

CASE REPORT

Spontaneous Spinal Epidural Hematoma: Unusual Emergency and its Management

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ABSTRACT

Spontaneous spinal epidural hematoma (SSEH) is one of the causes of sudden and profound neurological compromise. Although half of the cases have no aetiology, many medical and traumatic conditions have been incriminated for developing a hematoma. Neurological compromise range from mild back pain to complete paralysis. Timely intervention may revert neurological deficits in selected patients. Here we present a case of sudden onset paraplegia in an 18 year old male due to SSEH. The patient remarkably improved in neurology with surgical intervention.

Keywords: Epidural hematoma, Spinal cord compression, Paraplegia, Spinal emergency

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BACKGROUND

Spontaneous spinal epidural hematoma (SSEH) is defined as sudden epidural bleeding without a known cause, traumatic or iatrogenic^{1,4}. SSEH is one of the causes of sudden and profound neurological compromise. It is relatively rare and accounts for less than 1% of space occupying lesions in the spinal canal^{1,5}. Clinical presentations of the condition vary from simple sudden onset back pain to complex neurological symptoms of radicular pain, limb weakness, urinary retention etc. depending on the size and level of the lesion⁶.

As this rapidly progressive hematoma in epidural space results in devastating neurological deficits within a short span of time, hours to days, it is vital to intervene earliest possible for better outcomes^{7,8}.

Literature available cited coagulopathy, malignancy, vascular anomalies, infections as potential causes for SSEH, though in many cases etiology is unknown².

We are presenting the case of a young male presented to the emergency department with a history of sudden onset of both lower limb weakness and urinary retention due to SSEH in the dorsal spine.

CASE PRESENTATION

A 18 year old male, student presented to the emergency department with complaints of sudden onset of back pain in the night 2 days back and bilateral lower limb weakness and sensory deficits, noticed the following morning when tried to get up from bed. He also noticed that he couldn't pass urine even after straining. He has burning sensation in both lower limbs. There is no history of trauma, fever, weight loss, loss of appetite. Patient has no known comorbidities and past history not significant.

On examination, the patient has mild tenderness in the upper dorsal region. Neurologically, bilateral upper limbs are normal with full power, intact sensations and deep tendon reflexes. Abdominal reflexes absent. Sensations for pain, touch and pressure decreased from D7 level downwards including both lower limbs and perineal regions. Power in both lower limbs is 0/5, as per Medical Research Council grading, around hips, knees, ankle and fingers. Bilateral knee, ankle and plantar reflexes are mute. Voluntary anal

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contraction and bulbocavernosus reflex are absent.

Investigations for blood parameters showed normal liver and kidney functioning. Hemoglobin, white cell count, platelet count, erythrocyte sedimentation rate, C- reactive protein levels were within normal limits. Coagulogram revealed mild elevation in activated partial thromboplastin time (APTT), with normal prothrombin time and international normalized ratio.

Plain radiographs of the dorsolumbar spine were unremarkable. Magnetic resonance investigation (MRI) revealed oblong extradural collection, mild hyper intense on T1 and hypo intense on T2 imaging, extending from D1 to lower border of D4 vertebrae (measuring 10*73mm)), likely hematoma, causing canal narrowing with cord compression. T2 stir hyper intensity noted in cord at D2, D3 level suggestive of cord edema (figure 1). No vascular abnormality detected on spinal angiography.



Fig. 1: Magnetic resonance investigation(MRI) revealed oblong extradural collection, mild hyper intense on T1 and hypo intense on T2 imaging, extending from D1 to lower border of D4 vertebrae (measuring 10*73mm)), likely hematoma, causing canal narrowing with cord compression. T2 stir hyperintensity noted in cord at D2,D3 level suggestive of cord edema

Surgical management

Based on clinical and investigation findings, the final diagnosis was made as idiopathic SSEH from D1-D4 level with complete paraplegia and bladder involvement.

Decompressive spinal surgery is considered. Under general anaesthesia and prone position, D1 to D4 spinous process identified and midline skin incision given. Paraspinal subperiosteal dissection done to expose D2-D3 lamina. D2, D3 laminectomy done. Red colour granulation tissue was found in epidural space, which was removed meticulously with penfields to achieve decompression of cord (figure 2). Dura found to be intact. Thorough wash given. Underside of D1 and D4 lamina were found to be free without cord compression. Closure done in layers, over suction drain. Tissue obtained sent for histopathology study.

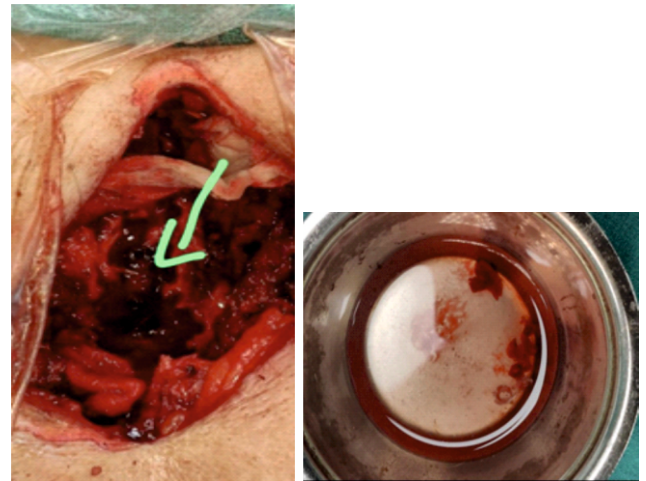


Fig. 2: Red Colour granulation tissue was found in epidural space, which was removed meticulously with pen fields to achieve decompression of cord intra operative finding.

Post-operative management

Postoperative supportive care is given to the patient. Antibiotics were given as per institutional protocol. Analgesics and neurotropic vitamins were given. With clinical haematology consultation, warfarin started at 2 mg once daily to prevent deep vein thrombosis. The patient showed improvement in the burning sensation of limbs on postoperative day 1. On day 2, an aseptic dressing of the surgical site was done and the suction drain was removed. In-bed physiotherapy started from day 1 and bladder training with clamping of foley catheter was taught to the patient and attenders. The patient was discharged on day 3 with the advice of physiotherapy and bladder training.

On 2 2-week follow-up, the patient has the power of $\frac{3}{5}$ grade at the hip, knee and ankles of both lower limbs. The patient has improvement in sensory symptoms.

DISCUSSION

Spinal hematoma ms can be categorised into subdural, epidural, subarachnoid and intramedullary with epidural being most common. Although there is confusion in the definition of SSEH, spinal epidural hematoma occurring without trauma is considered SSEH². Spontaneous spinal epidural hematoma is a rare but significant cause of sudden neurological deficit. Most commonly seen in men, in our case, with a 1.4:1 male-to-female ratio^{9,10}. Contrary to age in the present case, SSEH is mostly presented after the 4th or 5th decade¹¹. Junction of rigid and mobile spine, cervicodorsal and dorsolumbar, which have high stress are most commonly affected^{11,12}. The D1 to D4 epidural hematoma of our case can be considered as a junctional region. On evaluation, the cause for bleeding could not be established in our patient and on literature search no aetiology was found in approximately 40% of cases⁹. Some of the potential causes are prolonged aspirin or anti-platelet or anticoagulant therapy, haemophilia B, and vascular malformations^{13,14}. Keeping aside the aetiology, the origin of hematoma, whether it is venous or arterial is debatable. As the epidural venous plexus is thin-walled, delicate and valveless, a sudden rise in abdominal or thoracic pressure can directly be transmitted to this plexus resulting in bleeding. Thus activities like lifting heavy weights, coughing or sneezing can cause epidural bleeding in such scenarios^{9,11}. Some authors proposed arteries as a source of epidural hematoma¹⁴. Unlike venous pressure, pressure in arteries is more than intradural pressure, making arterial cause to be more likely¹⁵.

SSEH results in sudden back pain with radiation to limbs depending on the amount of cord compression and extent of oedema. Neurological deficit is acute, hours to days. The extent of neurological deficits depends on the amount and duration of compression, resulting in complete or incomplete deficits. Currently, MRI is the diagnostic method of choice⁵. Additional imaging with angiography is advised in case of vascular malformations². Promising results were seen with early surgical decompression, within 36 hours, even in complete deficit patients¹⁶. However, some authors advise conservative treatment even with a

complete deficit^{9,17}. Though our patient presented 2 days after symptom onset, we could perform decompression in our case within 48 hours of admission as the patient is young and has no medical comorbidities. Immediate postoperative improvement in sensory symptoms is seen. In consonance with spinal cord trauma, the most crucial prognostic tool for neurological recovery is the severity of preoperative neurological compromise^{3,4}.

CONCLUSION

Spontaneous spinal epidural hematoma is rare but a neurosurgical emergency. As soon as the coagulopathy cause of bleeding is excluded, decompressive surgery should be performed for better and faster recovery.

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Written Informed consent: Taken

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