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CASE REPORT

Spontaneous Spinal Epidural Hematoma – A Case Report and Literature Review

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Spontaneous spinal epidural hematomas (SSEH) are infrequent, with an incidence of about 0.1 per 100,000 annually. Representing <1% of all spinal canal lesions, they typically manifest without evident traumatic or iatrogenic origins. This study aims to provide a comprehensive understanding of SSEH by analyzing a case of SSEH perform aortic dissection-like syndrome and was diagnosed with SSEH by MRI, who was treated successfully through surgical intervention.

Key words: Spinal epidural hematoma, spontaneous epidural hematoma, spinal hematoma

INTRODUCTION

Spontaneous spinal epidural hematomas (SSEH) are infrequent, with an incidence of 0.1 per 100,000 annually. Representing <1% of spinal canal lesions, typically manifest without evident traumatic or iatrogenic origins.

Historically, Jackson first described SSEH in 1869.[1] Patients often experience acute onset of back or neck pain, then evolves into progressive motor, sensory disturbances, and potentially, full or partial motor deficits due to spinal cord or nerve root compression.[2]

The consensus regarding the time for surgical intervention remains debated. While many authors propose decompressive surgery within 12 h as to yield better results, a recent study suggests that factors such as preoperative neurological severity and use of anticoagulant play a more pivotal role in determining outcomes than the time elapsed since symptom onset.[3]

Regardless of the etiology, the critical treatment prompts the surgical decompression and hematoma evacuation.[4]

While some reports indicate successful conservative treatment. Our study aims to provide a comprehensive understanding of SSEH by analyzing a case presented with aortic dissection-like syndrome who was diagnosed by spinal magnetic resonance imaging (MRI).

CASE REPORT

An 80-year-old female presented to the emergency department in the morning, reporting a sudden onset of chest pain radiating down to the back, accompanied by cold sweating. Subsequently, she developed numbness and weakness in her lower limbs, rendering her unable to walk or stand. She had only a medical history of hypertension. Initial investigations included chest CT angiography, which ruled out the aortic dissection or rupture of abdominal aneurysm.

On further evaluation in the emergency department, progressive lower limb numbness persisted. Neurological examination demonstrated positive Babinski's sign. Muscle strength was graded based on the Medical Research Council Manual Muscle Testing scale. Sensory examination revealed impairment below T8. The spinal MRI identified a heterogeneous extradural lesion from T7 to T11, contributing to the spinal cord compression [Figures 1 and 2].

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The patient underwent an emergency laminectomy from T7 to T11, in which an epidural hematoma was identified and evacuated. Postoperatively, the strength of the lower limbs significantly improved (grading between 4 and 5), and previous refractory back pain subsided. Following a 2-week recovery period, she had no neurological deficits and was discharged uneventfully.

DISCUSSION

The term "spontaneous" signifies its atraumatic origin, which is attributed to factors such as hemophilia, neoplasms, arteriovenous malformations, hypertension, anticoagulant use, or activities such as lifting and sneezing. Interestingly, only 40%–60% SSEH are idiopathic.[5]

A dominant theory posits the venous system as the main bleeding source, suggesting increased abdominal or thoracic pressures could cause spinal epidural vein ruptures. This is consistent with the fact that 54% of SSEH reported an event involving strain during the initial episode.[6]

SSEH is a challenging diagnosis to confirm, especially before the trivial neurological deficit. The differential diagnoses include pulmonary emboli, spontaneous pneumothorax, and acute myocardial infarction. However, in the presence of neurological symptoms, differential diagnoses also consider transverse myelitis, Guillain–Barré syndrome, epidural subarachnoid hemorrhage, and acute spinal cord ischemia. Other potential conditions include acute herniated intervertebral disc, epidural tumors or abscesses, spondylitis, and even a dissecting aortic aneurysm.[7,8]

SSEH arises after procedures such as lumbar punctures or epidural anesthesia. Several factors increase the risk of SSEH, including thrombolysis, anticoagulation (about 17%), thrombocytopenia, blood disorders, tumors, coagulopathies, and underlying vascular anomalies.[9] Various medications have been reported to be associated with SSEH, including antiplatelet, anticoagulant, or thrombolytic medications, such as aspirin, warfarin, heparin, tissue plasminogen activator, and streptokinase. Interestingly, individuals with a prior history of alcoholism or intoxication have a higher risk.[10]

The exact origin of hemorrhage in SSEH remains a debate. These hemorrhages may develop from a rupture of epidural veins, epidural arteries, or a vascular malformation. While the majority of evidence highlights the venous system, particularly the posterior epidural venous plexus, as the primary bleeding source, there is notable support for an arterial origin. The rapid development of some cases suggests an arterial source due to its higher flow rate compared to the venous system.[6] On the other hand, slow progression of symptoms would support a venous source.

MRI is the preferred diagnostic tool for SSEH, being able to visualize the location, size, and extent of the hematoma and the resultant cord compression. Typically, an MRI reveals biconvex hematomas in the epidural space with distinct borders.[11]

Within 24 h of symptom onset, the hematoma appears isointense on T1-weighted and hyperintense on T2-weighted images (WI). Beyond 24 h, it often evolves into hyperintense on both T1 and T2 WI. Chronic hematomas might be hypointense on both WI. Fat suppression images can differentiate hematomas from epidural fat, and contrast medium may highlight active bleeding within the hematoma foci.[12,13]

Early diagnosis leads to better treatment outcomes and a complete recovery. In the decision between conservative management and surgical intervention, the timing between symptom onset and decompression is crucial due to neurological reversibility. Current management strategies need enhancement, underscored by a 5.7% disease-related mortality rate.[14]

SSEH primarily warrants an early surgical intervention. The standard procedure involves decompressive laminectomy and hematoma evacuation. For cases presenting with incomplete neurological deficits, surgery should commence within 48 h. However, if deficits are wide-ranging, the timeline narrows to 36 hours.[15]

Immediate decompressive laminectomy and hematoma evacuation are advised for catastrophic neurological deficits. Preoperative neurological status can forecast postsurgical outcomes. About 42% with initial incomplete sensorimotor deficits achieve full recovery. However, the rate dips to 26% for partial sensory dysfunction and plunges to around 11% with comprehensive sensorimotor impairments.[16]

Otherwise, conservative treatment may considered for those with inappropriateness for surgery, such as coagulopathy, and the refusal of surgery. In a comparative study of surgical versus conservative management of SSEH, one-third ($n = 5$) of 15 patients were managed conservatively. There was no significant difference between the two groups. The study emphasized conservative management in patients whose present neurologic status was American Spinal Injury Association (ASIA) Scale E or in whom early recovery of function has been initiated with ASIA Scale C or D. In a review of published literature, only 10 out of 64 conservatively treated patients had incomplete recovery. It can be explained by milder neurological deficits at presentation in conservatively treated patients in comparison to those who were surgically treated.[7]

The crucial prognostic determinant is preoperative neurological status. The ASIA score [Table 1],[16] a prominent measure of neural deficits, assists in forecasting outcomes and guiding treatment decisions. Patients presenting with ASIA

scores (A or B) need immediate intervention, while moderate deficits (C or D) still necessitate urgent care, albeit without extreme time pressure.[16]

In summary, anyone who was suspected of the SSEH, especially those on anticoagulants, should undergo a prompt neurological assessment and optimal imaging. Once SSEH is confirmed, the urgency and mode of treatment should correlate with the evolving neurological status. To refine therapeutic recommendations, comprehensive research comparing both surgical and conservative treatments is imperative.

Table 1. American Spinal Injury Association (ASIA) impairment scale.

ASIA scale	Degree of neurological impairment
A	Complete: no motor or sensory function preserved
B	Incomplete: sensory but no motor function preserved below the neurologic level
C	Incomplete motor function (>50%) of the key muscles below the neurologic level, motor grade<III
D	Incomplete motor function (>50%) of the key muscles below the neurologic level, motor grade≥III
E	Normal

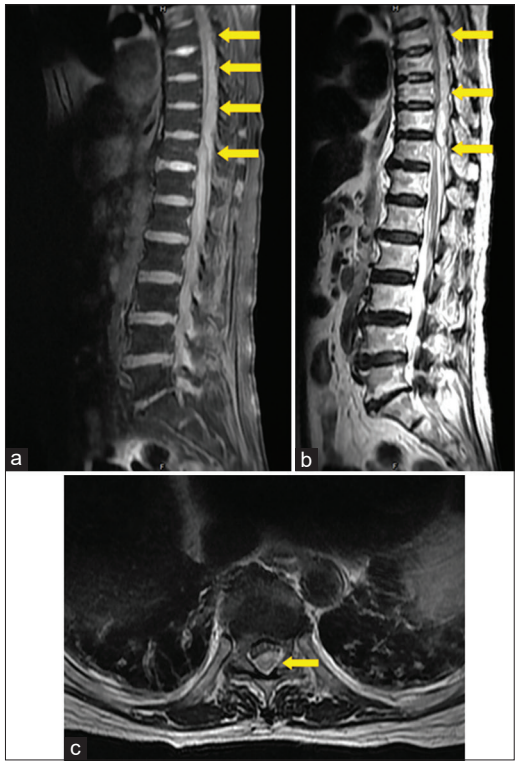

Figure 1: Magnetic resonance imaging of the lumbosacral spine. (a) T1-weighted imaging sagittal view revealing an isointense lesion in the dorsal epidural space from T3–T11, (b) T2-weighted imaging sagittal view revealing the same hyperintense lesion, (c) T2-weighted imaging axial view revealing the hyperintense lesion occupying the spinal canal and compressing the spinal cord

CONCLUSION

We describe a case of SSEH, while uncommon in adults, that can lead to severe complications, necessitating swift diagnosis and potential surgical intervention. SSEH typically presents with ambiguous symptoms, making it essential to recognize the potential signs, such as sudden neck or back pain combined with neurological deficits. The spinal MRI is the primary diagnostic tool for SSEH. Emphasis on emergent surgical intervention is of paramount importance for poor or deteriorating ASIA scores. Our case highlighted the significant recovery of the early spinal cord decompression and the well-suited rehabilitation program from a targeted center for spinal cord injury. In essence, the onset of symptoms like lower limb weakness should prompt thorough examination, even without clear risk factors. Clinicians must remain vigilant, prioritize imaging, and employ a comprehensive clinical approach based on available resources.

Declaration of patient consent

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and its

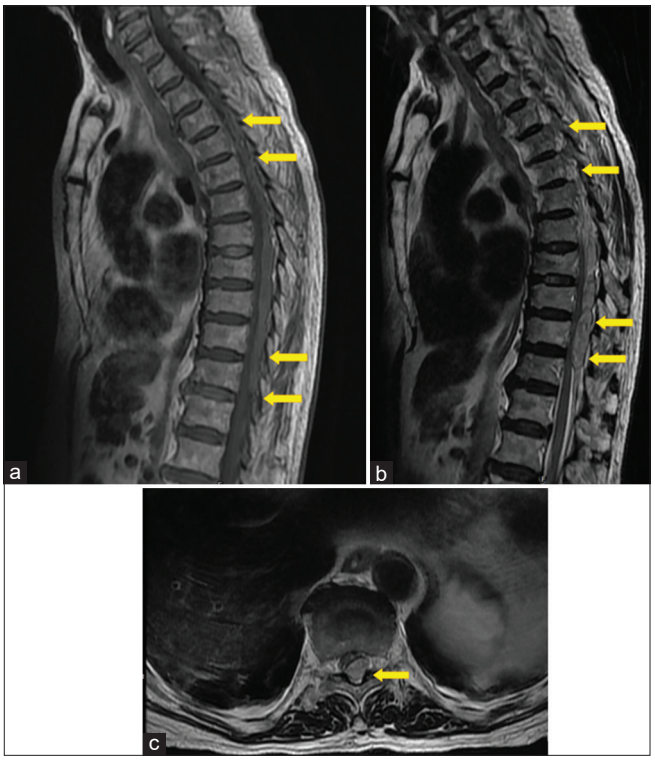

Figure 2: Four hours after the first magnetic resonance imaging of the thoracic spine. (a) T1-weighted imaging sagittal view revealing a hypointense lesion in the dorsal epidural space from T3–T11, (b) T2-weighted imaging sagittal view revealing the same isointense lesion, (c) T2-weighted imaging axial view revealing isointense lesion occupying the spinal canal and compressing the spinal cord

amendments. The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

Data availability statement

The data that support the findings of this study are available from the corresponding author, upon reasonable request.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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