

A Rare Cause of Spinal Cord Compression: Spontaneous Spinal Epidural Hematoma: A Case Report

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Abstract

Case Report

Introduction: Spontaneous spinal epidural hematoma (SSEH) is a relatively rare but significant spinal condition. Urgent surgical evacuation of a hematoma is generally indicated to prevent serious permanent neurological deficits. **Case presentation:** We encountered one case of spontaneous spinal epidural hematoma associated to a paraplegia with absent reflexes of the lower extremities and a level of hypoesthesia below the D4 segment, that were treated successfully by surgical intervention. **Conclusion:** MR imaging plays an especially important diagnostic role. Neurologic status will likely continue to dictate management. **Keywords:** Spinal epidural hematoma, Magnetic resonance imaging (MRI), Laminectomy.

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INTRODUCTION

Spontaneous spinal epidural hematoma is a rarely occurring condition. The clinical importance of compressive spinal hematoma is due to its acute and progressive course that can lead to permanent and irreversible neurological deficits if not treated early. Clinical presentation is characterized by acute radicular pain followed by cord compression syndrome. MR provides characteristic findings that allow a prompt diagnosis of acute epidural hematomas.

CASE PRESENTATION

We report the case of a 62 years old patient, followed for artery hypertension, admitted to emergency room with lower-limb heaviness. Symptoms started with the abrupt onset of cervicothoracic back pain one day before his admission. They were acute in onset resistant to commonly used analgesics, followed by lower-limb heaviness associated with genito-sphincteric disorders including acute urinary leak. Anamnesis showed no trauma or anticoagulants therapy or previous medico-surgical history. Clinical showed an aware, afebrile patient, with paraplegia. Urgent MRI of the spine showed posterior spinal extradural haematoma (epidural) extended from the seventh cervical vertebra (C7) to the seventh dorsal vertebra (D7), hyper intense in T1-weighted, hyper intense heterogeneous T2-weighted, not cleared after fat saturation, hypo intense in gradient recalled echo T2*-weighted, and not raised after gadolinium injection, with spinal cord compression with intra medullary focal well-defined hyper intense signal in the cord.

in gradient recalled echo T2*-weighted, and not raised after gadolinium injection, with spinal cord compression with intra medullary focal well-defined hyper intense signal in the cord (Fig 1). Laboratory tests, including haematic crasis were normal. The patient underwent emergency surgery via posteromedial incision with laminectomy extending from the seventh cervical vertebra up to the D7. Epidural hematoma was removed.



Fig-1: Spinal épidural h ematoma extended from C7 to D7, hyper intense in T1-weighted (a), hyper intense heterogeneous T2-weighted (b) not cleared after fat saturation (c,d), hypo intense in gradient recalled echo T2*-weighted (e), and not raised after gadolinium injection (f), with spinal cord compression with intra medullary focal well-defined hyper intense signal in the cord.

DISCUSSION

SSEH is a rare and serious disorder. It is a neurosurgical diagnostic and therapeutic emergency whose early management can determine the prognosis. Its diagnosis has become easy especially after the advent of MRI. Severity factors include acute and sudden neurological deficit onset, complete neurological deficit and chest hematoma [1]. Early neurological recovery, incomplete neurological deficit or primarily motor deficit, acervical or lumbosacral position is factors associated with a better recovery. It can be associated or not with trauma, anticoagulation therapy, blood dyscrasia, vascular malformation, neoplasm or iatrogenic complication [2-4].

The spinal epidural hematoma presents with severe and acute pain. Motor and sensory deficit develop, related to the level of the cord or cauda equina compression. Paraplegia and/or quadriplegia may result. Even death may occur [4-6]. The symptoms rarely have a slowly progressive course.

The etiology of spontaneous spinal epidural hematomas is not well understood. Venous bleeding within the valveless epidural venous plexus or arterial source of bleeding has been discussed in the literature [2, 3, 7]. Arteriovenous malformation or neoplasm can also cause this condition [4, 9].

When a SSEH is suspected, the imaging modality of choice is an MRI [8, 10]. When compared to the spinal cord within 24 h from symptom onset, the hematoma typically appears isointense on T1-weighted and hyperintense on T2-weighted MRI imaging [2, 6, 8]. After 24 h, the hematoma often appears hyperintense on both T1- and T2-weighted images [2, 8]. Chronic hematomas become hypointense on both T1- and T2-weighted images [2]. Fat suppression images may be used to distinguish hematoma from epidural fat [9]. Sometimes active bleeding into the hematoma will reveal a central area of enhancement when contrast is used [9]. Some authors have noted that occasionally an epidural hematoma will exhibit enhancement, which can be due to hyperemia of the dura or hypertrophic meninges [6, 9].

Once diagnosed, urgent surgical intervention is warranted [3, 10]. Some have suggested that for optimal neurologic improvement, patients should undergo surgical decompression within 12 to 48 h of symptom onset [3, 8, 9]. Yet, some studies have failed to prove a statistically significant difference in outcomes based solely on the time to surgery from symptom onset [10]. Given the potential for progressive deficits and that most of the available case reports are based on small numbers, the consensus is for emergent or at least urgent surgical intervention [4,8]. Many suggest decompression within 24–36 h of symptom onset for complete deficits and within 48 h for incomplete deficits [4,8]. More importantly for long term outcomes,

though, is the neurologic status of the patient prior to operative intervention [3,10]. This is the most important prognostic indicator [5, 6].

Patients who present with more severe symptoms within a shorter time frame tend to have larger hematomas; these are associated with worse outcomes, particularly when four or more spinal segments are involved [5,10]. Additionally, a lack of sensory sparing suggests a worse prognosis than an individual presenting with some degree of sensation [5]. Areas that have less space available for the cord, the thoracic spine, do not tolerate hematoma expansion well and portend a worse prognosis [5].

The treatment of choice for SSEHs typically is a hemilaminectomy or a laminectomy followed by irrigation and debridement [4, 8, 9]. If there is an obvious cause of coagulopathy, this should be addressed prior to surgical intervention [6]. If a patient is to be treated nonoperatively, the patient needs to be monitored with serial examinations while on strict bed rest [9]. Although the hemorrhage may resorb on serial MRI's even within four months [10], the outcomes typically are poor without surgical intervention. Non-operative management is reserved only for those who are not surgical candidates or who are asymptomatic [10].

For the patient in our case, we provided urgent surgical intervention and noted excellent results. There was no antecedent trauma other than a minor lifting incident, and he denied any anticoagulant use.

CONCLUSION

The clinical presentation as well as the MR imaging findings may be misleading in the diagnosis of acute (≤ 48 hours) SSEH. Small spontaneous spinal EH may mimic subarachnoid hemorrhage clinically, in which case, MR imaging plays an especially important diagnostic role. Isointensity to spinal cord on T1-weighted and heterogeneous hyperintensity to cord with focal hypointensity on T2-weighted images should suggest the diagnosis of acute SSEH. Neurologic status will likely continue to dictate management, because nonoperative treatment may be successful in cases with minimal neurologic deficits, despite cord compression on MR imaging.

Abbreviations

SSEH: Spontaneous spinal epidural hematoma.
MRI: Magnetic Resonance Imaging.

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ZB, FA, BB, MI and NI contributed equally to this article. All authors were involved in the clinical

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Ethics approval and consent to participate

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Consent for publication

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Competing interests

The authors declare that they have no competing interests.

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