SPINAL INFECTIONS

Mr. Yagnesh Vellore FRACS
Neurosurgeon and Spine Surgeon
EPIDEMIOLOGY

• 0.2-2 per 10000 hospital admissions

• Bimodal age peak: very young and very old

• M>F

• Immunocompromised more common: HIV/Transplant/Steroid/Diabetic
and nerve blocks, and are estimated to be responsible for 15% of all cases.1,3–7,11,18–23 In general, these infections are acquired either during the invasive procedure itself, or through ascending microorganism from the skin flora, when a device is left in place.24–26 On such catheters, there is a biofilm formation similar to that found on intravascular catheters. Another possible iatrogenic cause of SEA that clinicians should be aware of, is the paraspinal injection of analgesics and steroids (e.g. for local pain therapy).13,27–29 Spinal cord injury leading to neurological impairment is partially caused by direct mechanical compression by the inflammatory mass. Accordingly, there is notable neurological improvement after surgical decompression.30 Studies focussing on the indirect injury caused by vascular occlusion and ischaemia have shown diverging results.31,32 Mechanical compression and vascular occlusion may occur at different phases of the disease and cause additive adverse effects. However, the detailed pathogenesis of spinal cord injury remains poorly characterized.

Table 1  Primary sources of infection in spinal epidural abscess

<table>
<thead>
<tr>
<th>Source of infection</th>
<th>Median (%)</th>
<th>Range (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin and soft tissue</td>
<td>18</td>
<td>7–45</td>
</tr>
<tr>
<td>Urinary tract</td>
<td>10</td>
<td>2–36</td>
</tr>
<tr>
<td>Previous sepsis of unknown origin</td>
<td>8</td>
<td>5–11</td>
</tr>
<tr>
<td>Respiratory tract</td>
<td>5</td>
<td>3–16</td>
</tr>
<tr>
<td>Abdomen</td>
<td>4</td>
<td>2–11</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>3</td>
<td>1–8</td>
</tr>
<tr>
<td>Infected vascular access</td>
<td>2</td>
<td>1–8</td>
</tr>
<tr>
<td>Dental abscess</td>
<td>2</td>
<td>1–11</td>
</tr>
<tr>
<td>Ear, nose and throat</td>
<td>2</td>
<td>&lt;1–11</td>
</tr>
</tbody>
</table>

Table 2 Predisposing conditions in spinal epidural abscess

<table>
<thead>
<tr>
<th>Predisposing condition</th>
<th>Median (%)</th>
<th>Range (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes mellitus</td>
<td>21</td>
<td>15–46</td>
</tr>
<tr>
<td>Abnormality of the vertebral column</td>
<td>17</td>
<td>6–70</td>
</tr>
<tr>
<td>Trauma of the spine</td>
<td>15</td>
<td>5–33</td>
</tr>
<tr>
<td>Intravenous drug use</td>
<td>15</td>
<td>4–37</td>
</tr>
<tr>
<td>Immunosuppressive therapy</td>
<td>12</td>
<td>7–16</td>
</tr>
<tr>
<td>Cancer</td>
<td>7</td>
<td>2–15</td>
</tr>
<tr>
<td>HIV/AIDS</td>
<td>6</td>
<td>2–9</td>
</tr>
<tr>
<td>Alcoholism</td>
<td>5</td>
<td>4–18</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>4</td>
<td>2–13</td>
</tr>
</tbody>
</table>

Predisposing conditions for spinal epidural abscess

A large proportion of SEA patients have at least one predisposing factor (Table 2). Most of these, including diabetes mellitus, intravenous drug use, immunosuppressive therapy, cancer, HIV/AIDS and renal failure are predisposing conditions for any type of severe infection. Spinal abnormalities, such as degenerative joint disease or scoliosis, have been advocated to represent a locus minoris resistentiae. A history of previous spinal trauma is often evident in SEA. Haematoma and disruption of anatomic barriers favour the development of SEA. Alcoholism is found in a relatively high proportion of patients with SEA. Alcohol intoxication predisposes to injury, including spinal trauma, and decreases pain sensitivity, resulting in pressure sores or muscle damage. Moreover, there is a high risk for missing the diagnosis of SEA in this population, because symptoms might be misinterpreted as typical sequelae of alcoholism, such as pancreatitis, peripheral neuritis, and vitamin B12 deficiency.11 The risk of SEA in association with invasive procedures has been estimated for some invasive anaesthetic interventions and ranges from 1:1000 to 1:100 000, depending on the study population, and the location and duration of catheterization.26,33–40 In the case of temporary puncture, the risk of an epidural abscess is very low. Two recently published studies,23,41 each analysing the outcome of 48000 epidural catheters inserted for postoperative analgesia, calculated an SEA incidence of approximately 1:1350. However, if a peri- or epidural catheter is left in place for several days (e.g. for more than 2–4 days), the risk of developing both catheter site infection and epidural abscess increases.

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PATHOGENESIS

• Haematogenous
  Arterial
  Batsons vertebral venous plexus

• Contiguous spread eg from lung to T spine

• Iatrogenic eg LP, epidural anaesthesia, spine surgery
PATHOGENESIS

• Thrombus of metaphyseal artery->AVN->nidus for infection

• Equatorial zone less susceptible

• Endplates and disc more at risk

• Secondary septic thrombosis of epidural veins and resulting epidural abscess

• Sometimes don’t find the whole constellation

• Neurological deficits: compression or ischemia
MICROBIOLOGY

- S. aureus
- Strep species
- Pseudomonas (IVDU)
- TB (3rd world)
- Fungi (immunesuppressed eg cryptococcus in HIV)
- Rarely anaerobes
CLINICAL FINDINGS

- Back pain
- Fever
- Spine tenderness
- Heusner: pain, radiculopathy, weakness, paralysis
- Constitutional symptoms
- Nocturnal/recumbency pain
- Sphincter disturbance
- Neurological deficit
- Thoracolumbar most commonly affected
DIAGNOSIS

- ESR
- CRP: more useful in post-op
- WBC not a reliable marker
- Tuberculin test (not useful when BCG given)
- Test for HIV when high index of suspicion
IMAGING

- Xrays non-specific: help in deformity assessment
- MRI-gold standard
- T1 hypointensity esp marow
- T2 hyperintensity esp disc
- Loss of T2 intranuclear cleft
- Enhancement with gadolinium

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 (black arrow) enhancement 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.
EPIDURAL ABSCESS

- T2 hyper, T1 hypo
- Contrast enhancement
- Peripheral enhancement with central non-enhancement and T2 hyperintensity suggests liquid abscess
- Homogenous enhancement with T2 iso/hypo-intensity suggests solid phlegmon

Diehn 786

• T2 hyper, T1 hypo
• Contrast enhancement
• Peripheral enhancement with central non-enhancement and T2 hyperintensity suggests liquid abscess
• Homogenous enhancement with T2 iso/hypo-intensity suggests solid phlegmon
DIFFERENTIATING FROM DEGENERATION/MODIC 1

**Pyogenic spondylodiscitis: classic imaging**

- **Disc space**: T2 hyperintensity, enhancement, loss of the vacuum sign, as well as endplate irregularity and apparent disc space widening at L4-5 suspicious for phenomon at L4-5.

3 weeks later, she presented with worsening back pain. Lateral radiograph (Fig. 3. A) and T1 postgadolinium (Fig. 4. B) MRI demonstrate findings of spondylodiscitis.

- **Epidural space**: reactive enhancement/venous distention, phlegmon, abscess
- **Paraspinal soft tissues**: ill-defined inflammation, T1 hypo-, T2 hyperintensity, enhancement
- **Adjacent vertebral bodies**: endplate destruction, T1 hypo-, T2 hyperintensity, enhancement
- **Disc space**: T2 hyperintensity, enhancement, some enhancement may be present at the periphery of the disc, which could potentially be confused with an epidural abscess
- **Paraspinal soft tissues**:
  - Abscess
  - Abscess
  - Abscess
  - Abscess

In addition to a lack of clinical features 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, and correlate with pain in at least some patients.

If gadolinium contrast is administered, these abnormal signals may become more apparent and correlate with pain in some patients. If symptoms persist or worsen, surgical decompression may be indicated.

**Differential Diagnosis**

- Degenerative or age-related disc change, more specifically, the Modic type 1, active endplate changes (see Fig. 4. C, D).
- This distinction can be partially blurred due to the overlap of imaging findings.

**Box 1**

- Degenerative disc disease
- Age-related disc change
- Degenerative disc change (see Fig. 4. C, D).

**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 (Fig. 3. A) demonstrates a disc space vacuum phenomenon at L4-5. 3 weeks later, she presented with worsening back pain. Lateral radiograph (Fig. 3. B) demonstrates loss of the vacuum sign, as well as endplate irregularity and apparent disc space widening at L4-5 suspicious for phenomon at L4-5.
CT/NUCLEAR MEDICINE

- CT myelogram: pitfall- iatrogenic meningitis
- Nuclear med: technetium-blood flow
  - gallium-Fe binding
  - labelled WBC
- FDG PET
- Indium labelled biotin
BACTERIOLOGICAL DX

• Blood cultures: -ve in 40-75%

• Confounded by Abx

• Biopsy: CT guided vs open (80% +ve)

• Consider TB/fungi in –ve cases
OTHER IX

- HepB/C IVDU
- HIV
- TOE
- Fundoscopy
- Nail bed
- Retropharyngeal/Psoas abscess
DDX

- pyogenic arthritis of the hip
- septic or autoimmune sacroiliitis
- pyelonephritis
- primary psoas abscess
- autoimmune spondylitis
- spinal trauma
- osteoporotic compression fractures
- spinal epidural hematoma
- spontaneous spinal subarachnoid hemorrhage
- leptomeningeal metastatic disease
MANAGEMENT

- Antibiotics (iv 6/52) and immobilization
- Surgery for any neurological deficit
- Approach depends on collection site (ventral v dorsal, C/T/L spine) and consistency (liquid v phlegmon)
- Aggressive debridement
- Mild deficit such as radiculopathy may be closely observed
- Surgery: Failed medical Rx
  - Chronic pain
  - Instability
- Instrumentation/grafting
- Bracing
- f/u: serial CRP/ESR and clinical; routine MRI not indicated
Table 1. Common Diagnostic and Therapeutic Pitfalls and Recommended Approaches.

<table>
<thead>
<tr>
<th>Pitfall</th>
<th>Recommendation</th>
</tr>
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<tbody>
<tr>
<td>Ordering imaging studies of an area that is not the site of epidural infection</td>
<td>Clinically assess patients for spinal tenderness and level of neurologic deficit to more accurately identify the region to be imaged.</td>
</tr>
<tr>
<td>Identifying only one of multiple nonadjacent epidural abscesses</td>
<td>Suspect the presence of other undrained abscesses if bacteremia persists or neurologic level changes after surgery.</td>
</tr>
<tr>
<td>Ascribing all clinical and laboratory findings to vertebral osteomyelitis</td>
<td>Determine whether osteomyelitis is associated with epidural abscess, particularly if a neurologic deficit is evident.</td>
</tr>
<tr>
<td>Being unable to adequately evaluate sensorimotor function in patients with altered mental status</td>
<td>Check for depressed reflexes and bladder or bowel dysfunction, which can indicate spinal cord injury.</td>
</tr>
<tr>
<td>Asking nonphysicians who may not appreciate the urgency of the case to order consultations for patients with suspected or documented epidural abscess</td>
<td>Directly communicate with consultants to ensure timely diagnosis and treatment.</td>
</tr>
<tr>
<td>Surgically managing a spinal stimulator–associated epidural abscess by removing only the implant</td>
<td>Decompress the abscess to preserve neurologic function and remove the implant to increase the likelihood of curing the infection.</td>
</tr>
<tr>
<td>Medically treating <em>S. aureus</em> bacteremia without attempting to identify the source</td>
<td>Consider a spinal source of infection if clinically indicated.</td>
</tr>
</tbody>
</table>
How- ever, in these studies, the group of patients receiving antibiotics alone had no or minimal neurologic impairment or smaller abscesses, and in some of these patients, neurologic deterioration occurred despite the use of appropriate antibiotics. The true index of the success of nonsurgical therapy is difficult to discern both because cases may have been selectively reported and because unsuccessful attempts at conservative management are rarely reported once a decompressive laminectomy is performed. Figure 4 shows an algorithm for treating patients with diagnosed spinal epidural abscess. In clinical scenarios in which decompressive laminectomy is declined by the patient, contraindicated because of high operative risk, unlikely to reverse paralysis that has existed for more than 24 to 36 hours, or considered impractical because of panspinal infection, patients may be treated medically. Patients who are neurologically intact may also qualify for nonsurgical therapy if the microbial cause is identified and the patients’ clinical condition is closely monitored. Although controversial, this approach may be reasonable especially when the radiologic epidural abnormality and the symptoms can be explained by finding (including postoperative changes) that the inflammation is not caused by a true abscess. Antibiotic therapy must be guided by the results of blood cultures or a CT-guided needle aspiration of the abscess. Although emergency decompressive laminectomy is not indicated in patients with paralysis that lasts longer than 24 to 36 hours, this surgery may still be needed to treat the epidural infection and control sepsis. Because it is impractical to perform decompressive laminectomy along the whole spine in patients with panspinal epidural abscess (Fig. 5), the physician may want to consider less extensive surgery, such as a limited laminectomy or laminotomy with cranial and caudal insertion of epidural catheters for drainage and irrigation. Pending the results of cultures, empirical antibiotic therapy should provide coverage against staphylococci (usually with vancomycin to cover MRSA) and, because of the potentially serious consequences, gram-negative bacilli (potentially with a third- or a fourth-generation cephalosporin, such as ceftazidime or cefepime, respectively), particularly in the presence of documented or suspected gram-negative bacterial infection of the abscess.

**Figure 4. Management of Spinal Epidural Abscess.**
Debridement in the context of the overall and detailed health considerations involved in this process. The reduction of bed rest period. This issue must be viewed in the context of the spine, stabilization of the spinal column and restoration and maintenance of the sagittal alignment. The combination of radical debridement and instrumentation can help the body to combat the infection rather than to address the source of the disease.

In the era of the 1990s, internal fixation started gaining some acceptance in the treatment of infectious vertebral lesion. However, it was not until the 1990s of the last century, that posterior instrumentation was used in the management of pyogenic spinal infections. A number of reports had implicated that conservative treatment was the most commonly adopted therapy for the treatment of spinal tuberculosis represented the merits such as shorter operation time, less blood loss, and shorter convalescence period bridging the two surgeries have been emphasized by several researchers. The importance of immobilization for the suppression of pain, epidural abscesses and neurologic deficit despite the provision of long-term antibiotic therapy and other circumstances.

The well-known Hodgson's Hong Kong procedure reported a series of pyogenic vertebral osteomyelitis treated with debridement and instrumentation. Since then, radical debridement and autogenous strut-graft have become the golden standard for the treatment of spinal tuberculosis represented the effectiveness of the dual-stage approach to the management of vertebral osteomyelitis. The two-stage approach was also commonly used for pyogenic vertebral osteomyelitis because of the relatively high infection rate.

Table 1 Summary of most recent clinical series of pyogenic spondylodiscitis treated with debridement and instrumentation

<table>
<thead>
<tr>
<th>References</th>
<th>No. of patients</th>
<th>Average age (years)</th>
<th>Pre-op antibiotic duration (weeks)</th>
<th>No. of patients</th>
<th>Bone graft</th>
<th>Instrumentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage</td>
<td>Acute</td>
<td>Subacute</td>
<td>Chronic</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Approach</td>
<td>Anterior</td>
<td>Posterior</td>
<td>Combined</td>
<td></td>
<td></td>
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<tr>
<td>Masuda et al. [49]</td>
<td>5</td>
<td>63.8</td>
<td>1.5–2.5</td>
<td>3</td>
<td>2</td>
<td>0</td>
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<tr>
<td>Korovessis et al. [36]</td>
<td>17</td>
<td>54.4</td>
<td>N/A</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Nather et al. [56]</td>
<td>12</td>
<td>62.5</td>
<td>N/A</td>
<td>8</td>
<td>3</td>
<td>1</td>
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<tr>
<td>Dimar et al. [17]</td>
<td>42</td>
<td>60</td>
<td>N/A</td>
<td>0</td>
<td>0</td>
<td>42</td>
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<tr>
<td>Fayazi et al. [22]</td>
<td>11</td>
<td>56.3</td>
<td>13(0–60)</td>
<td>0</td>
<td>0</td>
<td>10</td>
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<tr>
<td>Mann et al. [47]</td>
<td>24</td>
<td>63</td>
<td>0–5 days</td>
<td>24</td>
<td>0</td>
<td>6</td>
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<tr>
<td>Lee et al. [42]</td>
<td>30</td>
<td>56.7</td>
<td>N/A</td>
<td>7</td>
<td>6</td>
<td>17</td>
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<tr>
<td>Fukuta et al. [25]</td>
<td>8</td>
<td>63.5</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>8</td>
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<tr>
<td>Liljenqvist et al. [44]</td>
<td>20</td>
<td>68</td>
<td>N/A</td>
<td>0</td>
<td>0</td>
<td>20</td>
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<tr>
<td>Hee et al. [32]</td>
<td>21</td>
<td>57</td>
<td>N/A</td>
<td>11</td>
<td>0</td>
<td>10</td>
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<tr>
<td>Przybylski et al. [65]</td>
<td>17</td>
<td>58.7</td>
<td>IV: 0–8 PO: 0–20</td>
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<td>11</td>
<td>0</td>
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<td>Schuster et al. [71]</td>
<td>47</td>
<td>49.3</td>
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<td>7</td>
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<td>40</td>
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<tr>
<td>Faraj et al. [21]</td>
<td>31</td>
<td>55.6</td>
<td>N/A</td>
<td>1</td>
<td>0</td>
<td>30</td>
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<tr>
<td>Total</td>
<td>287</td>
<td>42</td>
<td>17</td>
<td>195</td>
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CONTROVERSIES

- Hodgson 1956: bone graft
- Kostuik 1983: instrumentation
- No evidence to contraindicate instrumentation in the setting of infection
- Autograft vs allograft
- Titanium (porous nature allows abx delivery and vascular tissue attachment) vs stainless steel (?) glycocalyx for bacteria
- Controversial
- BMP- not FDA approved
OUTCOME: POORER PROGNOSIS

- older patients
- sepsis
- neurological deficits of longer than 72 hours duration
- significant compression of the spinal cord on imaging studies
- immunocompromised
IATROGENIC INFECTIONS

• 2%
• Immunosuppression, diabetes, steroid, malnutrition, neoplasm, radiation, intercurrent infection
• ?Bovie monopolar
• ?muscle retraction/ischemia
• Superficial vs deep: fascia
• ?r/o instrumentation in deep infection
PAEDIATRIC

- Frequent bacteremia
- Profuse anastomoses b/w intraosseous spinal arteries
- Disc (retains blood supply)/endplate more affected
- Child with fevers refuses to bear weight
- Clinical and radiographic disease in children may often be milder than that seen in adults
- Abx + immobilization usually adequate
- Surgery rare
TB: POTTS DISEASE

- Lungs: haematogenous/contiguous
- Indolent fashion
- Involves posterior elements
- Spares disc
- Deformity w/o neurological compromise
- Difficult to distinguish from neoplasm
TULI GRADING

- stage I: no weakness but there is clumsiness of gait and a suggestion of upper motoneuron signs
- stage II: weakness and clear upper motoneuron signs, but the patient is able to walk.
- stage III: bedridden because of total muscle weakness and maintains signs of upper motoneuron paraplegia, less than 50% sensory loss
- stage IV: complete motor weakness, greater than 50% loss of sensation, loss of bowel/bladder control, or any combination of these findings, as well as probably flaccid paraplegia and possibly flexor spasm.
on pathology, and confirms the lytic nature of the L3 spinous process lesion. The biopsy yielded caseating granulomatous inflammation.

A 42 year-old man was referred for possible metastatic disease. Sagittal T1- weighted (see Fig. 6A–D). There are multiple bony spinal lesions, including in the vertebral bodies (see Figs. 9A–D). Spread in the epidural space and partially effaced the thecal sac (see Fig. 10C). In addition to subligamentous spread, atypical, several of these and some may also occur (see Fig. 10B, C).

Imaging evaluation, differential diagnosis

Table 2

<table>
<thead>
<tr>
<th>Classic:</th>
<th>Atypical:</th>
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<tbody>
<tr>
<td>Similar to pyogenic spondylodiscitis</td>
<td>Disc sparing, with either single or multilevel bony involvement only</td>
</tr>
<tr>
<td>Disc space involvement less severe</td>
<td>Multilevel involvement, contiguous or skip lesions</td>
</tr>
<tr>
<td>Large paraspinal abscess, smooth wall, ± calcifications</td>
<td>Vertebra plana</td>
</tr>
<tr>
<td>Subligamentous spread</td>
<td>Posterior element involvement</td>
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<td></td>
<td>Panvertebral involvement</td>
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</tbody>
</table>

Although tuberculosis is far more common than in the anterior paravertebral regions, it is more commonly involved than in pyogenic spondylodiscitis. The MRI findings of brucellosis may be indistinguishable from those of tuberculous spondylitis. The MRI findings of brucellosis may be indistinguishable from those of tuberculous spondylitis. The MRI findings of brucellosis may be indistinguishable from those of tuberculous spondylitis. The MRI findings of brucellosis may be indistinguishable from those of tuberculous spondylitis.

Background

Brucellar Spondylitis

Although tuberculosis is far more common than in the anterior paravertebral regions, it is more commonly involved than in pyogenic spondylodiscitis. The MRI findings of brucellosis may be indistinguishable from those of tuberculous spondylitis. The MRI findings of brucellosis may be indistinguishable from those of tuberculous spondylitis. The MRI findings of brucellosis may be indistinguishable from those of tuberculous spondylitis. The MRI findings of brucellosis may be indistinguishable from those of tuberculous spondylitis.

Box 2

Imaging clues: tuberculous spondylitis

<table>
<thead>
<tr>
<th>Classic:</th>
<th>Atypical:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Similar to pyogenic spondylodiscitis</td>
<td>Disc sparing, with either single or multilevel bony involvement only</td>
</tr>
<tr>
<td>Disc space involvement less severe</td>
<td>Multilevel involvement, contiguous or skip lesions</td>
</tr>
<tr>
<td>Large paraspinal abscess, smooth wall, ± calcifications</td>
<td>Vertebra plana</td>
</tr>
<tr>
<td>Subligamentous spread</td>
<td>Posterior element involvement</td>
</tr>
<tr>
<td></td>
<td>Panvertebral involvement</td>
</tr>
</tbody>
</table>
SPINAL TB MX

- RIPE abx and immobilization
- Surgery for neurological deficit, instability or deformity
- Jain et al: >= 2 column damage
- MRI evidence of edema or myelitis within the spinal cord and compressive lesion is predominantly fluid in the extradural space will respond well to nonoperative therapy
- Extradural compression from a lesion that appears to be mostly granulation or caseous tissue, one that compresses the cord circumferentially, cord edema, myelitis, or myelomalacia are more likely to be candidates for early surgical intervention
- Poor prognosis: paralysis lasting longer than 6 months, late-onset paralysis with inactive disease and significant deformity, paralysis as a result of vascular injury to the spinal cord, atrophic-appearing spinal cord seen on MRI