Spontaneous spinal epidural haematomas
and the prognostic implications of interval to surgical decompression: a report of two cases

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INTRODUCTION

Spontaneous spinal epidural haematomas (SSEHs) are rare. Patients with thoracic SSEHs usually present with acute pain over the thoracic spine, with rapid progression of lower limb weakness. Emergency decompression laminectomy and haematoma evacuation may prevent permanent neurological deficits.

We present 2 cases of thoracic SSEHs with similar magnetic resonance imaging (MRI) features. One presented with a motor deficit at the outset, whereas in the other it only manifested later. The interval between onset and surgical decompression greatly influences the prognosis for neurological recovery.

CASE REPORT

Case 1

In November 2006, a 42-year-old man presented with a one-day history of acute pain in the thoracic spine, with weakness and numbness developing rapidly in both legs and acute urinary retention. He
had undergone an aortic valve replacement for acute infective endocarditis 6 months earlier and was on oral anticoagulants (coumadin 5 mg daily). His blood pressure was 120/63 mm Hg and his lower limb power was 0/5 bilaterally, with loss of sensation to light touch and pinprick from T6 dermatome distally. His lower limb reflexes were brisk and ankle clonus was present. His anal tone was lax with perianal anaesthesia. The upper limb examination was unremarkable.

His prothrombin time and international normalised ratio were 22.4 (normal range, 9.2–11.2) seconds and 2.23 (therapeutic range for anticoagulants, 2–3.5) respectively. MRI revealed a 5-cm-long thoracic epidural haematoma extending from T2 to T4 vertebrae, measuring 1.2x0.8 cm in the axial plane, compressing the thecal sac and spinal cord (Fig. a). The haematoma was located at the 2 to 7 o’clock position, and occupied up to 50% of the spinal canal in some places.

The coumadin was stopped and the anticoagulant effect reversed with intravenous vitamin K and fresh frozen plasma transfusion before surgery. An emergency decompression laminectomy (T2 to T4) and haematoma evacuation was performed 51 hours after presentation, when his prothrombin time and international normalised ratio had fallen to 12.7 seconds and 1.25, respectively. Cerebrospinal fluid pulsations were visualised upon completion.

On postoperative day 1, there was patchy return of sensation to the left leg and return of power (3/5) to the right leg. On day 7, the power had recovered to 4-/5 and 3/5 for the right and left legs, respectively. Anticoagulation with low-molecular-weight heparin

Figure  Cases (a) 1 and (b) 2: sagittal and axial T2-weighted magnetic resonance images showing epidural haematomas (arrows) extending from T2 to T4 compressing and displacing the thecal sac and spinal cord anteriorly.
was started 6 days after surgery and converted to oral coumadin on day 9 (4.5 mg daily). He was enrolled in a supervised rehabilitation programme. At the 2-month follow-up, the patient was able to squat and ambulate unassisted, with a power of 4+/5 in both legs. At 15 months, his lower limb power was still 4+/5. He had no haematoma recurrence after resumption of anticoagulation.

Case 2

In February 2007, a 33-year old Filipino woman presented with an 8-hour history of acute upper back and neck pain. She had essential hypertension and a history of pre-eclampsia. Her blood pressure was 210/114 mm Hg and her lower limb power deteriorated to 0/5 from 4/5 bilaterally during the 5 hours she spent in the observation ward (Table). She had loss of sensation to light touch and pinprick from T8 dermatome distally from the onset, with lower-limb areflexia and preserved anal tone.

Her prothrombin time, activated partial thromboplastic time and platelet count were all normal. MRI revealed a thoracic epidural haematoma (from the 4 to 10 o'clock position) extending from T2 to T4 vertebrae compressing the thecal sac and spinal cord (Fig. b).

Urgent antihypertensive therapy was administered and an emergency decompression laminectomy (T2 to T4) and haematoma evacuation was performed 14 hours after the onset of symptoms.

On postoperative day 1, her distal left leg power returned to 4/5, although the power in her proximal left leg and right leg remained at 1/5 and 0/5, respectively. By day 7, she had full power (5/5) in the left and proximal right legs and mildly reduced power (4+/5) in her distal right leg. She was discharged on day 9 and returned to her home country for further rehabilitation. At the 15-month follow-up, she had recovered full power and had no residual functional limitation or haematoma recurrence.

DISCUSSION

SSEHs are rare, with an incidence of approximately 1 in 1 million individuals per year.\(^1,2\) They are more common in males (approximately 2–3:1) aged 42 to 52 years.\(^2–4\) The upper thoracic spine is most commonly affected, which has been attributed to the reduced spinal canal dimensions (compared with the lumbar and cervical regions) and lack of collateral arterial supply (making it more susceptible to ischaemia).\(^5\) Most haematomas measure approximately 3.6 vertebral levels in length, and are located dorsal to the spinal cord,\(^4\) thought to be due to firm adherence of the dural sac to the posterior longitudinal ligament in its ventral aspect,\(^6\) owing to the presence of anterior dural (Hoffman) ligaments.\(^7\)

A common aetiology is bleeding from disrupted valveless epidural veins secondary to raised intra-abdominal pressure or increased physical activity.\(^4,6\) The 3 major factors involved are: (1) bleeding diathesis stemming from oral anticoagulants (as in patient 1), antithrombotics/thrombolytics, and inherited disorders such as haemophilia;\(^3\) (2) raised intravascular pressure from essential hypertension (as in patient 2), straining (sneezing, lifting, Valsalva manoeuvre), and pregnancy;\(^2–4\); and (3) vascular pathology including spinal vascular (malformations or vasculitis) or hypervascularity states (e.g. Paget’s disease). Nonetheless, hypertension may not be causative; its presence may be coincidental or be a response to severe back pain.\(^8\) Acute back pain is indicative of cord compression. Weakness and numbness then ensues and may progress rapidly to paraparesis, quadriplegia, cauda equina syndrome, or hemiparesis, depending on the level of the lesion.\(^1–3,9\)

MRI is the best means of investigating the nature, size, location, and position of the lesion, and the degree of spinal cord compression and oedema.\(^3,6,10\) Myelography is an invasive alternative that can neither reveal the nature of the compression nor its true extent. Spinal computed tomography is not ideal in the thoracic spine, where resolution is poor because of the high contrast between lung parenchyma and vertebral bone.\(^3\)

SSEH is a surgical emergency. In young patients with mild, non-progressive or rapidly improving symptoms, a trial of non-operative management may succeed.\(^1,10\) In both of our patients, serial physical examinations just prior to anaesthetic induction revealed persistent dense sensorimotor deficits, precluding non-operative treatment. Prompt and aggressive surgical decompression and haematoma evacuation is the only way to avert a permanent neurological deficit.\(^1–3,5,9\) In patient 1, the bleeding diathesis had to be corrected prior to surgery in order to reduce the haematoma expansion and facilitate

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<th>Lower-limb power</th>
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<th>2.25 h</th>
<th>3.75 h</th>
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<td>Right</td>
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surgical exploration. The interval to surgery is a delicate balance between taking the time necessary for correction of the bleeding tendency and limiting the period of cord compression. From a comprehensive review of 333 cases, it is recommended that surgery be performed within 36 hours for complete neurological deficits, and within 48 hours for incomplete deficits. Other critical intervals range from 8 to 48 hours. Regardless of the differences in the optimal surgical interval, early surgery is indicated even in patients with complete or long standing sensorimotor deficits. The efficacy of less invasive options such as percutaneous drainage (both inadvertent and intentional) has yet to be compared with surgical decompression.

The prognosis for neurological recovery strongly depends on the preoperative neurological status and the interval to surgery. In patients with complete neurological deficits, the prognosis is less favourable. Localised (single-level) haematomas and a lumbosacral location usually have a more favourable outcome. In patients with associated risk factors who present with atraumatic back pain, a high index of suspicion is necessary for early diagnosis. In patient 1, a longer interval to surgery was unavoidable owing to the presence of coagulopathy, which needed to be corrected as quickly as possible. In patient 2, it was possible to control the elevated blood pressure rapidly, enabling a shorter interval to surgery.

CONCLUSION

In the management of SSEHs, a longer interval to surgery is detrimental to a positive neurological outcome. In patients presenting with acute back pain followed by rapidly evolving symptoms of spinal cord compression, an urgent MRI is indicated. Where there is coagulopathy, the surgeon has to weigh the time needed for correction against the need for urgent decompression, and decide whether to correct completely, partially, or not at all.

REFERENCES