

## CASE REPORT

# Rare cause of back pain: *Staphylococcus aureus* vertebral osteomyelitis complicated by recurrent epidural abscess and severe sepsis

Louise Dunphy,<sup>1</sup> Shabnam Iyer,<sup>2</sup> Christopher Brown<sup>1</sup>

<sup>1</sup>Department of Trauma and Orthopaedic Surgery, Royal Berkshire NHS Foundation Trust, Reading, UK  
<sup>2</sup>Department of Microbiology, Royal Berkshire NHS Foundation Trust, Reading, UK

**Correspondence to**  
 Dr Louise Dunphy,  
 dunphyimb@yahoo.com

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## SUMMARY

An epidural abscess represents a rare acute medical emergency, with a reported incidence of 2.5/10 000 hospital admissions annually. The clinical features include fever, spinal pain, radiating nerve root pain and leg weakness. When sepsis is present, prompt recognition is required to initiate appropriate antimicrobial therapy and surgical decompression. We present the case of a man aged 68 years presenting to the emergency department with a 3-day history of fever, low back, right hip and leg pain. He was hypoxic, tachycardic and hypotensive. He required intubation and ventilation. An MRI spine confirmed a posterior epidural abscess from T12 to L4. Blood cultures revealed *Staphylococcus aureus*. He started treatment with linezolid and underwent incision and drainage. He remained septic and 8 days later, a repeat MRI spine showed a peripherally enhancing posterior epidural collection from L2/L3 to L4/L5, consistent with a recurrent epidural abscess. Further drainage was performed. He developed bilateral knee pain requiring washout. His right knee synovial biopsy cultured *S. aureus*. He continued treatment with linezolid for 6 weeks until his C reactive protein was 0.8 ng/L. He started neurorehabilitation. 10 weeks later, he became feverish with lumbar spine tenderness. An MRI spine showed discitis of the L5/S1 endplate. A CT-guided biopsy confirmed discitis and osteomyelitis. Histology was positive for *S. aureus* and he started treatment with oral linezolid. After 19 days, he was discharged with 1 week of oral linezolid 600 mg 2 times per day, followed by 1 further week of oral clindamycin 600 mg 4 times daily. This case report reinforces the importance of maintaining a high clinical suspicion, with a prompt diagnosis and combined medical and surgical treatment to prevent adverse outcomes in this patient cohort. With spinal surgical services centralised, physicians may not encounter this clinical diagnosis more often in day-to-day hospital medical practice. The unique aspect of this case is the persistence and then the recurrence (despite 6 weeks of antimicrobial therapy and a second debridement) of *S. aureus* infection. Furthermore, the paucity of clinical recommendations and the controversy regarding the adequate duration of antimicrobial therapy are notable features of this case.

he reported decreased urinary frequency and acute-onset confusion. He was a non-smoker and consumed 8 units of alcohol weekly. His medical history included hypercholesterolaemia. He was taking simvastatin 20 mg daily. Observations were as follows: RR 14, SpO<sub>2</sub> 96% on 12 L FiO<sub>2</sub>, BP 90/57 mm Hg, HR 110 bpm and temperature 38.6°C. Respiratory examination confirmed good air entry bilaterally and breath sounds were vesicular. His GCS was 14/15 (E4M6V4). Upper limb examination confirmed normal power, tone and reflexes, with intact sensation and proprioception. Lower limb examination confirmed normal tone, reduced power of hip flexors (3/5) and reduced sensation from L2 to L4. Rectal examination demonstrated normal anal tone and sensation. Mild nuchal rigidity was elicited.

## INVESTIGATIONS

Haematological investigations confirmed an elevated C reactive protein (CRP) (311.3 mg/L) and an acute kidney injury. His INR was 2.7 mmol/L, corrected with 10 mg of vitamin K intravenously (table 1).

A venous blood gas revealed: pH 7.341, base excess -3.3 mmol/L, HCO<sub>3</sub> -21.4 mmol/L and lactate 2.7 mmol/L. A chest radiograph showed atelectasis bilaterally within the lung bases (figure 1). An unenhanced CT head showed no evidence of intracranial bleed, extracerebral collection or focal mass lesion (figure 2). He started treatment with acyclovir and ceftriaxone. However, an MRI head with contrast displayed no evidence of leptomeningeal disease (figure 3). He received intravenous teicoplanin and gentamicin for sepsis of unknown origin. HIV and hepatitis serology were negative. He remained feverish, tachycardic, hypotensive and hypoxic. He was admitted to the Department of Intensive Care Medicine requiring intubation, ventilation and inotropic support. An MRI spine demonstrated a posterior epidural collection extending from T12 to L4 (figure 4), with mixed signal intensity on STIR and T2-weighted images, as well as low to intermediate signal intensity on T1-weighted imaging. The lesion favoured the right posterolateral aspect of the epidural space cranially and more caudally the left posterolateral epidural space. Moulding of the adjacent posterolateral margin of the thecal sac, most pronounced at the L2-L3 level, measuring 17×7 mm in the axial section, with subtle rim enhancement was noted. He was reviewed urgently by the Spinal Team.

## CASE PRESENTATION

A man aged 68 years, born and resident in the UK presented to the emergency department with a 3-day history of bilateral leg weakness, fatigue, fever, lower back and right hip pain. In addition,



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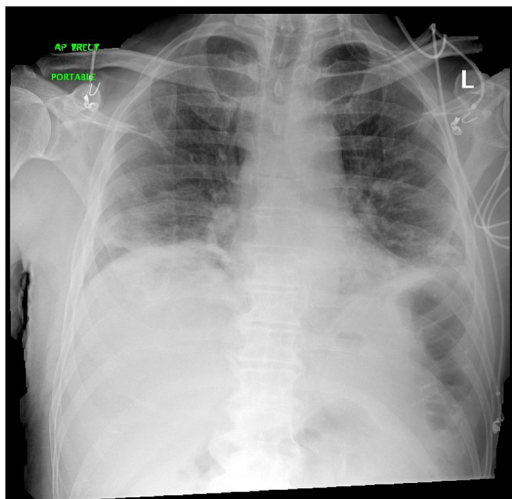
**Table 1** Haematological investigations showed an elevated C reactive protein, deranged liver function tests and an acute kidney injury

Hb 160 g/L	Magnesium 0.78 mmol/L	Urea 14.0 mmol/L
WCC $10.70 \times 10^9/L$	Albumin 39 g/L	Creatinine 150 $\mu\text{mol/L}$
Neutrophils $9.86 \times 10^9/L$	Alkaline phosphatase 87 IU/L	eGFR 40 mL/min/L
Platelets $154 \times 10^9/L$	ALT 315 IU/L	Na 134 mmol/L
INR 2.7 mmol/L	CRP 311.3 mg/L	K 4.8 mmol/L

On microbiology advise, he stopped treatment with gentamicin and teicoplanin and started treatment with meropenem (12 days), clarithromycin (2 days) and clindamycin (8 days). His methicillin-resistant *Staphylococcus aureus* (MRSA) screening swabs were negative.

### TREATMENT

Two days postadmission, he underwent emergency washout of his epidural abscess via a midline incision to enter the spinal canal at the point of most marked stenosis, L2–L3 (figure 5). A partial laminectomy with flavectomy to decompress the dura was performed. Blood-stained pus was evacuated and copious saline lavage carried out. *Staphylococcus aureus* was isolated from the aerobic and anaerobic culture bottles. Pus and wound swabs cultured *S. aureus* as well as tissue from the ligamentum flavum. Postoperatively, he remained septic, with no reduction in his inflammatory markers and an elevated white cell count ( $18.9 \times 10^9/L$ ) with a neutrophilia ( $14 \times 10^9/L$ ). On microbiology advise, he started treatment with intravenous linezolid. His urinary *Legionella pneumophila* and Pneumococcal antigens were negative. He developed transaminitis and thrombocytopenia. An ejection systolic murmur was audible on auscultation, but a transoesophageal echocardiogram showed no evidence of infective endocarditis. His CRP remained elevated at 319.4 g/L, with a white cell count of  $12.7 \times 10^9/L$ . Repeat blood cultures 48 and 72 hours after starting antimicrobial therapy showed no growth. A repeat MRI spine 6 days later showed a discrete peripherally enhancing posterior epidural collection from L2/L3 to L4/L5, consistent with a recurrent epidural abscess, larger than the preoperative MRI. Severe distortion and compression of the

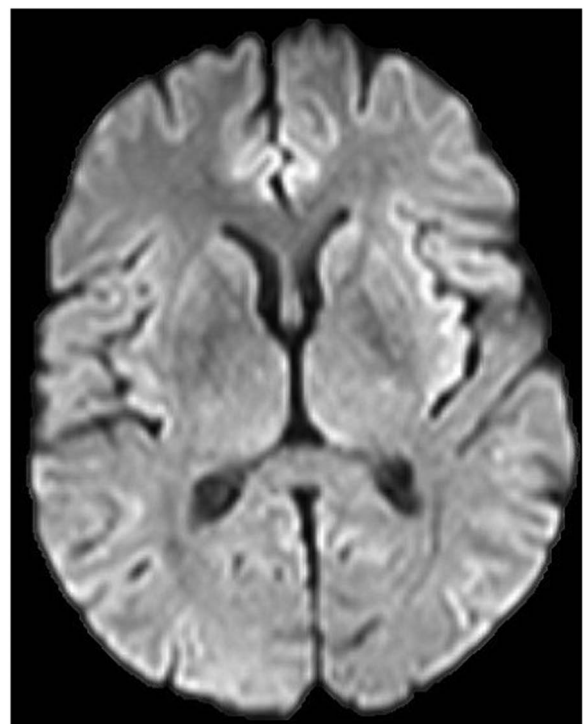


**Figure 1** Chest radiograph showed atelectasis within the lung bases bilaterally.

cauda equine was noted (figure 6). Furthermore, there was evidence of discitis in the L2/L3 and L5/S1 discs, with increasing endplate oedema at these levels, particularly at L5/S1 with extension of the abscess into the paraspinal soft tissues. Eight days postadmission, he returned to theatre, the old wound was



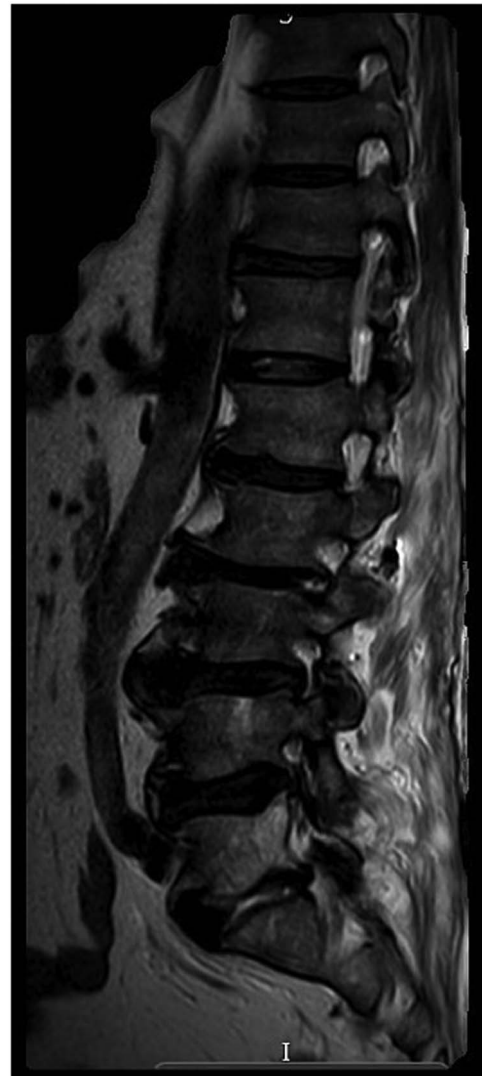
**Figure 2** Unenhanced CT head. An unenhanced CT head showed no evidence of intracranial bleed, extracerebral collection or focal mass lesion.



**Figure 3** MRI head with contrast. An MRI head with contrast displayed no evidence of leptomeningeal disease.



**Figure 4** MRI whole spine with contrast. An MRI spine demonstrated normal alignment and vertebral body height. A posterior epidural collection extending from T12 to L4 was observed.



**Figure 6** MRI whole spine with contrast. An epidural abscess from L2/L3 to L4/L5 causing significant central canal stenosis and distortion of the cauda equine was observed as well as progressive discitis at L2/L3 and L5/S1.



**Figure 5** Mobile image intensifier lumbar spine demonstrated the epidural abscess intraoperatively.

opened and pus was evident superficially. The laminectomy site was reopened and pus was visualised adjacent to the dura. A further midline incision was made at L3–L4. A right-sided L5–S1 laminectomy was performed and the dura was unremarkable. Wound and pus swabs cultured *S. aureus*. Macroscopically, the ligament adjacent to the spinal abscess, comprised a brown fibrous piece of tissue, measuring 20×10×7 mm. Histological examination identified microscopic bony fragments with marrow, fibro-fatty tissue and skeletal muscle, with no evidence of significant inflammation. His spinal tissue revealed a scanty growth of *S. aureus*, sensitive to clindamycin, linezolid and flucloxacillin. He continued treatment with linezolid intravenously.

#### OUTCOME AND FOLLOW-UP

He failed to wean off the ventilator requiring a percutaneous tracheostomy. Two weeks later, he was decannulated successfully. He remained feverish (temperature 38.7°C), and clinical examination confirmed septic arthritis of his knees. He underwent bilateral arthroscopic wash out of both his knees. Straw-coloured fluid was aspirated from his right knee. A

**Table 2** The patient's linezolid was stopped after 6 weeks as his C reactive protein was 0.8 mg/L and his observations were stable

Hb 85 g/L	CRP 0.8 mg/L	Na 138 mmol/L
WCC $50 \times 10^9/L$	Albumin 30 g/L	K 5.2 mmol/L
Neutrophils $6.98 \times 10^9/L$	ALP 136 IU/L	Urea 5.2 mmol/L
Platelets $437 \times 10^9/L$	ALT 23 IU/L	Creatinine 58 $\mu\text{mol/L}$

synovectomy and a chondroplasty was performed. His left knee revealed a serous effusion with no evidence of infection. His right knee synovial fluid showed chronic inflammation, with acute inflammatory cells. No crystals were identified. His knee fluid cultured Gram-positive cocci in clumps, further identified as *S. aureus*. After 29 days in intensive care unit, he was stepped down to the Orthopaedic Ward, requiring neurorehabilitation for his critical illness polyneuropathy. He required a second arthroscopic washout of his right knee. The synovial biopsy comprised fibroconnective tissue with fibropurulent exudate on its surface. The infiltrate included numerous polymorph neutrophils in keeping with a bacterial infection. He continued treatment with linezolid for 6 weeks until his CRP was 0.8 mg/L (table 2).

He was subsequently discharged to the Neurorehabilitation Department. Four weeks later, he was readmitted with non-specific symptoms of general malaise, fatigue and lower back pain. Neurological examination demonstrated tenderness on palpation of L5–S1. Haematological investigations revealed a leucocytosis ( $14.20 \times 10^9/L$ ) with a neutrophilia ( $8.53 \times 10^9/L$ ) and a CRP of 252 g/L. A chest radiograph demonstrated some linear atelectasis on the left side at the lung base. A CT thorax, abdomen and pelvis revealed normal lungs and pleura, with no hilar or mediastinal lymphadenopathy. His liver, gallbladder, pancreas, spleen, adrenals, kidneys, small and large bowels were unremarkable. There was no evidence of intra-abdominal or pelvic lymphadenopathy. Destruction of the L5/S1 endplate consistent with discitis (figure 7) as well as inflammatory

**Figure 7** CT thorax, abdomen and pelvis showed destruction of the L5/S1 endplate consistent with known discitis.

paravertebral changes were shown. An MRI lumbar and sacral spine with contrast showed increased bone oedema within the inferior endplate of L2 (figure 8). Signal change within the L2–L3 and L5–S1 intervertebral discs persisted in keeping with ongoing infective discitis. The previously demonstrated SEA lying between L3 and L5 appeared to have resolved. Extensive areas of pathological enhancement were demonstrated within the interspinous ligaments between L3 and L5, the inferior vertebral endplates of L2, L5 and S1 vertebral bodies, the sacral canal and the perivertebral soft tissues mainly at L5 and S1 levels, in keeping with an ongoing infectious process. He underwent a CT-guided biopsy, using an 11 G core biopsy of his right-sided L5–S1 disc (figure 9). A second needle was inserted and multiple 18 G core biopsies were obtained of the disc itself. Macroscopically, the biopsy specimen comprised pieces of fibrocartilage from an intervertebral disc, focally infiltrated by polymorph neutrophils with some vascularisation. Fragments of necrotic material and collections of polymorph neutrophils admixed with macrophages were noted. The histological features confirmed an acute discitis. The L5/S1 biopsy consisted of

**Figure 8** MRI whole spine with contrast demonstrated extensive areas of pathological enhancement within the interspinous ligaments between L3 and L5, the inferior vertebral endplates of L2, L5 and S1 vertebral bodies, the sacral canal and the perivertebral soft tissues mainly at L5 and S1 levels, in keeping with an ongoing infectious/inflammatory process.



**Figure 9** CT-guided biopsy of L5–S1. He underwent a CT-guided biopsy, using an 11 G core biopsy of his right-sided L5–S1 disc.

a core of largely necrotic lamellar bone with fibrosis in the marrow space, suggestive of osteomyelitis. The specimens cultured a scanty growth of *S. aureus*, sensitive to flucloxacillin, erythromycin, clindamycin, linezolid and tetracycline. On microbiology advice, he started treatment with oral linezolid, 600 mg two times per day. After 19 days, he was discharged with 1 week of oral linezolid, followed by 1 week of oral clindamycin 600 mg four times daily. He remains committed to his neurorehabilitation.

## DISCUSSION

An epidural abscess is a rare suppurative infection of the central nervous system, first reported in 1761 by Sir Percival Pott, an English surgeon, who also described tuberculosis of the spine (Pott's disease).<sup>1</sup> Until the 1980s, it accounted for 0.2–1.2/10 000 hospital admissions annually, with the current annual incidence estimated to be 2.5–3/10 000 hospital admissions.<sup>2–3</sup> This increase can partly be explained by an ageing population with multiple comorbidities, spinal abnormalities, anaesthetic interventions and enhanced imaging modalities. It has a male predilection and a peak incidence in the fifth to seventh decade, with a reported death of 2–20%. Risk factors include immunosuppression, steroids, epidural catheter placement, paraspinal injections, intravenous drugs, HIV, cancer and chronic renal failure. It is postulated that trauma may result in vertebral haematoma formation, thus providing a nidus for infection. Studies have reported diabetes as a risk factor in 18–54% of cases, with a preponderance of lower thoracic and lumbar abscesses.<sup>4–5</sup> The initial manifestations of SEA are non-specific, with the classical diagnostic triad of fever in 50%, spinal pain and neurological deficits, including motor weakness, sensory change, bladder or bowel dysfunction and paralysis, present in a small proportion.<sup>6</sup> Admittedly, in the emergency department, SEA is not always considered, as neurological symptoms may not always be apparent in the early stages.<sup>6</sup> With cervical lesions, a history of neck stiffness might be reported and patients may present with symptoms mimicking other pathologies such as pancreatitis or heart disease. When sepsis dominates the clinical presentation, a high index of clinical suspicion is required to discern the neurological symptoms. In 1948, Heusner<sup>7</sup> summarised the clinical features into four stages, ranging from non-specific symptoms in Stage I to paralysis in Stage IV (box 1).

Haematological investigations demonstrate leucocytosis with polymorphonuclear predominance, an elevated erythrocyte sedimentation rate and hyponatraemia.<sup>8</sup> Blood cultures may identify the infecting organism, although they are negative in 40% of

### Box 1 Stages according to the clinical progression of spinal epidural abscess

#### Stage clinical signs

- I. Back pain, fever and tenderness
- II. Radicular pain, nuchal rigidity, neck stiffness, reflex changes
- III. Sensory abnormalities, motor weakness, bowel and bladder dysfunction
- IV. Paralysis

cases. Lumbar puncture should not be routinely performed. A high index of clinical suspicion is required in those individuals intoxicated with alcohol, as symptoms might be misinterpreted as sequelae of alcohol. Sendi *et al*<sup>6</sup> describe a SEA as a collection of pus or inflammatory granulation tissue between the dura mater and the overlying vertebral column. Its pathophysiology includes haematogenous spread of bacteria from a cutaneous (vertebral body, psoas muscle) or mucosal source (dental abscess, furuncle, pharyngitis). Skin, soft tissue, urinary and respiratory tract infections are frequent primary sources of haematogenous seeding. The direct spread of infection into the epidural space from discitis and vertebral osteomyelitis have also been described.<sup>9–10</sup> The majority of SEA are primarily located in the posterior aspect of the spinal cord, with anterior SEAs usually occurring below L1.<sup>6</sup> It is important to consider iatrogenic causes such as spinal surgery, epidural catheter placement and nerve block injections. MRI is the imaging modality of choice, with abscesses demonstrating fluid equivalent signal intensity on T2-weighted images with rim enhancement and a hypointense centre. MRI is the most sensitive and specific test for the detection of vertebral osteomyelitis.<sup>10</sup> MRI findings may also correlate with outcome; in a study of 18 patients, central stenosis of >50% and an abscess length of >3 cm were associated with a worse outcome.<sup>11</sup> A large variety of pathogens are causative, including mycobacteria, fungi and parasites, but *S. aureus* is the most frequently encountered organism, occurring in 57–93%, followed by *Streptococcus* (18%) and Gram-negative bacilli (*Escherichia coli*, *Klebsiella* spp., *Pseudomonas aeruginosa*).<sup>12</sup> In the UK, around 12 500 cases of *S. aureus* bacteraemia (SAB) are reported annually, associated with significant mortality and morbidity, including vertebral osteomyelitis, with infection resulting from haematogenous seeding of the vertebral bodies by arterial or venous vessels.<sup>13</sup> Fever is common with tenderness over the involved vertebrae and neurological deficits in 15–20%. The Waldvogel classification system divides osteomyelitis into haematogenous, contiguous and chronic complex disease states<sup>14</sup> (box 2). *Haemophilus parainfluenzae*, *Brucella* and *Actinomyces israeli* are among other isolates described.<sup>15</sup> It is important to consider other

### Box 2 The Waldvogel classification of osteomyelitis

#### Waldvogel classification system for osteomyelitis

- ▶ Haematogenous osteomyelitis
- ▶ Osteomyelitis secondary to contiguous focus of infection
- ▶ No generalised vascular disease
- ▶ Generalised vascular disease
- ▶ Chronic osteomyelitis (necrotic bone)

pathologies presenting with back pain, fever and spinal tenderness, including tuberculosis, degenerative disease, metastatic tumours, vertebral discitis, osteomyelitis, meningitis, neurological disease or herpes zoster. Early surgical decompression and drainage, with prolonged antibiotic therapy, is the mainstay of treatment. However, indications for percutaneous drainage of abscesses and for surgical intervention in patients with *S. aureus* haemolytic vertebral osteomyelitis have not been standardised.<sup>16</sup> Antimicrobial therapy should be guided by culture and sensitivity (positive in 60%) as well as empirical therapy, with several authors advocating CT-guided biopsy to further delineate treatment. Rapid surgical intervention is not only needed to reduce neurological damage, but also for controlling sepsis. Conservative therapy with antibiotics alone might be required in those individuals posing a high anaesthetic risk. There is a paucity of uniform recommendations about antimicrobial treatment duration and it remains controversial with no prospective, randomised, double-blind clinical trials to date. Several studies suggest a total duration of therapy of between 4 and 16 weeks with resolution of the SEA generally achieved after 4–6 weeks. In the case of concomitant vertebral osteomyelitis, parenteral antibiotics are given for 6–8 weeks.<sup>3</sup> In 2015, Professor Bernard *et al*<sup>17</sup> performed an open label, non-inferiority, randomised, controlled trial and suggested that the standard antibiotic treatment duration could be 6 weeks. The nucleus pulposus and the inner two-thirds of the annulus fibrosis of normal intervertebral discs are avascular; therefore, the penetration of intravenous antibiotics into intervertebral discs depends on passive diffusion.<sup>18</sup> Clindamycin, vancomycin and teicoplanin have been shown to penetrate into rabbit nucleus pulposus, the anatomy and biochemical characteristics of the aforementioned is similar to that of humans.<sup>19–21</sup> Clindamycin has particularly good bone penetration, attaining a high bone-to-serum ratio. Vancomycin has excellent penetration into the bones of experimental animals.<sup>22</sup> Tai *et al* and Vaverka and Petzelova have shown that gentamicin can penetrate diseased intervertebral discs.<sup>23</sup> Pharmacokinetic studies have shown that non- $\beta$  lactams such as clindamycin, aminoglycosides and glycopeptides achieve therapeutic concentrations in discs, but  $\beta$  lactams such as penicillin and cephalosporins do not.<sup>24</sup> The British Society for Antimicrobial Chemotherapy concurred that most of the published studies on the use of antibiotics in spinal surgery are retrospective and that the only absolute requirement of a drug selected for use in patients undergoing procedures involving disc penetration is that it possesses *in vitro* activity against *Staphylococci*. Linezolid has *in vitro* activity against methicillin-susceptible *S. aureus* (MSSA) and MRSA, with clinical activity confirmed in nosocomial pneumonia, ventilator-associated pneumonia, complicated skin and soft tissue infections and MRSA infections, such as staphylococcal and vancomycin-resistant enterococcal osteomyelitis.<sup>25</sup> Other surgeons choose an antibiotic based on their own favourable experience.<sup>26</sup> Antibiotics that can be used for the treatment of MSSA bacteraemia include the penicillinase-resistant semisynthetic penicillins, such as flucloxacillin, first-generation cephalosporins, such as cefazolin and the cyclic lipopeptide daptomycin.<sup>27</sup> Leder *et al* demonstrated the clinical efficacy of continuous-infusion flucloxacillin in serious *Staphylococcal* sepsis in 20 patients, with a clinical and microbiological cure achieved for 82%; Mehtar *et al* demonstrated clinical success rates of 89%.<sup>29</sup> The aforementioned may prompt the question, ‘why then was flucloxacillin not used in our case? Was linezolid causative in his recurrent epidural abscess?’. In our case, *S. aureus* sensitivities

to clindamycin, linezolid and flucloxacillin were reported. According to Gibson *et al*,<sup>31</sup> flucloxacillin does not penetrate the avascular normal human vertebral discs, therefore it was not the antimicrobial of choice in our case. Our microbiologist, experienced in orthopaedic pathologies, deemed linezolid the antimicrobial of choice, demonstrating excellent tissue penetration and equivalent bioavailability between oral and intravenous therapy.<sup>25</sup> Linezolid, a member of the oxazolidinone class of antibiotics, is indicated for the treatment of skin and soft tissue infections caused by MSSA, MRSA or vancomycin-resistant enterococci and other susceptible microorganisms. Linezolid blocks the 50S ribosomal subunit and has bacteriocidal activity against Gram-positive organisms such as enterococci, staphylococci, streptococci and *Mycobacterium tuberculosis*. Linezolid has been suggested as an alternative to vancomycin in patients with SAB, but data are lacking; hence, clinicians may have concerns about the efficacy of linezolid when the blood culture is positive for *S. aureus*. Shorr’s pooled analysis of five prospective, randomised, controlled studies showed that linezolid appeared to be well tolerated and associated with clinical, microbiological and survival outcomes that were not inferior to those of vancomycin in patients with secondary SAB.<sup>32</sup> Two recent meta-analyses have demonstrated the superior efficacy of linezolid in the treatment of bone and joint infections as well as skin and soft tissue infections.<sup>34</sup> A meta-analysis by Fu *et al*<sup>36</sup> showed that linezolid is associated with better clinical and microbiological outcomes than glycopeptides for the treatment of *S. aureus* infections. Caution is advised due to side effects of anaemia, neutropenia, thrombocytopenia, leucopenia, pancytopenia and raised serum transaminase levels. It requires initiation under the supervision of a microbiologist. It is contraindicated with the concomitant use of serotonergic agents, tricyclic antidepressants and serotonin agonists because of the risk of serotonin syndrome. The main determinant of outcome is the neurological status at the time of diagnosis. Other predictors include age <60 years, <50% degree of thecal compression, <72 hours of cord symptoms and no comorbidities. In 1926, a mortality of 81% was reported, but with the advent of antimicrobial therapy, enhanced imaging techniques and prompt surgical decompression, the mortality rate now ranges from 2% to 20%.<sup>37</sup> Death may be due to sepsis or prolonged immobilisation, with subsequent pneumonia.<sup>38</sup> This case report reinforces the importance of considering a SEA in the differential diagnosis in patients presenting with sepsis, back pain, nerve root pain, motor weakness, sensory change and bladder or bowel dysfunction. Prompt recognition and a multidisciplinary approach with early initiation of antimicrobial therapy and surgical decompression is required to reduce morbidity and mortality. It also demonstrates the paucity of guidelines on antimicrobial therapy and its duration. The authors are not advocating linezolid as first-line antimicrobial therapy for *S. aureus* vertebral osteomyelitis, just reporting our experience. Perhaps our patients relapse was due to the use of linezolid, albeit a reduction in inflammatory markers was observed (CRP 0.8 mg/L). Prospective, randomised, double-blind clinical trials or large prospective observational studies are required to further delineate the efficacy of linezolid in the treatment of methicillin-sensitive *S. aureus* and to determine its ability to penetrate the avascular vertebral disc. This case also demonstrates the challenges posed in the treatment of *S. aureus* vertebral osteomyelitis and discitis with linezolid, the importance of initiation under the guidance of a microbiologist with close monitoring for signs of immune suppression.

## Learning points

- ▶ Spinal epidural abscess is a rare but severe infection requiring prompt recognition. It poses a diagnostic challenge to physicians as prior to the development of neurological signs, its presentation can mimic a broad spectrum of clinical pathologies.
- ▶ It has a male predominance, with a predilection for the fifth to seventh decade of life.
- ▶ Predisposing conditions include diabetes mellitus, trauma to the spine, intravenous drug use, immunosuppressive therapy, cancer, HIV/AIDs, alcoholism and chronic renal failure.
- ▶ Symptoms and signs at diagnosis include back pain, tenderness, motor weakness, radicular pain, sensory abnormalities, fever, bladder and bowel dysfunction, paralysis, neck stiffness, confusion, headache, nausea and vomiting.
- ▶ Inflammatory markers such as white cell count, C reactive protein and erythrocyte sedimentation rate (ESR) are generally elevated. Leucocytosis is found in 60–80%, and an ESR >20 mm/hour in up to 95% of reported cases.
- ▶ MRI with gadolinium has a specificity and sensitivity >90% to detect SEA and is therefore the diagnostic method of choice. In most studies, SEA is predominantly located in the thoracic and lumbosacral region.
- ▶ In addition to blood cultures, which remain negative in 40% of the cases, CT-guided needle aspiration of the abscess should be attempted because it has a higher sensitivity in identifying the causative microorganism.
- ▶ The management of SEA should always be multidisciplinary, involving spinal surgeons, radiologists and microbiologists. Drainage of the abscess and antimicrobial therapy are the basic principles of treatment. This case also emphasises the paucity of recommendations on antimicrobial treatment duration. *Staphylococcus aureus* infection can actually be very heterogenous and management needs to be tailored to the severity and characteristics of the individual case. The main determinant of outcome is the neurological status at the time of diagnosis.

**Contributors** All authors contributed to the writing of this manuscript. LD is responsible for writing the case report and literature review. SI is responsible for microbiology advise and management, literature review. CB is responsible for operation notes.

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**Patient consent** Obtained.

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