**Spontaneous Spinal Subdural Hematoma: A Case Report**

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**ABSTRACT**

Spinal epidural hematomas (SEH) are a rare form of hematoma affecting the nervous axis. Spontaneous epidural hematomas are defined as the presence of blood in the epidural space without any known cause. They may be associated with coagulopathies or arteriovenous malformations. HSSE can lead to disabling neurological deficits. Imaging and rapid intervention should be initiated as soon as a patient presents with symptoms suggestive of HSSE. We report the case of a 36-year-old patient who presented with SEH in a setting of acute spinal cord compression.

**Keywords:** Coagulopathy, compression, epidural, hematoma.

1. **Introduction**

Spinal epidural hematomas (SEH) are a rare occurrence, accounting for less than 1% of all spinal canal space-occupying lesions [1], [2]. Spontaneous spinal epidural hematomas (SSEH), defined as blood within the epidural space without known traumatic or iatrogenic cause, have an estimated incidence of 0.1 in 100,000 per year [2], [3]. They may be associated with coagulopathies or arteriovenous malformations [1]. SSEH can produce devastating neurologic deficits [4], [5]. Appropriate imaging and intervention must be initiated early when a patient presents with symptoms concerning an SSEH. A paucity of literature regarding this pathologic entity currently exists.

2. **Case Report**

Our patient is a 36-year-old man, followed in cardiology for 15 years for valve replacement by a metal valve on antivitamin K (4 mg/day), with no notion of even minimal spinal trauma. He presented with rapidly progressive heaviness in both lower limbs, becoming incapacitating in three days, with a notion of tingling and urinary retention.

On clinical examination, the patient was conscious (GCS 15/15), paraplegic (0) with mamma sensory level and ROts Abolis.

MRI (Figs. 1, 2) shows epidural hematoma compressing the medulla extending from C5 to D4.

A biological workup showed a hypocoagulated patient with PT < 10%, INR > 8, and normal platelet counts of 160,000. The patient underwent decompressive laminectomy of D2, and D3 with evacuation of the hematoma after correction of the PT and stabilization of her condition (two days after hospitalization).

Post-operatively, after several physical rehabilitation sessions, the patient was down to 1/5th. He is currently continuing his physiotherapy sessions.

3. **Discussion**

Spinal epidural hematoma (SEH) can occur at any age, with extremes ranging from 2 to 79 years. It may be spontaneous in most cases, or secondary to trauma, sometimes minimal, and favored by congenital or acquired coagulation disorders [3], [4]. The origin of bleeding is most often venous, linked to the rich epidural venous network, made up of veins with few valves and vulnerable to sudden changes in venous pressure [1], [4], [5]. The arterial origin may be suggested by the rapid onset of clinical signs, the frequent association with arterial hypertension and the lateral location of the hematoma [6].

For some, the anterior epidural space is almost always spared from hematoma. For others [2], [3], it is more frequently affected than the posterior epidural space. Our cases illustrate the 2 types of localization.
SEH mainly affects the epidural space around the hinges of the spine, particularly the cervicothoracic [4]. It is often localized at the level of two or three vertebrae, the low pressure in the epidural veins, compared with the intrathecal compartment, generally preventing it from progressing, but extension throughout the medullary canal remains possible [3], [5]. The high lumbar site has a better clinical tolerance than a cervical or thoracic site, due to the rarity of spinal cord involvement and the paucity of local signs, which often mimic those of a lumbar disc herniation.

Clinical symptoms often begin with sudden, violent spinal pain, reflecting intraductal hypertension secondary to the sudden eruption of blood in the epidural space. It is frequently associated with headaches and is rapidly followed by a sensory-motor deficit of variable severity, which often progresses to paraplegia or tetraplegia.

MRI is currently the tool of choice for the definitive diagnosis of SEH [7]. The hematoma signal is variable over time and evolves as a function of two main factors: the oxygenation state of the hemoglobin, which conditions the relaxation and magnetic susceptibility properties, and the state of the red blood cell membranes (intact or lysed). Membranes (intact or lysed). The T1-weighted sequence is the most and the change in signal over time is quite characteristic, even pathognomonic, of hematoma [2], [3], [5] If MRI is performed at a very early stage, a few hours up to D2 of clinical symptomatology i.e., at the deoxyhemoglobin stage, the blood signal is isointense in T1 and hyperintense in T2, i.e., aspecific.
A T1 hypointense signal may be seen, posing the problem of differential diagnosis with lymphoma or epidural metastases, especially in a neoplastic context [8]. Following the example of the encephalon, sagittal gradient echo sequences, given their magnetic susceptibility, can resolve the problem at an early stage when the hematoma is iso signal on T1 SE-weighted sequences. T1 sequences with fat signal suppression can differentiate subacute hypertense hematoma from epidural fat. Gadolinium injection often confirms the diagnosis, showing peripheral enhancement, enabling a better definition of the lesion’s lesion boundaries. Enhancement is sometimes central, due to extravasation of contrast through the veins of the posterolateral plexus. Although rare, this type of enhancement must be interpreted with caution, as it may be related to may be related to bleeding that is still active, thus justifying emergency surgery and should not lead the diagnosis astray towards a neoplastic or infectious origin or infectious origin [2], [9], [10]. In terms of differential diagnosis MRI can rapidly rule out a herniated disc or spinal cord injury such as infarction or myelitis. Neoplastic or infectious epidural infiltration infectious epidural infiltration. In fact, an epidural abscess or neoplastic lesion may present an MRI signal identical to that of early SEH but the frequent association with disc abnormalities of infectious or vertebral abnormalities of tumoral origin and the clinical and biological context helps to orient the diagnosis [4], [10].

SEH always required surgical decompression by laminectomy or transforaminal aspiration [11]. Today, with the increasing availability of MRI, which more often than not provides a diagnosis with certainty, a conservative attitude is increasingly adopted. This decision is still based on clinical presentation rather than MRI data [2], [3], [12]. Abstention is recommended in cases of minimal neurological deficit, neurological deterioration followed by early and sustained recovery, or when surgery is contraindicated [12]. In such cases, evolution may be favorable, with complete resolution of the clot within 8 to 10 days. Clinical worsening, with the onset of myelomalacia, is always a cause for concern and dreaded [13]. After surgical treatment, functional prognosis depends on the severity of the initial severity of the initial neurological deficit, early management and the location of the hematoma [1], [12].

4. Conclusion

The majority of the literature describes HSEA as a surgical emergency that can progress rapidly to permanent neurologic deficits. Although certain risk factors for the development of HGSS have been described, there is no clear consensus. Several case studies report no clear predisposing etiology in affected individuals. We report the case of a middle-aged man who presented with massive HESS after daily activities and concomitant amphetamine use. It is possible that the use of stimulants increases the potential of an individual predisposed to developing HESS. The infrequency of this pathology, however, makes it difficult to study the underlying risk factors. Further study is needed, but attending physicians should be aware of the subtle signs of HGSS in the setting of minimal or non-obvious prior trauma, and should initiate appropriate imaging and treatment.

Conflict of interest

Authors declare that they do not have any conflict of interest.

REFERENCES