Reminder of important clinical lesson

Spontaneous abscess of the lumbar spine presenting as subacute back pain

Jon M Dickson,¹ Daniel J Warren,² Ann L N Chapman,³ Unni Anoop,⁴ Haleema Hayat,⁵ Debapriya Bhattacharya⁶

¹ SHO (GP VTS1), Health Care of the Elderly, Northern General Hospital, Sheffield, UK

² Department of Radiology, Royal Hallamshire Hospital, Sheffield, UK

³ Department of Infection and Tropical Medicine, Royal Hallamshire Hospital, Sheffield, UK

⁴ Department of Radiology, Chesterfield Royal Hospital, Chesterfield, UK

⁵ Department of General Internal Medicine, Chesterfield Royal Hospital, Chesterfield, UK

⁶ Department of Neurosurgery, Royal Hallamshire Hospital, Sheffield NHS Teaching Hospitals Trust, Sheffield, UK

Correspondence to Jon M Dickson, jon.dickson@thejce.com

Summary

A 52-year-old lady was admitted with back pain, fever and reduced sensation in her left leg. Soon after admission she developed acute urinary retention. She underwent urgent MRI of the spine which showed an epidural abscess compressing the thecal sac which was treated with neurosurgical decompression and intravenous/oral antibiotics. Cultures from theatre grew *Staphylococcus aureus* sensitive to flucloxacillin. After 12 weeks of intravenous/oral antibiotics, she was discharged without any neurological disability.

BACKGROUND

This case acts as a reminder of an important clinical lesson. Epidural abscess is a rare but potentially life-threatening condition which can cause permanent neurological disability if it is not diagnosed and treated quickly. The triad of back pain, fever and neurological signs is the classical presentation of epidural abscess, but these features are neither sensitive nor specific. Initial tests such as full blood count, erythrocyte sedimentation rate (ESR) and C reactive protein (CRP) may help with the diagnosis, but the key investigation is MRI. More research is needed to provide data which will allow an evidence-based approach to the clinical diagnosis of spinal epidural abscess (SEA).

CASE PRESENTATION

A 52-year-old lady was admitted to the local emergency management unit by her general practitioner with a 5-week history of lumbar back pain. She had no significant past medical history and, of note, was not diabetic.

Her symptoms began acutely whilst picking up a heavy box. She experienced sudden onset of severe (10/10) lumbar-back pain with radiation into the left leg. Her general practitioner (GP) diagnosed acute posterolateral disc herniation, and she was treated with standard doses of oral analgesia (paracetamol, tramadol, gabapentin) and oral diazepam which gave partial relief from her symptoms. She was not treated with intravenous or spinal injections of any sort. Over the next 4 weeks, her pain was constant but exacerbated by movement. She was unable to return to work and stayed at home where she was self-caring but with severely restricted mobility. Throughout this period neurological examination was normal, except for reduced straight leg raise.

In the week preceding her admission she developed concurrent systemic symptoms; she complained of nausea,

vomiting and intermittent fevers. In addition, the severity of her back pain at rest became unbearable and her sciatica progressed to affect both legs.

On admission, she was afebrile (but her GP had recorded a temperature of 37.7°C earlier that day), blood pressure was 184/82, heart rate was 95/min (regular), oxygen saturations were 99% (room air) and respiratory rate was 16/min. She complained of 10/10 pain. The lumbar spine was tender, but it was not warm to touch and there were no skin colour changes. Her neurological examination was restricted by pain, but there was no abnormality detected in tone, power or reflexes in the lower limbs. She was unable to tolerate straight leg raise beyond 15° bilaterally, and there was reduced sensation in the left L4/L5 distribution. She had normal anal tone and normal perianal sensation. A few hours after hospital admission she developed acute urinary retention and was catheterised.

INVESTIGATIONS

Routine blood tests showed raised white-cell count and raised inflammatory markers (see table 1). Her chest radiograph and urine dipstick showed no signs of infection, and there was no obvious focus for her sepsis¹ (tachycardia, CRP=268, white-cell count = 22.9) other than her lumbar spine so she underwent an urgent MR scan. This revealed an abscess within the right erector spinae muscle at L5/S1 tracking superiorly to the L4/L5 level and into the posterior epidural space where there was an epidural abscess partially compressing the thecal sac. The cerebrospinal fluid within the distal canal had an abnormally increased T1 signal, and there was associated abnormal contrast enhancement surrounding the cauda equina and overlying the spinal cord – features suggestive of arachnoiditis (figure 1). There was no evidence of discitis or vertebral body destruction, the

Figure 1 (A) Midline sagittal T1-weighted MR scan of the lumbar spine demonstrates abnormal increased T1 signal of the CSF within the spinal canal (white arrow). Normal intervertebral disk spaces and vertebral body signal. Note the urinary bladder distension (white asterisk). (B) Midline sagittal T1-weighted MR scan of the lumbar spine with gadolinium enhancement. Extensive cauda equina and distal cord enhancement (black arrow) with associated posterior epidural collection (black arrowhead) compressing the thecal sac between L3/L4 and L4/L5. (C) Postcontrast axial T1-weighted MR scan of the lumbar spine at L4/L5. Inflammatory change within the right erector spinae muscle (black asterisk) and a moderate right-sided epidural collection (white arrowhead) results in thecal effacement and compression of the cauda equina nerve roots.

vertebral marrow signal was entirely normal and there was no end plate irregularity.

Table 1 Blood test results

		Day 1	Day 15	Day 82
Full blood count				
White-cell count ($\times 10^9$)	(3.9–9.5)	22.9	7.2	5.4
Inflammatory markers				
C reactive protein (mg/l)	(0–7.5)	268	18.8	4.4
Erythrocyte sedimentation rate (mm/h)	(1–15)	117	102	29

DIFFERENTIAL DIAGNOSIS

There is a broad differential diagnosis for back pain: musculoskeletal (mechanical, degenerative facet joint disease, prolapsed intervertebral disk, vertebral fracture or collapse), malignancy (vertebral metastasis, myeloma) and infective (spondylodiscitis, pyogenic discitis, septic arthritis, bacterial meningitis). The onset of symptoms in this case was typical for acute posterolateral intervertebral disk prolapse, and she was treated on this basis for the first 5 weeks of her illness. She was relatively young and previously well, so malignancy was thought to be unlikely. She was referred to hospital when she started to develop systemic features suggestive of an infective cause for her pain. Raised white-cell

count and inflammatory markers in the absence of another source of infection made this the most likely diagnosis, and, at this stage, an urgent MR scan was essential.

TREATMENT

Empirical antibiotics (intravenous teicoplanin 400 mg once daily and oral rifampicin 600 mg twice daily) were commenced, and she was urgently transferred to the local neurosurgical unit where she underwent emergency spinal neurosurgery with evacuation of the epidural and paraspinous abscesses, including partial laminectomies of L3 and L4. The initial blood culture was negative but culture of the pus taken intra-operatively grew *Staphylococcus aureus*, sensitive to flucloxacillin, fusidic acid, penicillin, erythromycin and rifampicin. Antibiotic treatment was accordingly switched to intravenous flucloxacillin and oral rifampicin. Cultures for acid-fast bacilli were negative. Tests for HIV, methicillin-resistant *Staphylococcus aureus* and fungal infection were all negative. Echocardiography was undertaken to rule out infective endocarditis and was normal.

OUTCOME AND FOLLOW-UP

The patient was discharged from hospital 2 weeks after admission with a lumbar corset and a plan for 12 weeks of antibiotics. The first 8 weeks were given intravenous (given after discharge by the Outpatient and Home Parenteral Antibiotic Therapy Service) with an additional 4 weeks of oral therapy. Follow-up imaging at week 3 confirmed that

Figure 2 (A) Sagittal T1-weighted MR scan of the lumbar spine after decompression and 3 weeks of antibiotic therapy. Postoperative laminectomy L3 and L4. Residual inflammatory changes noted within the posterior soft tissue between L3 and L5. The CSF signal is less bright than on the presentation scan (white arrow). (B) Sagittal T1-weighted MR scan of the lumbar spine with gadolinium enhancement. Abnormal enhancement within the distal spinal canal remains, though considerably improved (black arrow). A residual collection is present at the surgical site, posterior to the spinal canal at L4 (black arrowhead). (C) Postcontrast, axial, T1-weighted MR scan of the lumbar spine confirms the enhancing collection at L4 posterior to the spinal canal (white arrowhead) but with partial relief of the thecal compression following surgical decompression. There remains a thin epidural collection and abnormal enhancement within the erector spinae muscle (black asterisk).

there had been partial decompression of the epidural collection with resultant relief of the cauda equina compression. There remained some residual abnormal enhancement within the lumbar and lower thoracic spinal canal (figure 2), but the imaging appearances were much improved compared with the initial scans. She was followed up in the neurosurgical outpatient clinic: At week 16 she was mobilising normally without a corset, she had no neurological deficit or disability and her bladder and bowel function was normal. Serial inflammatory markers were monitored during her recovery and were essentially normal by week 11 (see table 1).

DISCUSSION

Epidemiology

SEA is rare (less than two cases per 10 000 hospital admissions²) and usually occurs in patients with risk factors³ such as recent epidural cannulation,⁴ intravenous drug use,⁵ diabetes mellitus or infection at another site such as endocarditis.⁶ The mortality is around 15%.⁷

Pathology and microbiology

The abscess is usually only one or two vertebral bodies in length and most commonly affects the lumbosacral spine^{6,8}, but other regions can be affected and, in extreme cases, the abscess can extend throughout the whole spinal canal.^{9–11} It is usually secondary to discitis, osteomyelitis or pyomyositis of the erector spinae muscles.¹² *S aureus* (including MRSA) accounts for more than 50% of cases¹³, but a wide range of other organisms can be responsible, including Streptococci,^{14–17} *Mycobacterium tuberculosis*¹⁸ and Brucella species. SEA caused by Gram negative organisms usually

occurs in patients with diabetes mellitus, old age or concurrent (or multiple previous) urinary tract infections.

Symptoms

Symptoms develop in four stages⁷: (1) back pain and fever, (2) symptoms of radicular irritation; (3) muscle weakness, sensory deficits, bladder and bowel dysfunction; and (4) paralysis. Back pain at presentation is almost universal, but many patients are initially afebrile.^{8–19} Once patients develop weakness they are at high risk of permanent neurological disability.^{3,20}

Blood tests

White-cell count and inflammatory markers are often raised, but they are not specific to SEA. ESR is the most sensitive test, and it has been suggested that low ESR (in the absence of risk factors) could be used to exclude the diagnosis without MRI.^{3,6,21} Persistently raised posttreatment inflammatory markers may predict treatment failure.¹³

Imaging etc

MR scanning is the essential investigation for the diagnosis and management of SEA, but the utility of follow-up MRI after treatment for lumbar abscess is not certain.^{13,22} Although many imaging parameters improve after treatment, residual paraspinal inflammation and epidural enhancement often persist. So overall improvement in the imaging signs associated with improving inflammatory markers are the key predictors of a good outcome.¹³

Treatment

Laminectomy and intravenous antibiotics are essential for patients with neurological signs²³, but this type of surgery

may cause spinal instability and deformity. Recent studies have shown that MRI can be used to decide which patients may be suitable for less invasive techniques²⁴ or medical management.^{2 8 25–29}

There are no UK guidelines for antibiotic treatment of SEA. Standard practice in the UK is to treat empirically for Gram-positive organisms, including MRSA, and consider also covering Gram-negative bacteria if the patient has risk factors for this type of infection (see the section on Pathology and Microbiology). In this case, intravenous teicoplanin and rifampicin were chosen as empirical therapy because teicoplanin provides good cover against *S aureus*, including MRSA, and rifampicin is also active against Gram-positive organisms and has excellent tissue penetration. Empirical Gram-negative cover was thought to be unnecessary in this case – this was confirmed by the culture results. When culture results were available, the antibiotics were switched to intravenous flucloxacillin and oral rifampicin. There are no controlled trials comparing flucloxacillin with the combination of flucloxacillin and rifampicin. However, the addition of rifampicin as a second agent is frequent practice in the UK.

Learning points

- ▶ The differential diagnosis for back pain, with or without neurological symptoms and signs, includes SEA.
- ▶ This diagnosis should be taken especially seriously in any patient with back pain plus fever without another obvious source of infection.
- ▶ Clinical assessment should include history (especially risk factors for SEA, neurological symptoms and bladder/bowel function), clinical examination and blood tests (full blood count, CRP, ESR).
- ▶ Urgent MRI and surgical intervention is essential to minimise the risk of permanent neurological disability.

Competing interests None.

Patient consent Obtained.

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