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Case Report

A Case Report of Spontaneous Spinal Epidural Hematoma inElderly Patient with Surgical Decompression

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Abstract

Spontaneous spinal epidural hematoma (SSEH), uncommon disorder with potentially devastating consequences as high morbidity. Incidence around 0.1 in 100,000 per/year, male- female ratio is approximately 1.4:1, most cases in 4th-5th decades.

The etiology was associated with lumbar puncture and myelography, arteriovenous malformations, bleeding disorders, pregnancy, spinal manipulation. Anticoagulation is also a risk factor for spontaneous spinal epidural hematomas, warfarin is the most common related with SSEH.

SSEH present clinically as a cord compression syndrome, acute onset pain and neurological deficit such as progressive motor or sensory symptoms, sphincter dysfunction, ultimately leads to complete/incomplete motor deficit caused by spinal cord/nerve root compression or cauda equina. The symptoms depending on the location of the hematoma.

The worst prognostic factors are thoracic location, anticoagulation, severe neurologic deficits on admission, sphincter dysfunction, rapid progression.

Magnetic resonance imaging (MRI) is the gold standard technique to diagnose SSEH. Once the diagnosis is established with clinical and imaging results, early surgical intervention should be considered.

Currently, appropriate management is emergence decompression laminectomy and hematoma evacuation.

We present the case of 87-year-old female, on warfarin for atrial fibrillation. Without recent trauma events. Presented sudden thoracic and lumbar pain, paresthesias with irradiation to legs. Progression with sudden paraplegic and anesthesia in lower extremities. Thoracolumbar MRI revealed a posterior SSEH extended from T9-T12. Was released a laminectomy and SSEH aspiration within 6 hours from the symptom's onset.

The patient was discharged one week after the surgery, symptoms improved during the follow-up.

Key words: epidural hematoma; dorsal spine; laminectomy; anticoagulation; lower limb weakness, pain

Introduction

Spontaneous spinal epidural hematoma (SSEH) is uncommon disorder with potentially devastating consequences, described the first time in 1869 by Jackson. [1] The incidence is rare,

0.1 per 100,000 per year, frequency less than 1% of spinal epidural space-occupying lesions, and the male-to-female ratio is approximately 1.4: [1-4].

SSEH can occur at any age and occurs without a known episode of trauma, but the clinical associations that have been reported included spinal arteriovenous malformations, anticoagulant therapy, anti-platelet therapy, bleeding disorders and pregnancy [5-7].

Progressive sensory loss or motor weakness of limbs can be observed below the compressed spinal cord level, depending on the location of the lesion or level of the spine affection the symptoms are different. As is known, the involvement of a higher level is associated with greater complications and deficits or dead. An affectation in the first cervical levels even implies a risk of life given the potential affectation of the vital centers located in the brainstem. SSEH requires early diagnosis and prompt surgical intervention. However, SSEH presents as a neurological emergency as it can lead to irreversible neurological deficit.8 We presenta clinical case of 87-year-old female anticoagulated with warfarin for atrial fibrillation. She presents a sudden excruciating thoracic and lumbar pain with irradiation to legs and associated paresthesias. with worsed evolution with sudden paraplegic and anesthesia in lower extremities. No trauma events before started the symptoms. Next, we describe the treatment carried out and the follow-up [7,8].

Case Presentation:

A woman with 87 years old, with good general health and functional condition for the daily tasks, a medical history notable for atrial fibrillation therapeutically anti coagulated with warfarin. Present a sudden excruciating thoracic and lumbar pain with irradiation to legs and associated paresthesias. No trauma events before started the symptoms. Progressive extremity sensory and motor changes.

Upon the patient arrival to our hospital, her neurologic examination showed diminished sensation throughout her lower extremities. Strength testing was notable for grade 3/5 strength in lower extremity. Plantar reflexes responded in flexion, no clonus. Voluntary rectal tone waspresent but decreased and post void residual bladder volume was 270 cc. Bulbocavernosus reflex was intact.

After a few minutes the patient present neurological deterioration with sudden paraplegic and anesthesia in lower extremities. In the initialevaluation, her INR was found to be 3,2, above her therapeutic goal. Dueto progressive neurologic deficit, she was given Human Prothrombin Complex, Octaplex® to reverse the effect of warfarin.

Emergent MRI reported a dorsal epidural hematoma spanning T9-T12 with spinal cord compression (Figure 1).

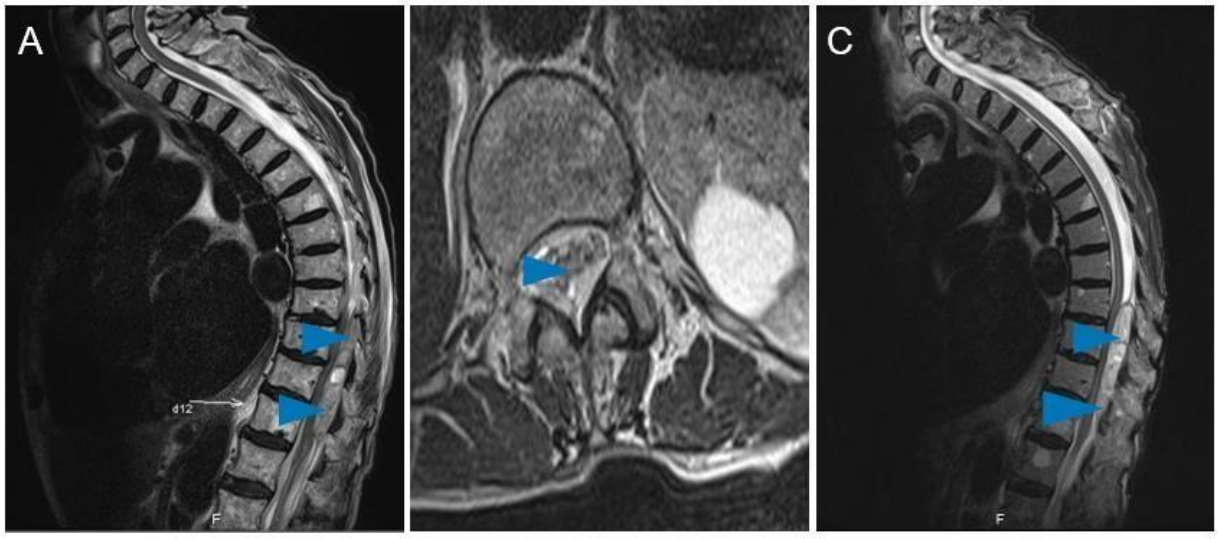

Figure 1 (A,B,C): Mid- sagittal (A,C) and axial (B) MRI T2 sequences demonstrating the dorsal hematoma spanning T9-T12 with multilevel cord compression. Without apparent myelopathy.

A central laminectomy from T9-T12 was observed a epidural hematoma. Following gentle hematoma evacuation the dura was gently palpated with a Penfield 4 elevator and no found tension. The dura was visualized in all extension of the laminectomy. There was no active bleeding. The wound was closed in multiple layers over a submuscular drain.

Following the surgery, the patient's hospital course was good without complications. The patient improved the neurologic examination showed recover of the sensation throughout her lower extremities. In intraoperative period stated steroids administration. Strength testing was notable for grade 3/5 in lower extremity in the postoperative period. Thefirst day after the surgery started worked with physical therapy and physical medicine and rehabilitation specialists, with good progression. An MRI was performed during the first 24h postoperatively (Figure 2).

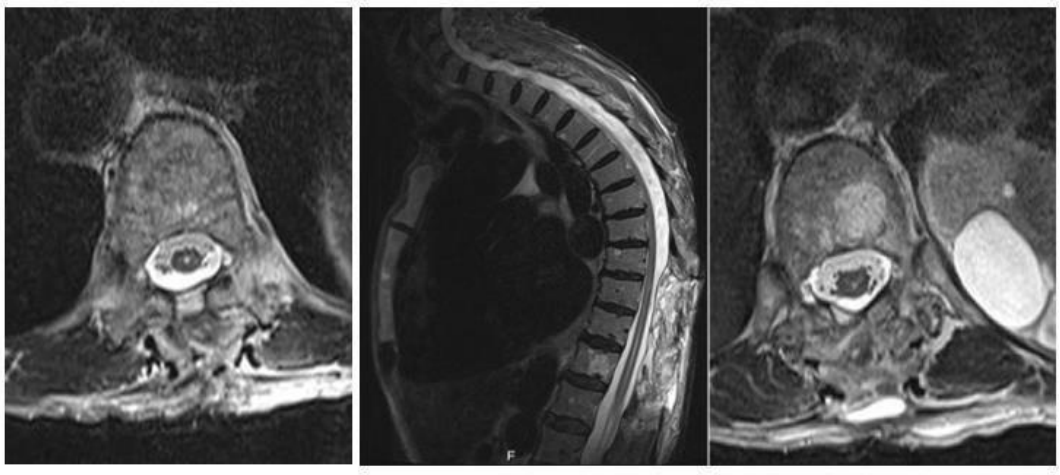

Figure 2 (A, B, C): Thoracic MRI; laminectomy T9-T12 with good decompression of the canal without medullar compression and no myelopathy.

At the time of discharge, approximately one week after her surgery, sensorimotor function recover of the sensation throughout her lower extremities and in the strength testing present grade 4/5 in all myotomes of lower extremity. Able to mobilize with a walker. No bowel or bladder affection. During the follow-up the patients still improve the recover andafter 1 year was able to walk without any support.

Discussion:

SSEH is a uncommon entity, the incidence vary between 0.3-0.9% of all epidural space- occupying lesions [8].

Actually, and depending the different studies the incidence of SSEH is estimated to occur in 1 patient per 100,000 cases/per year. Separated by gender, males present a higher incidence than females with a ratio 1.4:1.9

The first described of SSEH was released by Jackson in a 14-year-old patient in 1896, Bain did the first reported of surgically-treated case in 1897 [10].

The definition and pathophysiology of the SSEH remains controversial. Many causes or risk factors are described and include hypertension, vascular malformation, neoplasia, anticoagulant or anti platelet drug usage, bleeding disorders, and pregnancy [11].

Some authors include hematomas secondary to vascular malformation, hypertension, coagulopathy, and tumors, while others support that only hematomas with idiopathic origin can be labelled as spontaneous [12].

In our case the patient was anticoagulated with warfarin that decrease blood coagulation by inhibiting the enzyme that is responsible for oxidation of vitamin K in the liver. Vitamin K is important for the normal process of coagulation. Oral anticoagulation medications are increasingly being used for stroke prevention in non-valvular atrial fibrillation and deep vein thrombosis, as they overcome some of the difficulties when using the older standard medications that are more complex to administer or require closer monitoring [13].

SSEH frequently presented as sudden onset of pain and weakness. Mainly the pain is radicular and may radiate to the superior or inferior extremities,

depend the level of the lesion. Within hours or days the compression of the spinal cord may lead to varying degrees of motor and sensory loss. Diagnosis of SSEH is difficult to establish prior to the onset of neurologic deficit [14].

Actually, the MRI remains the method of choice in the diagnosis of SSEH. With this technique we can visualize the hematoma with clear location and the size as well as the presence of the spinal cord compression and edema. SSEH yields an isointense signal change on T1-weighted images within the first 24 hours after bleeding and a hyperintense signal change on T2-weighted images after 24 hours [15].

Surgical decompression and hematoma evacuation through laminectomy remains the first choice of treatment. The emergent management to obtain a diagnosed and successfully treated is mandatory and in this case with a urgent decompressive laminectomy. These two points associated with the neurological status of the patients before the treatment would be related with the good or worst evolution and large follow-up. To improve the knowledge and as we know the preoperative neurological deficits and the time interval between symptom onset and surgical decompression are two of the most important factors in determining the prognosis of this pathology. If we compared patients with complete deficits with others that have incomplete deficits the second group present a better recovery. Some authors reported that the recovery rate in cases with incomplete deficit was 89%, and in patients with complete deficit only 37.5% at 1-year follow up [16].

Other groups presented results with Japanese Orthopedic Association (JOA) score of patients who underwent surgery before 12 hours (84%) is better if compared with who underwent surgery between 12-24 hours (63.6%) after onset of symptoms. The results was more worst in cases who underwent surgery more than 24 hours (46.7%) after initial symptoms.[17]

In a clinical study published by Rajz et al. both aspects as time interval and preoperative neurological status represented a main role in the prognosis of patients with SSEH. However, the time interval from symptom onset to surgery appeared have less impact than preoperative neurological status on the time to recover.[18]

For the other side was described some patients with SSEH who were successfully treated through conservative treatment. Generally, in these cases the signs and symptoms were mild and there was no significant masseffect on the image test as CT or MRI. Nevertheless, in cases with conservative treatment was recommended a close observation because of the worst outcomes in this pathology without the correct management.[19]

The use of steroid its unclear and difficult to evaluate without a control group, but as in our clinical case some studies presented that steroid administration accelerated the recovery of neurological deficits in SSEHafter surgery.[20]

According to the National Acute Spinal Cord Injury Study II (NASCIS II) steroids treatment reducing cellular edema and the production of free radicals getting a cells protection. This group recommended a dose of 30 mg/kg methylprednisolone over 15 minutes, with a 45- minute pause andposteriorly a maintenance dose of 5.4 mg/kg/h. [21]

This pathology requires urgent therapeutic guidance, particularly in termsof diagnosis and surgical approach. Subsequently, pharmacological treatment as well as rehabilitation are equally important in the recovery of the patient. Good or worst evolution in the short- and long-term follow-up are related with all these factors, and with neurological status immediately after start the symptoms.

Conclusions:

SSEH is a uncommon entity that requires a prompt diagnosis and treatment. Should be suspected in the setting of acute and significant backor neck pain with an associated neurological deficit.

This case highlights the need for urgent treatment of spinal hematomas particularly in cases with progressive neurological decline.

Actually, the MRI is the gold standard to diagnose a spinal compressive hematoma.

Prompt and appropriate management can improve patient outcomes especially performing a early decompressive laminectomy and hematoma evacuation.

The patients need multidisciplinary support with special relevance to the motor and sensory rehabilitation plan in the months after the procedure.

Conflicts of interest:

No conflicts of interest to declare.

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