Case report

Resolution of group B streptococcal panspinal epidural abscess in a patient with diabetes after treatment with ceftriaxone and linezolid

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SUMMARY

Panspinal epidural abscesses are an extremely rare yet potentially fatal condition. Whether cases are best managed surgically or medically is currently controversial. A 63-year-old patient with diabetes presented initially with abdominal pain, back pain, urinary retention and constipation. He subsequently developed fevers, radicular pain and new-onset weakness in the right leg. MRI confirmed a panspinal epidural abscess extending from C7 to L5, with group B *Streptococcus* (GBS) cultured on sampling. Due to the significant risks of surgery he was managed conservatively, initially with ceftriaxone, and subsequently in combination with linezolid. Repeat MRI 3 months after presentation revealed complete resolution of the abscess. This case illustrates how conservative management is a valid option for patients with this condition, and supports the use of synergistic linezolid in this scenario. It also highlights how some cases may not initially present with the classically described triad of fever, back pain and loss of neurological function.

BACKGROUND

Spinal epidural abscesses (SEA) are an uncommon yet dangerous condition with significant risks of morbidity from irreversible loss of neurological function, and also mortality, which has been estimated to be between 6% and 32%.¹ Incidence is reported to be rising, likely due to increasingly elderly and comorbid populations, along with expanded use of spinal procedures and recreational intravenous drugs.² Other commonly observed risk factors include diabetes mellitus and concurrent infection, the latter most frequently from skin and soft tissue sources.³ The most common presenting symptoms include: fever, lower back pain and loss of neurological function. A typical progression pattern has been described whereby localised spinal pain and fever progress to radicular pain, from spinal root irritation, and subsequently to paralysis and death.³

SEAs may rarely extend over multiple levels. These are termed panspinal or holospinal epidural abscesses, and have been reported in only a handful of case reports and case series in the literature.^{4–11} Surgical decompression and drainage, usually via laminectomy, is generally recommended for SEAs extending to one or a few levels, particularly in patients who experience neurological sequelae.¹ However, extended laminectomy is not usually

recommended for treatment of panspinal epidural abscess spanning many levels due to the increased risks of intervention,² with mixed outcomes being reported for both surgical and conservative management in this subset of patients.⁴

Due to the rarity of panspinal epidural abscesses and associated significant risks of morbidity and mortality, there have been no prospective studies examining surgical versus conservative management, and evidence-based recommendations of how to best manage these cases are lacking.

CASE PRESENTATION

A 63-year-old male patient with diabetes presented to the on-call surgical team via the emergency department with a 9-day history of constipation, abdominal pain in the right lower quadrant and lower back pain. His comorbidities included ischaemic heart disease, hypertension, insulin dependent type 2 diabetes mellitus and peripheral vascular disease, for which he had undergone amputation of all toes of the right foot 5 months prior to presentation. He remained independent for activities of daily living and mobilised with a stick. His surgical history included a laparotomy 24 years prior for complications of alcoholic pancreatitis.

On examination he was afebrile and his observations were unremarkable. He had a reducible, non-tender incisional hernia in the midline at the site of the previous laparotomy. He was tender to palpation in the right iliac fossa and lumbar regions, and was found to be in urinary retention for which he was catheterised with a residual volume of 800 mL. Neurological examination of the lower limbs demonstrated increased tone bilaterally, normal power in all muscle groups, reduced ankle jerks bilaterally and reduced sensation globally in both feet. Cranial nerve, upper limb and rectal examination, including perineal sensation, were unremarkable.

Inflammatory markers were raised, with C-reactive protein (CRP) 318 mg/L (reference range <5), white cell count $11.8 \times 10^9/L$ (3.0–10.0), predominantly neutrophils and alkaline phosphatase 265 IU/L (30–130). HbA1C was 111 mmol/mol Hb (12.3% total Hb), indicating poor long-term diabetic control. Other admission blood tests were unremarkable. A CT scan of the abdomen and pelvis was completed which confirmed the abdominal wall hernia, but showed no evidence of

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Figure 1 MRI scans of the lumbar spine (A) Postcontrast sagittal T1-weighted scan demonstrating enhancement around the abscess (arrows), spinal cord and cauda equina. (B) Axial section showing extension of the abscess into right psoas major and erector spinae muscles. (C) T2-weighted sagittal scan showing spinal epidural abscess extending cranially. (D) T2-axial scan showing extension of the abscess into surrounding muscles. (E, F) Resolution of epidural abscess on T2 MRI seen after 12 weeks of antibiotics.

bowel obstruction or strangulation, nor any other acute intra-abdominal pathology.

Four days into the admission, the patient became febrile and developed debilitating radicular pain in the right leg. His bowel function had returned to normal but palpation of the lumbar spine revealed tenderness over multiple spinous processes, and pus was found around the wound site of the right foot. Empirical intravenous co-amoxiclav was given. Up to this point, the patient was not treated with any antibiotics, as despite a raised CRP, he did not have any infective symptoms, and no source of infection could be identified.

The admission scan was also re-reviewed and a soft tissue density posterior to the L5 vertebral body was identified. Due to concerns regarding spinal canal pathology, MRI of the lumbar spine was completed on day 6 of admission, which required arranging at a specialist imaging centre as the patient had an implantable cardioverter-defibrillator that had to be switched off for the duration of his scan. The MRI revealed an epidural abscess extending from L5 cranially towards the thoracic spine, with paraspinal invasion into the surrounding psoas and erector spinae muscles (figure 1A–D). After 2 days of intravenous co-amoxiclav, he was switched to intravenous ceftriaxone once the epidural abscess was diagnosed on MRI.

Following the initial MRI scan and on day 6 of the admission, he developed a frontal headache, neck pain and stiffness, followed by new-onset weakness of the right lower limb on day 12. Power in the right lower limb diminished to 4+/5 for hip extension, 2/5 for hip flexion, 3+/5 for knee extension and 4–/5 for knee flexion. Power in the left lower limb, and rest of the body, remained normal. Further imaging of the cranial and thoracic spine demonstrated the abscess extending cranially from L5 (as shown in the previous MRI) up to C7, displacing the spinal cord anteriorly (figure 2A, B). CT head did not reveal any meningeal enhancement or intracranial collections.

The patient was deemed unsuitable for neurosurgical intervention due to the extent of the abscess and significant risk of morbidity and mortality from intervention. Samples for culture were obtained via CT-guided drainage. GBS was grown which was resistant to clarithromycin, clindamycin and doxycycline,

Figure 2 MRI scans of the cervical and thoracic spine. (A, B) T2-weighted scans demonstrating T2-hyperintense spinal epidural abscess (arrows) displacing the thoracic spinal cord anteriorly in mid-sagittal and axial views. (C, D) Resolution of spinal epidural abscess is seen after 12 weeks of antibiotic therapy in mid-sagittal (T2) and axial views (T1).

but sensitive to ceftriaxone, penicillin and linezolid. The source was thought to be from the wound site of his diabetic foot where previous swabs had cultured *Staphylococcus aureus* and GBS.

Despite 5 days of treatment with ceftriaxone, the patient continued to deteriorate and oral linezolid was added as a second agent. Over the subsequent 3 weeks the patient improved clinically on this regime with his inflammatory markers normalising (figure 3) and oral linezolid was stopped after a course of 16 days. He received ongoing neurophysiotherapy, input from the diabetes team and podiatry review. On day 24 of admission, power in the lower limbs was 5/5 bilaterally for hip flexion, hip extension, knee flexion and knee extension.

OUTCOME AND FOLLOW-UP

After a 5-week admission, he was discharged on outpatient parenteral antimicrobial therapy, with intravenous ceftriaxone administered via a peripherally inserted central catheter (PICC) line, completing a total course of 12 weeks of ceftriaxone 2g once daily. Follow-up MRI at completion demonstrated complete resolution of the abscess (figures 1E, F and 2C, D). He mobilised with a stick without pain, and was able to void urine

Figure 3 Progression of the patient's C-reactive protein (CRP) and white cell count (WCC) from initial presentation.

independently. He has since been discharged from our Infectious Diseases clinic, and receives ongoing diabetes input.

DISCUSSION

This is the first report of a panspinal epidural abscess (C7 to L5) caused by GBS that has resolved after treatment with ceftriaxone in combination with linezolid. Given the vertical extension of the abscess, extended laminectomy was not feasible and the patient was deemed to be inoperable. Moreover, the neurosurgical team had significant concerns that the patient would not survive this illness, regardless of whether surgery was pursued or not. Nevertheless, he made a full neurological recovery with no lasting sequelae and remained pain free, demonstrating that medical treatment with antibiotics can lead to resolution of panspinal epidural abscesses in patients who have developed focal neurological signs, and may result in outcomes comparable to those treated surgically.^{4 12}

In this case, linezolid was added as a second antibiotic after the patient was deemed inoperable and continued to deteriorate despite 5 days of ceftriaxone. It was chosen partly due to the resistance pattern of the causative organism, but also for its good penetration into bone and cerebral spinal fluid,¹³ and its alternative mechanism of action to ceftriaxone, whereby it was hoped it would reduce the rate of bacterial growth and toxin production, thereby reducing the local inflammation.¹⁴ Once the patient's inflammatory markers and clinical condition normalised, linezolid was discontinued.

Linezolid is indicated in the treatment of pneumonia and complicated skin and soft tissue infections caused by Gram-positive organisms. Potential side effects include optic neuropathy and myelosuppression resulting in thrombocytopenia.¹⁵ It remains off licence for treating infections of the central nervous system (CNS).¹⁵ Existing case series have shown promising outcomes for SEAs and CNS infections treated with linezolid,^{7 16} including those caused by methicillin-resistant *S. aureus*,¹⁷ the incidence of which (SEAs) has been reported to be increasing.² Resolution of meningitis in a 67-year-old patient caused by GBS has been reported after treatment with linezolid in conjunction with meropenem, where improvement occurred after linezolid was given.¹⁸ Nevertheless, further systematic clinical trials are required to strengthen the evidence base for the use of linezolid in the treatment of SEAs.

Learning points

- Medical treatment of panspinal epidural abscesses in patients who have developed focal neurological signs can result in complete resolution with no lasting neurological deficits.
- Antibiotic choice directed by culture and sensitivities is essential; in this case ceftriaxone in combination with linezolid successfully treated a panspinal epidural abscess caused by group B *Streptococcus*. There may be future uses of linezolid in central nervous system infections.
- Panspinal epidural abscesses may present atypically, and must be considered as a differential diagnosis in patients with raised inflammatory markers where acute intra-abdominal pathology has been ruled out.

This case also highlights that epidural abscesses may not initially present typically with all three classic symptoms of back pain, fever and focal neurological deficits, or follow the typically described progression pattern from localised spinal pain and fever to radicular pain and subsequently to paralysis.³ Indeed, our patient initially presented to the on-call surgical team with an acute abdomen, which was subsequently highlighted as constipation and urological retention. Other cases of SEA have also been reported to present atypically, initially with abdominal pain, urinary retention and constipation,⁸ and it is pertinent to have a high index of suspicion for SEA in these cases where acute intra-abdominal pathology has been ruled out, especially in patients with raised inflammatory markers and risk factors such as diabetes.

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