylodiscitis is uncommon, but the immunocompromised population susceptible to fungal infections is ever increasing. A wide variety of fungal organisms can cause spine infection, including *Candida* and *Aspergillus* species. An early report of the MRI findings in 3 cases of fungal spondylitis in immunocompromised patients described lack of T2 hyperintensity in the disc spaces,71 in contrast to pyogenic spondylodiscitis. A more recent report of 15 cases suggested that similar to tuberculous spondylitis, *Aspergillus* spondylitis should be suspected in immunocompromised patients when multiple vertebral levels are involved with skip lesions or subligamentous spread, or when a serrated appearance of the endplates and subchondral T2 hypointensity is present.72

**IMAGE-GUIDED SAMPLING OF SPINAL INFECTION**

The identification of a specific organism causing a spine infection is useful to direct antibiotic therapy. Blood or urine cultures are positive in some cases, but targeted sampling may be necessary even in some of these cases to definitively identify the pathogen. Thus, in many centers, radiologists play an additional important role in the diagnostic algorithm by performing image-guided percutaneous sampling procedures. If blood or urine cultures are positive, the risks of minimally invasive sampling need to be weighed against the likelihood that a separate concurrent infection is present. Either fluoroscopy (see Fig. 2G) or CT (see Figs. 7F, 9E, 10F) can be used for guidance. Ultrasound (see Fig. 8F) may also be used for relatively superficial paraspinal processes. For typical
pyogenic spondylodiscitis, the author most often uses fluoroscopy and achieves sampling of the disc space and both adjacent endplates via an angled, caudocranial transpedicular approach.\textsuperscript{73}

The reported diagnostic yield of image-guided sampling varies widely, but overall is relatively low. One of the most recent studies retrospectively assessing percutaneous image-guided (fluoroscopy or CT) needle biopsy in patients with vertebral osteomyelitis in routine clinical practice demonstrated a positive culture rate of 30\% in 92 cases where clinical and imaging evaluation were consistent with infection. The yield was lower (16\%) when imaging was indeterminate for infection.\textsuperscript{74} False negatives may result for a variety of reasons, including fungal infections\textsuperscript{75} and initiation of antibiotic therapy before sampling (eg, see patient in Fig. 3). In the appropriate patient, delaying the antibiotic therapy until this sampling has been performed is likely to increase the yield.\textsuperscript{76,77} It is common to repeat the procedure in cases of suspected infection where the initial sampling is nondiagnostic. In fact, negative culture results are difficult to interpret. For example, the aforementioned study demonstrated a 5\% positive culture yield even in cases where the probability of infection was considered low based on the radiographic appearance.\textsuperscript{74} In some centers, surgical percutaneous endoscopic discectomy and drainage are performed, and at least 1 study suggests that the culture yield is higher compared with CT-guided biopsy.\textsuperscript{78} In select patients, open biopsy is performed to attempt to establish the diagnosis.

The combination of histopathologic with microbiologic analysis of the biopsy specimen has been shown to improve the diagnostic performance of image-guided sampling.\textsuperscript{1,4,75,79,80} This advantage stems from the potential for histology to distinguish pyogenic and granulomatous infections, diagnose culture-negative chronic osteomyelitis, and detect unsuspected neoplasm.\textsuperscript{9,79} Typically, the microbiologic analysis includes aerobic, anaerobic, fungal, and mycobacterial tests and cultures. Some authors recommend drawing blood cultures within a few hours of the sampling procedure to further increase the chance of pathogen identification.\textsuperscript{81}

**SUMMARY**

Spinal infections are increasing in incidence. Radiologists play a crucial role in the evaluation of these potentially life-threatening conditions. MRI is the modality of choice for imaging spinal infection. When extradural spinal infection is being considered, it is important to include fat-suppressed T2- and fat-suppressed, contrast-enhanced T1-weighted sequences. Pyogenic spondylodiscitis has characteristic imaging findings centered about an abnormal disc space, but may have atypical appearances. It can be difficult to distinguish pyogenic spondylodiscitis from other conditions, especially Modic type 1 endplate changes. MRI is critical in diagnosing both spinal epidural and the rare subdural abscess, which are both usually treated with emergent surgery. Facet joint infection and paraspinal pyomyositis are being increasingly recognized. Tuberculous spondylitis is the most common granulomatous spondylitis, and the most common spine infection worldwide. It has typical and atypical appearances. Features such as disc space sparing, multilevel subligamentous spread, and large abscesses are suggestive of this diagnosis. Brucellar spondylitis is most commonly seen in endemic regions. Although it can be indistinguishable from tuberculous spondylitis, it generally has a less aggressive appearance. Fungal spondylitis is rare and has a relatively nonspecific imaging appearance. Image-guided sampling is an important part of the diagnostic algorithm in spine infection.

**REFERENCES**


