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A case of spinal epidural abscess concealed by delirium in a young man

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ABSTRACT

Spinal epidural abscess is a rare but serious condition with poor outcomes. It's classic triad of new back pain, neurological deficit and fever is only present in 15% of cases at presentation and is initially misdiagnosed in 75–89%.[6,7] Delaying treatment is associated with worse outcomes. Delirium is itself a risk factor for mortality but the disturbance in cognition and memory can also complicate clinical assessment.[1–5]

We present a case of delirium caused by, and obscuring, a spinal epidural abscess. This case highlights the difficulties in diagnosing spinal epidural abscesses, the need for a high index of suspicion for the condition and timely action to minimise morbidity. In addition, it demonstrates the value of treating unexplained delirium as an emergency and the danger of diagnostic premature closure. Finally, the importance of persistent clinical examination of the confused and non-cooperative patient.

Introduction

Delirium is a heterogenous syndrome of acute disturbance in cognition, orientation and alertness, representing a state of cerebral insufficiency.[1] It is an independent risk factor for mortality solely from its presence,[2–4] this risk increasing with prolonged duration.[5]

Commonly seen in older age groups, it can also arise in younger people. We present a case of delirium caused by, and obscuring, a severe deep-seated infection.

Case

A male orthopaedic inpatient in his mid-twenties presented with aggressive and disruptive behaviour, removing IV access devices, and repeated falling. Previously fit and well apart from heavy alcohol use, he was admitted 2 weeks before, following a road traffic accident while intoxicated. Admission CT trauma revealed a sub-dural haematoma, managed non-operatively. Pelvic fractures with a bleeding vessel which had required interventional radiological embolisation. In addition, he had multiple fractures (right T2–4 transverse processes, right frontal bone, left 6–8th rib), and several soft tissue wounds. In preceding days his behaviour had prevented a planned CT angiogram to examine a femoral pseudoaneurysm and interrupted antibiotic treatment. This had been attributed to alcohol withdrawal; however, his CIWA score was low, and his confusion was not improving.

Clinical assessment was confounded by his inability to cooperate. He reported widespread severe pain in his pelvis, gluteal region and both feet, worse on the left. He was unable to detail the events of the admission, 4AT score was 5.

Examination

A thin young man with numerous dry, non-erythematous wounds across the head, trunk and limbs including his right ear. Widespread excoriation. No embolic phenomena, fever or tremor.

Respiratory: Normal.

Cardiac: Warm peripheries. No murmurs. Severe bilateral pitting oedema up to the lower abdomen. Heart rate 110 beats per minute. Blood pressure 120/75 mmHg.

Neurology: Alert. Cranial nerves normal. Moving all four limbs.

Abdomen: Soft, tender lower abdomen, non-distended.

Musculoskeletal:. No erythematous joints. Used his arms to lift his legs into the bed.

He was undergoing treatment with Co-amoxiclav for infection of unclear source and topical chloramphenicol for a right ear infection. In addition, he was requiring significant doses of opioid painkillers. Available test results are displayed in Table 1.

At this stage the working diagnosis was delirium of infective aetiology. The source was unclear, but he was treated for a worst-case scenario of intracranial staphylococcal infection with meropenem and teicoplanin. Differentials being infective endocarditis or an intra-abdominal infection. The cause of his severe oedema was unclear, potentially due to heart failure from infective endocarditis, or nephrotic syndrome. A CT scan (results in Table 2) was performed with 1:1 nursing, and a small dose of oral benzodiazepine.

He was reassessed. Lower limb neurological examination, limited by poor cooperation, pain and severe leg oedema revealed:

Bilateral reduced tone and power in hip and knee flexion.

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Table 1
Available results initially.

Blood cultures:	*Staphylococcus epidermidis* 3 days previously Unidentified Gram-positive cocci 2 days previously
Right ear swab 5 days previously	*Staphylococcus aureus*
Ultrasound doppler for DVT (Bilateral) 3 days previously	No DVTs. Incidental right CFA pseudoaneurysm at the site of previous IR embolisation.
Abnormal blood tests 1 day previously:	Albumin 26 g/L, CRP 288 mg/L, WCC 33.1×10^9/L, Neutrophils 28.1×10^9/L.

CFA: common femoral artery, CRP: C-reactive protein, DVT: deep vein thrombosis, IR: interventional radiology, WCC: white cell count

Table 2
Results after first round of investigations.

CT Head, abdomen and pelvic angiogram.	- No evidence of intracranial infection. - Improving SDH. - Severely comminuted, displaced sacral fractures. - No clear focus of infection. - Small thrombosed 5 mm right CFA pseudoaneurysm.
Urine dip	Normal
Blood cultures	2 blood cultures growing *Staphylococcus epidermidis*

CFA: common femoral artery. SDH: sub-dural haematoma.

Table 3
Results after second round of investigations.

Echocardiogram:	Normal.
MRI Whole Spine:	Complex lumbosacral fracture with multi-compartmental collections within the lower lumbar and sacral spine effacing the cauda equina nerve roots.

Globally reduced power in ankles and toes.

Asymmetric knee, absent ankle reflexes. Left plantar down-going, right: no movement.

Possible reduced distal sensation. Refused perianal sensation and anal tone examination.

His confusion subsequently deteriorated rapidly, IV access was lost. Administration of treatment was impossible. He was catheterised for urinary retention.

At this point, with the underlying diagnosis still unclear, he was transferred to intensive care, where he was sedated and ventilated to allow investigations and treatment. Magnetic resonance imaging (MRI) of his whole spine was obtained, seeking signs of neurological damage from the sacral fractures and rule out a spinal epidural abscess (SEA). Infective endocarditis remained a differential. These results are shown in Table 3 and Fig. 1.

He underwent emergency surgical decompression that night, subsequently spending 10 days in intensive care. His delirium and oedema improved with treatment of the infection. He remained 6 weeks in hospital for IV antibiotics, before discharge to a spinal rehabilitation unit. At the time of discharge he was progressing well, walking with supervision.

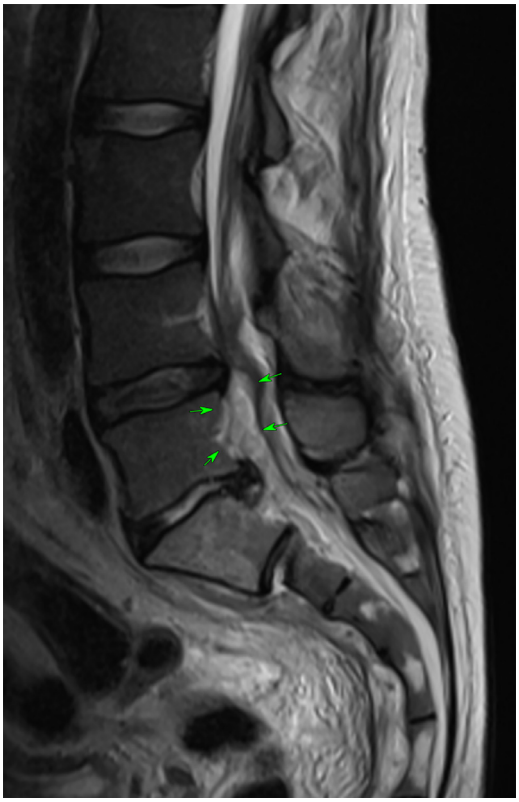

Fig. 1. This sagittal T2 image shows the epidural collection ventral to the cauda equina at L5 level.

Discussion

This case demonstrates the value of approaching delirium as an emergency. This premise drove the aggressive treatment, investigation, and use of intensive care when reasonable non-invasive methods had failed.

Identifying spinal epidural abscesses (SEA) is known to be challenging, data suggests that up to 75–89% are initially misdiagnosed. The classic triad of back pain, fever and neurological deficit is only present in 10–15% of patients at first contact.[6] A deliberate alertness to their development can prove vital to making the diagnosis. These features are highly specific, some evidence suggesting as few as 0.8% of patients with all three do not have SEA.[7] A valuable observation from this case is that the triad can be hiding in plain sight, obscured by distractors such as pre-existing back pain, or a confused patient. This case demonstrates the importance of persisting with clinical examination even under suboptimal conditions. The neurological findings could not be explained by existing diagnoses, therefore indicating an additional pathology.

SEA has been described as having four stages:

1. Back pain, spinal tenderness and fever.
2. Radicular pain, neck stiffness, reduced tendon reflexes.
3. Muscle weakness. Sensory abnormalities, bowel and bladder dysfunction.
4. Complete paralysis.

Progression from one stage to the next is variable, but potentially rapid.[6,7]

The evidence shows urgent surgical decompression improves outcomes in SEA. Prompt diagnostic MRI should be prioritised once it is suspected.[6,7] In this case, profound delirium necessitated admission to intensive care to achieve this. The danger of diagnostic premature closure is evident; a continued assumption that the pain and neurology were completely explained by the fractures could have cost this man the ability to walk.

An awareness of risk factors is valuable at alerting clinicians to the possibility of SEA. The strongest association is bacterial infection or bacteraemia of any source. Secondly immunosuppression by any cause, rel-

evant in this case is alcohol use. Invasive procedures, especially those in proximity to the spine or involving vascular access. Finally, trauma or significant medical comorbidities.[6,7]

Conclusion

The aim of this article is to raise awareness of spinal epidural abscess, its difficult diagnosis and the need for urgent investigation and treatment if suspected. In addition, demonstrating the value of considering inadequately explained delirium as an emergency that not only indicates poor prognosis but veils its own aetiology.

Author contributions

The manuscript was planned, researched and written by Dr Benjamin Page. Dr Sam Waddy planned, oversaw and edited the manuscript.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Consent

Written consent to publish was obtained from the patient.

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