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10 Levels thoracic no-intrumented laminectomy for huge spontaneous spinal subdural hematoma removal. Report of the first case and literature review

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ABSTRACT

INTRODUCTION: Spontaneous idiopathic acute spinal subdural hematoma (SSDH) is a rare cause of acute back pain followed by signs and symptoms of nerve root and/or spinal cord compression, frequently associated with coagulopathies, blood dyscrasias and arterio-venous malformations. Standard management includes non-operative treatment and timely (within 24 h) surgical decompression.
PRESENTATION OF CASE: We report on the case of a huge 10 levels SSDH treated with decompressive thoracic no-instrumented laminectomy in a 45-year-old woman with good neurological recovery (from ASIA A to D).
DISCUSSION: Spontaneous SSDHs without detectable structural lesion or anticoagulant therapy are very rare. Among 26 cases documented the literature harbouring SSDHs, the thoracic spine was found to be the preferred site, and the compression was usually extending over several vertebral levels. Nonoperative treatment for SSDH may be justified in presence of minimal neurologic deficits, otherwise, early decompressive laminectomy along with evacuation of hematoma are considered the treatment of choice in presence of major deficits.
CONCLUSION: To our knowledge, the present case is the most extensive laminectomy for a SSDH removal never described before. No postoperative instability occurs in 10 levels thoracic laminectomy in case the articular processes are spared. When major neurological deficits are documented, early decompressive laminectomy with evacuation of hematoma should be considered the best treatment for SSDH.

1. Introduction

Spontaneous idiopathic acute spinal subdural hematoma (SSDH) is a rare cause of back pain, associated with high morbidity. Neurological symptoms are usually severe and timely diagnosis with Magnetic Resonance Imaging (MRI) is mandatory [1,2].

Frequently the onset of symptoms is acute with a severe, often radiating, back pain followed by the stigmata of nerve root and/or spinal cord compression, developing from minutes to days later. The true etiology of SSDHs still remains unknown, but associations with some predisposing conditions, such as coagulopathies, blood dyscrasias and arteriovenous malformations, have been reported [1,3]. Whether surgical evacuation is necessary or not is still a matter of debate.

We report on the case of a 45 year-old female who underwent ten levels laminectomy and durotomy within 24 h from progressively severe paraparesis caused by a spontaneous acute SSDH. A subtotal recovery was documented at 36 months follow up.

Huge thoracic decompressive laminectomy is an uncommon procedure to dealing with multilevel thoracic spine pathology; no more extended decompressive procedures have been described so far, according to the literature review.

2. Case report

A 45 year old woman (HIV+ and HCV+) with history of drug abuse, was admitted to our Institution (Catholic University of Medicine of Rome) with an acute and rapidly progressive onset of sensory/motor deficits involving the trunk and the lower limbs. Laboratory exams did not show any coagulopathy neither blood dyscrasias. No anticoagulant therapy was ongoing. During the previous 20 h the patient complained bowel and bladder dysfunctions

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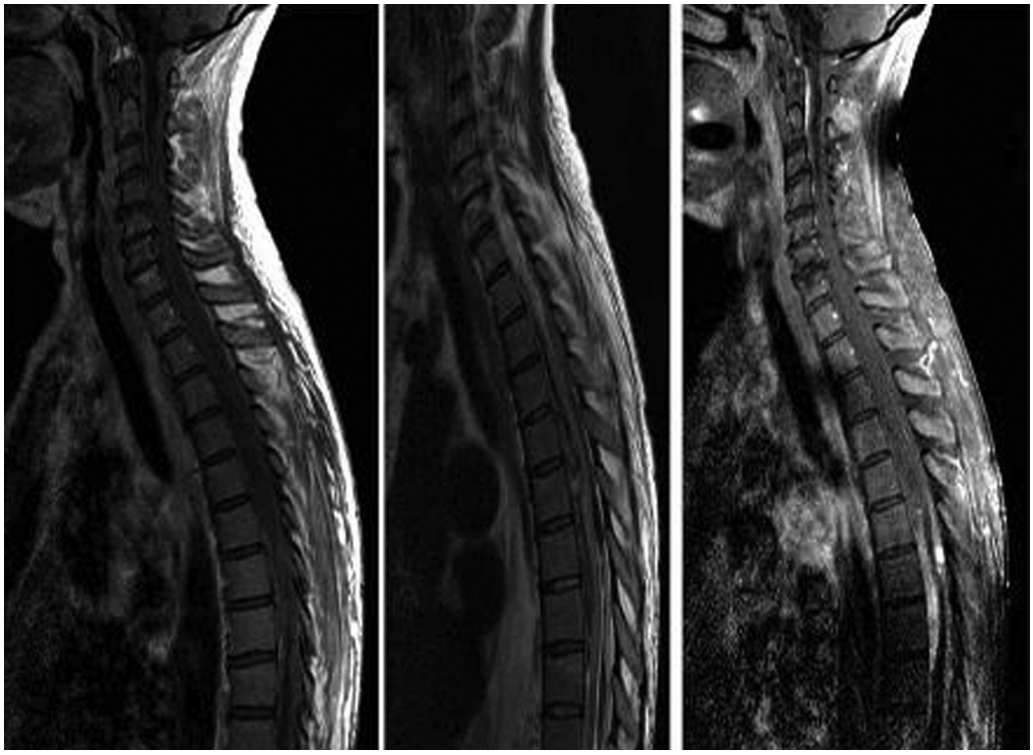

Fig. 1. Sagittal T1 (left) T2 (middle) and Tr long images (right) MR reconstructions. Areas of hypo-intensity in T1, hyper-intensity in T2 along with an hyperintense spot at Th6 in Tr Long images are consistent with a Th 1–Th 10 SSDH with onset before 24 h.

along with intense back pain, poorly responsive to common analgesic therapy. The neurological examination in emergency showed total anaesthesia from Th2 level, paraplegia, deep and superficial areflexia at the lower limbs and the trunk. According to the American Spinal Injury Association (ASIA Scale) the clinical status was scored A.

The pre-operative spinal MRI documented an extramedullary lesional pattern anterior and posterior to the cord, spanning from T1 to T10 (Fig. 1) with areas of hypointensity in T2-weighted images and an hyperintense spot at Th6 Tr long images consistent with SSDH. The spinal cord was compressed, mainly from Th5 to Th8 with T2 hyperintense swelling signal without intramedullary contrast enhancement.

The patient underwent surgical decompression within 24 h from the onset of the symptoms by means of Th1–Th10 conservative laminectomy; the intersomatic joints and the two posterior interapophyseal articulations were spared. The d9ura was opened for the entire length of the exposition and the SSDH was completely evacuated without apparent medullary damage. Post-operative MRI and 3 D CT scan confirmed the extension and the effectiveness of the surgical procedure, along with the spinal cord lesional pattern (Fig. 2). At 36 months follow up, dynamic spine X-ray confirmed the stability of the thoracic spine; the ASIA Score improved to D (Fig. 3).

3. Discussion

SSDH is a rare condition determining spinal cord compression (less than 1%). Spontaneous SSDHs without detectable structural lesions or anticoagulant therapy are further rare. Among 26 cases harbouring SSDHs, documented in the literature, the thoracic spine was found to be the preferred site and the compression was usually spanning over several vertebral elements[1–4] (Table 1).

3.1. Pathophysiology

SSDHs often result from major or minor spine trauma or from spine puncture, including spinal anesthesia. “Spontaneous” acute SSDHs are even more rare and have mostly been observed in conjunction with coagulopathies or anticoagulant therapy, intraspinal tumor and vascular anomalies such as aneurysms or spinal dural arteriovenous fistulas [2,3,15].

The physiopathology of spontaneous idiopathic SSDHs is little understood [1,12,14]. Rupture of valveless radiculo-medullary veins in the subarachnoid space after increased intra-abdominal or intra-thoracic pressure or from minor trauma are some possible mechanisms [2]. This hypothesis could explain clinical signs of subarachnoid hemorrhage (SAH) in many patients with SSDH, the reported combination of SSDH and SAH and the potential dilution of such hematomas by the cerebrospinal fluid (CSF) [2,3,21]. Conversely, SSDH has been thought to arise from the few thin, delicate extra-arachnoidal vessels located on the inner dural surface and then breaking through the arachnoid into the subarachnoid space: it is usually impossible to determine the origin [3,12,25]. In either case, the diluting effect of the CSF prevents clot formation, unless the hematoma is sufficiently large to block CSF flow [15,26].

In idiopathic SSDHs, therapy is limited to the hematoma management, as there is no underlying pathology to face with surgically [2,3,21]. Platelet dysfunction has been shown to be associated with SSDH as shown in Table 1 [10]. Discontinuation of anti-aggregating therapy, however, must be weighed against potential thrombotic complications, and depends on the individual indication of such a treatment.

3.2. Clinical presentation

The clinical presentation of SSDH is characteristic of a sudden onset of severe back or neck pain around the involved vertebrae with radiating pain around the corresponding dermatomes. The