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Spontaneous spinal epidural haematoma — an unusual cause of spinal cord compression

自發性脊椎硬腦膜外血腫——一宗脊椎壓縮的異常病例

Spinal epidural haematoma is uncommon. Spontaneous spinal epidural haematoma of unknown aetiology is a rarity. This report is of two patients with spontaneous spinal epidural haematoma with different outcomes and discussion of the clinical picture and possible aetiology of this rare condition.

脊椎硬腦膜外血腫是一種罕見的疾病，而因不明病原引起的自發性脊椎硬腦膜外血腫的情況更為罕有。本報告報導兩宗脊椎硬腦膜外血腫的病例，以及病人的不同結果；並討論病例的臨床照片及這種罕見情況的可能病原。

Introduction

Spinal epidural haematoma (SEH) is rare. Approximately 300 patients have been reported in the literature and theories about the aetiology are diverse. Spinal epidural haematoma is a serious condition and early treatment confers a significant prognostic advantage. Spinal epidural haematoma may be primary or secondary—the majority of cases are secondary to other underlying causes such as trauma, anticoagulant therapy, vascular anomalies, blood dyscrasias, and epidural anaesthesia.¹ Primary or spontaneous SEH (SSEH) is of unknown aetiology and the pathogenesis remains unclear.² Spontaneous recovery by these patients has been reported³ but the standard treatment remains surgical decompression and evacuation of the blood clot. Spontaneous SEH should be considered as one of the differential diagnoses of cord compression when more common causes have been excluded.

Case reports

Case 1

A 77-year-old woman presented to the Department of Medicine of a local hospital in November 1997 with interscapular pain 2 hours after performing household work. The pain initially radiated down the left leg followed by pain down the right leg. There was associated paraesthesia and weakness of both lower limbs. The patient had no history of trauma or anticoagulation therapy. She was hypertensive but not receiving regular medications.

Physical examination at admission to hospital showed a conscious woman with a blood pressure of 190/100 mm Hg. Her upper limbs were normal. Her lower limbs were completely flaccid with Medical Research Council (MRC) grade 0 muscle power and absent reflexes. The urinary bladder was palpable and the anal sphincter was lax. There was complete loss of all sensory modalities from T4 downwards.

Investigations showed that the complete blood picture and the clotting profile were normal. X-ray of the spine was unremarkable. Orthopaedic surgeons were consulted the next day and acute cord compression was suspected. Since magnetic resonance imaging (MRI) was not readily available in the hospital, an urgent computed tomography (CT) myelogram was performed

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關鍵詞：

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Fig 1. Myelogram film of case 1 (anteroposterior view)

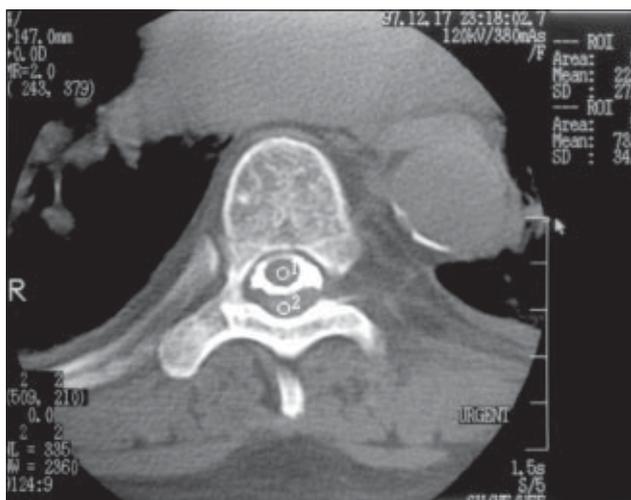


Fig 2. Computed tomography scan of case 1 demonstrating epidural haematoma

instead. The CT myelogram was done 28 hours after admission and showed an extradural space-filling defect that extended from C7 to T3 with tapering ends (Fig 1). The corresponding CT scan showed a homogenous haematoma causing extradural compression (Fig 2). A diagnosis of acute cord compression was made. Emergency decompression was performed 34 hours after admission.

Intra-operatively, a large dark gelatinous clot from C7 to T3 was seen in the epidural space. Profuse bleeding was encountered. There were prominent blood vessels outside the dura mater that were coagulated. After the blood clot was removed, the dura mater appeared to be free with good dural pulsation. The epidural bleeding was controlled with some difficulty. Laminectomy of C7 to T3 was performed. Histological examination revealed an organising thrombus.

Postoperatively, the patient had a complicated recovery due to continuous oozing of the wound. Exploration of the wound using general anaesthesia was performed 1 week later. A haematoma of 50 mL was drained from the subcutaneous plane beneath the wound. Coagulation of the subcutaneous bleeding vessels was performed using diathermy. The deep wound was then explored. The dura was free from any compression and the laminectomy was judged to be

adequate. Postoperative recovery was uneventful. The patient remained paraplegic, however, and required a wheelchair and long-term urinary catheterization at follow-up after 4 years.

Case 2

A 52-year-old woman presented in February 1996 with sudden onset of back pain, bilateral lower limb paraesthesia, and weakness without any history of recent trauma. She had chronic renal failure that required haemodialysis for the past 5 years. Six months prior to admission to hospital, she had endometrial carcinoma and total hysterectomy and bilateral salpingo-oophorectomy were performed.

Physical examination at the time of admission showed that the patient was in pain and there was diffuse tenderness over the lower thoracic spine. Both upper limbs were normal. There was weakness of both lower limbs of MRC grade 3 muscle power. Her reflexes were diminished in both lower limbs. There was a decrease in pinprick sensation below T10. The urinary bladder was not palpable but the anal sphincter was lax.

Her haemoglobin level was 82 g/L (normal range, 120-150 g/L), which was compatible with her renal status. The white cell count, platelet count, and clotting profile were all normal. Renal function was impaired with an elevated creatinine level of 480 $\mu\text{mol/L}$ (normal range, 53-106 $\mu\text{mol/L}$).

Computed tomography and lumbar myelogram was performed 12 hours after admission. There was a homogenous fluid collection from T8 to T10 extradurally, which was causing cord compression. This fluid had a higher density than cerebrospinal fluid.

Decompression was performed 15 hours after admission. Intra-operatively, there was a large brownish gelatinous clot extending from T8 to T10 in the epidural space—this was evacuated. The epidural vessels appeared to be engorged and were coagulated. Laminectomy was performed from T8 to T10. The spinal cord appeared to have good pulsation afterwards. The postoperative course was uneventful. The patient regained full muscle power of both lower limbs. The paraesthesia had largely diminished and the patient ambulated independently 1 year after the operation.

Discussion

Spontaneous SEH is a rare condition that has been reported to occur in different age-groups, from paediatric patients to elderly people.⁴ The presenting symptoms vary. Radicular pain is one of the earliest symptoms. The clinical picture is typical, in that, without preceding trauma, patients experience an acute onset of local pain, sometimes with radicular paraesthesia. Within hours, signs of spinal cord compression may appear, which present with progressive paraplegia and loss of sensory function.⁵ In this series

both patients appear to have followed the same pattern of presentation.

The aetiology of SSEH remains unclear. By definition, SSEH excludes that due to obvious trauma, anticoagulant therapy, blood dyscrasias, and spinal haemangioma.⁶ The need for histological examination cannot be over-emphasised in view of the possible aetiologies. The consensus of most authors, however, is that spontaneous haematoma is venous in origin. In the loose areolar tissue of the epidural space, there is an extensive system of veins that do not contain valves. Sudden increases in intrathoracic or intra-abdominal pressure associated with coughing, straining, sneezing, or lifting a heavy object may distend the veins causing them to rupture. The ratio for men to women is 1.5:1, and approximately half the patients are between the ages of 50 and 80 years.⁴

Before the advent of MRI, CT with opaque myelogram was routinely used to diagnose such emergency conditions. Four distinct CT appearances have been described⁷:

- (1) a large asymmetrical epidural mass that is inseparable from the dura;
- (2) a rounded epidural mass situated centrally in the epidural fat;
- (3) a narrow asymmetrical biconvex mass based on the dura; and
- (4) a narrow asymmetrical thickening of the posterior epidural fat.

Myelogram has been reported to aggravate spinal cord compression and may cause spinal clonning in 14% of cases,⁸ so MRI has replaced CT myelogram almost exclusively. On sagittal sections of MRI, the SSEH appears as a biconvex mass, dorsal to the thecal sac, clearly outlined, and with tapering superior and inferior margins. The dura mater has a curvilinear low signal separating the haematoma from the cord. Within 24 hours of onset, the haematoma is isointense with the cord on T1-weighted images and heterogeneous on T2-weighted images. Later, the haematoma gives high signal intensity on both T1- and T2-weighted images.⁹

Conservative treatment has been described for SSEH and spontaneous resolution and repeated relapses have been reported.^{3,10} For selected patients with incomplete and non-progressing deficit, conservative management may be possible. Surgery is nevertheless indicated for patients with progressive neurology and complete cord compression. In a review article, Groen and van Alphen² suggested that for patients with incomplete sensorimotor deficit, favourable outcomes correlated highly with surgery within 48 hours. This critical time is sharply reduced to 12 hours for patients with complete sensorimotor deficit.

All these points are well illustrated in these two patients. Both patients had pain first followed by neurological deficit. Blood investigations did not reveal any bleeding diathesis. Computed tomography and myelographic appearances were suggestive of epidural compression. Treatment was decompression and laminectomy.

Both patients were operated by the same spinal surgeon who has more than 20 years' experience. The first patient had complete paraplegia preoperatively. After several inter-departmental consultations and further investigations, she received surgical decompression 34 hours after admission. She had no significant recovery. The second patient had paraparesis preoperatively and was operated on 15 hours after admission. Her recovery was almost complete. It is therefore vital to recognise this condition early and perform emergency decompression. Histological examination is mandatory to exclude other pathologies.

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