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Spinal Epidural Abscess Without Fever: Challenging the Classic Triad

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Abstract

Introduction
We report three cases of spinal epidural abscess (SEA).

Case 1
A 71 year old recurrently presented with paraspinal pain, with no fever or neurological defects on initial reviews. She later developed bilateral leg weakness and became unresponsive.
Case 2
A 62 year old presented with a six day history of back pain and sciatica which then spread to her thoracic spine. She was apyrexial but transiently needed inotropic support.
Case 3
A 56 year old with presented with an eight day history of radicular back pain. She denied fever. She was treated for septic shock.

Outcome
Mean CRP was 446. All had SEA on MRI. All grew S*taphylococcus Aureus*. Case 1 passed away shortly after admission. Cases 2 and 3 remain in hospital on targeted antibiotic therapy.

Discussion
Current advice is that SEA should be suspected in febrile patients with spinal pain with radiculopathy or other focal neurology. Of note in our case series is that fever was not a feature, though all had grossly elevated CRP. This raises questions as to whether there is a role to check inflammatory markers in patients with refractory back pain even in the absence of fever in order to improve early detection of SEA.

Introduction

Spinal epidural abscesses are a rare but potentially catastrophic cause of back pain. Unfortunately, the clinical manifestations are easy to miss and dismiss. The classic diagnostic triad of focal spine pain, neurological deficit and fever is not always present in practice. Three

female patients, in a short time frame, presented to a regional centre with back pain, neurological sequelae and high inflammatory markers, however, none had fever.
Herein we discuss the need for clinical pathways to include assessment of inflammatory markers when patients present with refractory back pain, associated with neurological features, with the hope of improving early detection of this pathology.

Case 1

A 71 year old female with a history of hypothyroidism and hyperlipidaemia developed acute posterior neck and paraspinal pain after lifting a bucket at home. She presented to her general practitioner (GP) twice in the next two days. She then presented to the emergency department (ED) three times in the following six days. At the time of these reviews she was clinically well, despite ongoing pain. She was alert, orientated, mobilising independently, had no focal neurology, and was apyrexial. There was no documented fever, headache, or rash. X-ray C-spine showed no obvious fracture or dislocation but had severe spondolytic changes. She had multiple drugs prescribed for the pain, including gabapentin, tramadol, paracetamol, and anti-inflammatories. Eight days after her original presentation to her GP she called an ambulance reporting feeling unwell with nausea, vomiting, malaise, and poor oral intake. Significantly she also reported new onset bilateral leg paraesthesia and poor mobility. Her neck pain had improved but she now had pain intrascapularly and was brought to ED. The attending doctor noted MRC muscle power scale of zero in both her legs. She then became acutely unresponsive during assessment. An urgent computed tomography (CT) scan of her brain and C-spine showed no acute injuries but an increased prominence of the ventricular system compared to a prior CT scan of the brain that had been done two years previously to assess for the cause of headache. Inflammatory markers were grossly elevated with white cell count (WCC) of 16.99 x 10^9 and C-reactive protein of 399 mg/L. She was moved to the resuscitation area, intubated, ventilated. She was commenced on ceftriaxone, vancomycin, and aciclovir. The working diagnosis was central nervous system infection secondary to a spinal collection. Magnetic resonance imaging (MRI) of the brain and spine showed cerebral oedema along with features of meningitis and ventriculitis, a diffusely abnormal cord with intramedullary high signal in the cervical cord, and an epidural collection at C4-C5 with enhancing paraspinal collections in the erector spinae muscles. Blood cultures and urine sample grew *Staphylococcus aureus*. She unfortunately passed away shortly after the MRI was filmed.

Case 2

Approximately four weeks after Case 1, a 62 year old lady was brought in by ambulance. Her background history was significant for psoriasis, ankylosing spondylitis, and osteoporosis. She was on ixetuzymab (a biologic medication used in the treatment of ankylosing spondylitis) and alendronic acid regularly. She reported an eight day history of lower back pain radiating

down her right buttock, which had come on whilst walking. Her mobility was significantly impacted. She was seen by out of hours GP services three times, attended a chiropractor, and was seen in a private hospital. She had an XR at that time and was booked for an outpatient MRI. She began to feel unwell with malaise, nausea, and vomiting. She denied any headache, fever, or rash. The pain began to ascend her spine into the intrascapular and posterior neck region. On presentation, she was alert and orientated, but globally weak, with power of four out of five on in both arms and two out of five in her legs. She denied any urinary or bowel symptoms. She became hypotensive and was commenced on inotropic support. Bloods revealed grossly elevated inflammatory markers with WCC 24 x 10^9 and CRP of 371 mg/L. CT of her lungs, aorta and spine showed extensive infiltration in the lower lobes of both lungs but no evidence of aortic pathology, pulmonary embolism, or spinal collection. She was commenced on ceftriaxone and vancomycin. Subsequent MRI showed extensive epidural abscess extending from C1 level to S3 with associated mass effect on the thecal sac. There were also features suggestive of septic arthritis in the L4-5 facet joint, along with paraspinous soft tissue enhancement and oedema in bilateral psoas muscles. Blood cultures grew Staphylococcus aureus sensitive to flucloxacillin. Neurosurgical colleagues advised targeted antibiotic cover and interval imaging. Antibiotic cover was rationalised to flucloxacillin monotherapy. Repeat MRI after two weeks showed overall improvement. Inflammatory markers have returned to normal, with a WCC of 5.6 x 10^9 and CRP of 0.4 mg/L. She continues to rehabilitate in hospital.

Case 3

On the same afternoon as Case 2, a 56 year old lady was admitted with septic shock. She had a background history of a prior L4-L5 discectomy several years prior. She was otherwise healthy and on no regular medications. She reported a six day history of radicular back pain radiating down the right leg. The pain was refractory to codeine, anti-inflammatories, and diazepam. She also reported a two day history of feeling unwell with cough, loose bowel motions and malaise. On presentation she was pale and lethargic with cool peripheries. Her blood pressure was 85/68 mmHg, heart rate of 120 beats per minute, and saturations of 93% on room air. Initial venous blood gas showed a pH of 7.27, lactate of 8.1 mmol/L and glucose of 3.1. Her CRP was 576 mg/L. She also had an acute kidney injury and deranged liver function tests. She was commenced on fluids, inotropes and broad-spectrum antibiotics (vancomycin and ceftriaxone) for septic shock with possible spinal source. Chest X-ray showed bilateral consolidation. CT imaging of brain, abdomen and pelvis showed extensive multifocal infiltrates in both lungs but were otherwise unremarkable. She was booked for MRI spine but was unfortunately too unstable to have this. She deteriorated from a respiratory perspective and was intubated. Blood cultures grew Staphylococcus aureus. Sputum samples taken during bronchoscopy also grew Staphylococcus aureus. Transoesophageal echocardiography was normal. Cerebral spinal fluid obtained on lumbar puncture showed a very high protein count

but no growth. She was transferred to another centre to facilitate MRI whilst on a ventilator. MRI spine showed L4-L5 discitis-osteomyelitis with an associated epidural collection extending from L4-S1 levels with compression of the thecal sac. There was also a small presacral collection and inflammatory changes within the paraspinal muscles. She remains in hospital.

Discussion

Spinal epidural abscesses (SEA) are uncommon, with figures of around 5 cases per 10,000 admissions reported in American literature[1]. Two of our three cases had risk factors for SEA, one- prior discectomy (albeit a distant history) and the other- ankylosing spondylitis on biologic therapy. *Staphylococcus aureus* is the causative pathogen in about two thirds of cases[2,3], and was diagnosed in all three of our patients.

The diagnosis of SEA is notoriously easy to miss, with patients often presenting multiple times prior to SEA even being suspected[4]. Current advice is that the diagnosis of SEA should be suspected in febrile patients with spinal pain accompanied by radiculopathy or other focal neurological findings[4]. Literature reports back pain, fever, and neurological deterioration as a classic triad of symptoms in SEA[5]. However, in practice only a small proportion have all three components at presentation[4]. Of note in our case series is that fever was not a feature in any of our cases, though they all had grossly elevated CRP. Our series highlight that failing to meet all three criteria in the classic triad does not out rule SEA. In patients with recurrent presentations for back pain with neurological deficits, CRP testing is highly sensitive and moderately specific in identifying patients with SEA[6]. This raises the question as to whether there is a role for clinical pathways to check inflammatory markers in patients presenting with refractory back pain with radicular or neurological features, even in the absence of fever, to improve early detection of this pathology. Lumbar punctures are typically not performed in SEA as the diagnostic yield is low and there is a potential risk of introducing infection into the central nervous system if the needle goes through an affected area. In most cases, the CSF findings are limited to a nonspecific elevation of protein and white cells[3]. CSF Gram stain is usually negative[3]. Furthermore, CT imaging failed to show definitive evidence of SEA in two of our cases, highlighting the importance of obtaining MRI imaging where there is suspicion of SEA.

Declarations of Conflicts of Interest:
None declared.

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