

An 85-year-old woman with acute back pain

Introduction

This article describes a case of an 85-year-old woman who was admitted via the emergency department with reduced mobility and general malaise. She was assessed by the physiotherapists, who recommended a period of inpatient rehabilitation. She was commenced on trimethoprim for a possible urinary tract infection and low molecular weight heparin for venous thromboembolism prophylaxis. Two days into her admission she developed acute lumbar pain and clinical signs of cord compression. Urgent magnetic resonance imaging revealed a spontaneous spinal epidural haematoma causing cord compression. This article reviews the literature on this condition and discusses its management.

Discussion

In the absence of any trauma, as in this case, the main differential diagnoses of acute lumbar pain include metastatic spinal cord compression, acute spinal cord ischaemia, transverse myelitis, a spontaneous spinal epidural haematoma or epidural abscess. In view of the patient's past medical history, spinal cord compression as a result of metastatic disease was high on the differential diagnoses, although the presentation of this is usually more subacute (days to weeks).

Epidural abscess also usually presents more subacutely, and in the absence of a significant inflammatory response (pyrexia, inflammatory markers) was felt to be less likely.

Acute spinal cord ischaemia is usually marked by an acute onset, often heralded by sudden and severe spinal (back) pain. This is often associated with bilateral weakness, paraesthesia and sensory loss. Loss of sphincter control with hesitancy and inability to void or defecate becomes evident within a few hours (Weber et al, 2009). This patient certainly had risk factors for this.

Transverse myelitis describes a heterogeneous group of inflammatory disorders characterized by acute or subacute motor, sensory and autonomic (bladder, bowel, and sexual) spinal cord dysfunction (Frohman and Wingerchuk, 2010). The clinical signs are caused by an interruption in ascending and descending neuro-anatomical pathways in the transverse plane of the spinal cord, and a resulting sensory level is characteristic of the syndrome. Lower back pain is also a recognized symptom. Transverse myelitis most often occurs as an autoimmune phenomenon after an infection or vaccination (accounting for 60% of cases in children), or as a result of a direct infection, an underlying systemic autoimmune disease or an acquired demyelinating disease such as multiple sclerosis. Symptoms and signs of transverse myelitis typically evolve over the course of hours to days and are usually bilateral. Although the disorder can develop at any age, there is a bimodal peak in incidence at 10–19 years of age and at 30–39 years (Frohman and Wingerchuk, 2010). In view of this patient's age, this would be a less likely cause of her presentation.

Spontaneous spinal epidural haematoma is a relatively rare but significant spinal condition. The yearly incidence is thought to be approximately 0.1 per 100 000 people (Chan et al, 2004; Bisson et al, 2007). Spinal epidural haematoma was first described by Jackson (1869). Certain precipitating factors, including anticoagulant therapy for prosthetic cardiac valves, therapeutic thrombolysis for acute myocardial infarction, haemophilia B, factor XI deficiency, long-term aspirin use as a platelet aggregation inhibitor, and vascular malformation, have all been suggested to correlate with spontaneous spinal epidural haematoma (Bisson et al, 2007; Solheim et al, 2007). In up to 40% of cases of spontaneous spinal epidural haematoma, no aetiological factor can be identified as the cause of bleeding. Cases related to anticoagulant therapy and vascular malformations are the second and third most common categories (Kreppel et al, 2003).

Spontaneous development of spinal epidural haematomas has a male:female ratio of 1.4:1 (Poonai et al, 2007). It is most frequent after the fourth or fifth decade (Guzel et al, 2007), but has been reported in all age groups.

Case Report

An 85-year-old woman was admitted via the emergency department with reduced mobility and general malaise. Her medical history included chronic obstructive pulmonary disease, previous bronchial and bladder carcinoma, angina and pulmonary embolism. On examination, baseline observations were normal. Cardiovascular, respiratory and gastrointestinal examinations were within normal limits. She was frail and generally weak but there was no focal neurological deficit. She was mobile with the assistance of one. Baseline bloods were normal except for a minor elevated white cell count of $13.6 \times 10^9/\text{litre}$. Her urine dipstick was positive for nitrite and leucocytes. She was started on trimethoprim for a possible urinary tract infection and low molecular weight heparin for venous thromboembolism prophylaxis. She was assessed by the physiotherapists, who felt that inpatient rehabilitation would be required.

Two days into her admission, she developed acute lumbar pain in the middle of the night and was hypertensive and tachycardic. She was given analgesics by the on-call doctor which relieved the pain to a degree. The following morning she complained of pain in her lower abdomen and weakness of her legs. Examination revealed a palpable bladder, reduced sensation with a sensory level to T10 and reduced power (2/5) globally in her lower limbs. Per rectum examination revealed a lack of anal tone and she was catheterized with a 1 litre residual volume.

Urgent magnetic resonance imaging of her thoracolumbar spine was arranged, which showed an acute epidural haematoma (Figures 1a and b).

After discussion with the neurosurgical team, in view of her multiple comorbidities, general frailty and high operative risk, it was decided to manage the patient conservatively. She regained some power in her legs but essentially remained wheelchair and bed-bound and was discharged home with a full care package in place.

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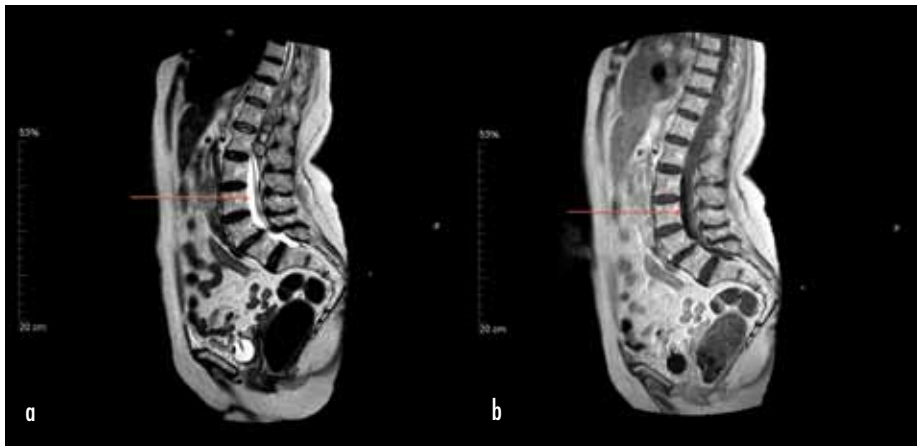


Figure 1. a. T2 weighted image and (b) T1 weighted image showing an 11 x 11.5 cm abnormality in the spinal canal extending from T9/10 down to L1/2, with predominantly intermediate signal mass, most likely representing reasonably acute blood. It appears to arise in the extradural space posteriorly and causes significant spinal stenosis with compression of the lower spinal cord, conus and cauda equina. There is no evidence of bony metastatic disease.

Magnetic resonance imaging is the first choice diagnostic method for spontaneous spinal epidural haematoma (Matsumura et al, 2007). The most common site of a spontaneous spinal epidural haematoma is the cervico-thoracic region or thoracolumbar region (Poonai et al, 2007). Early surgical intervention is the general treatment for spontaneous spinal epidural haematomas (Matsumura et al, 2007). The procedure includes decompressive laminectomy and haematoma removal. In cases with incomplete neurological deficits, surgery should be performed within 48 hours of the onset of the initial symptoms; if complete it should be done within 36 hours (Bose et al, 2007). The results of operative decompression depend on the duration of symptoms, the level of preoperative neurological deficit and time lost during diagnostic procedures (Poonai et al, 2007; Liu et al, 2008). Many authors have found that the speed of surgical intervention correlates with better neurological and functional recovery. A time frame of less than 12 hours from the initial incident seems to be the best therapeutic window.

Conservative treatment has also been documented, and in the past decade an increasing number of spontaneous epidural haematomas have been treated conservatively. It is generally only considered in mild cases with a benign course and provided that the patient is followed up neurologically and with repeated magnetic resonance imaging (Groen, 2004). However, there are reports of patients with severe neurological deficit after spontaneous spinal epidural

haematoma who were managed conservatively because of the coexistence of a serious coagulopathy and/or the anticipated risks of operative treatment (Garcia Lopez et al, 1999). Other reported reasons for not operating were patient refusal (Saito et al, 1994), and the presence of tetraplegia for more than 7 days at the moment the diagnosis was clear (Muranjan and Deshmunkh, 1999). Operative decompression of neural structures and removal of the haematoma continues to be the most appropriate treatment in most patients with spontaneous spinal epidural haematoma.

Conclusions

With an increasing number of hospitalized patients being given low molecular weight heparin for venous thromboembolism prophylaxis and an ever-ageing population who may also be on antiplatelet therapies, it is important to remember spontaneous spinal epidural haematoma and to act quickly to prevent serious neurological deficit. **BJHM**

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LEARNING POINTS

- In the absence of trauma, the main differential diagnoses for acute lumbar pain with cord compression include metastatic disease, acute spinal cord ischaemia, transverse myelitis, spontaneous spinal epidural haematoma or epidural abscess.
- Spontaneous spinal epidural haematoma is a relatively rare but significant spinal condition.
- Early surgical intervention is the general treatment for spontaneous spinal epidural haematomas to prevent serious permanent neurological deficits
- It is important to be mindful of spontaneous spinal epidural haematoma and to act quickly to prevent serious neurological deficit.