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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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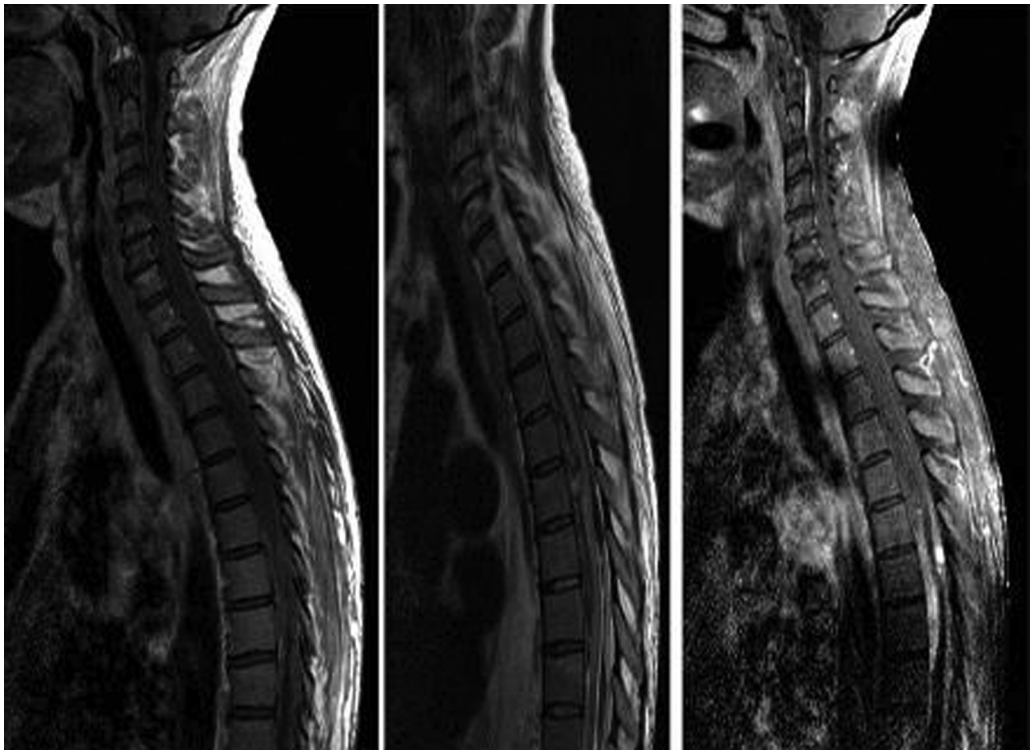

Fig. 1. Sagittal T1 (left) T2 (middle) and Tr long images (right) MR reconstructions. Areas of hypo-intensity in T1, hyper-intensity in T2 along with an hyperintense spot at Th6 in Tr Long images are consistent with a Th 1–Th 10 SSDH with onset before 24 h.

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

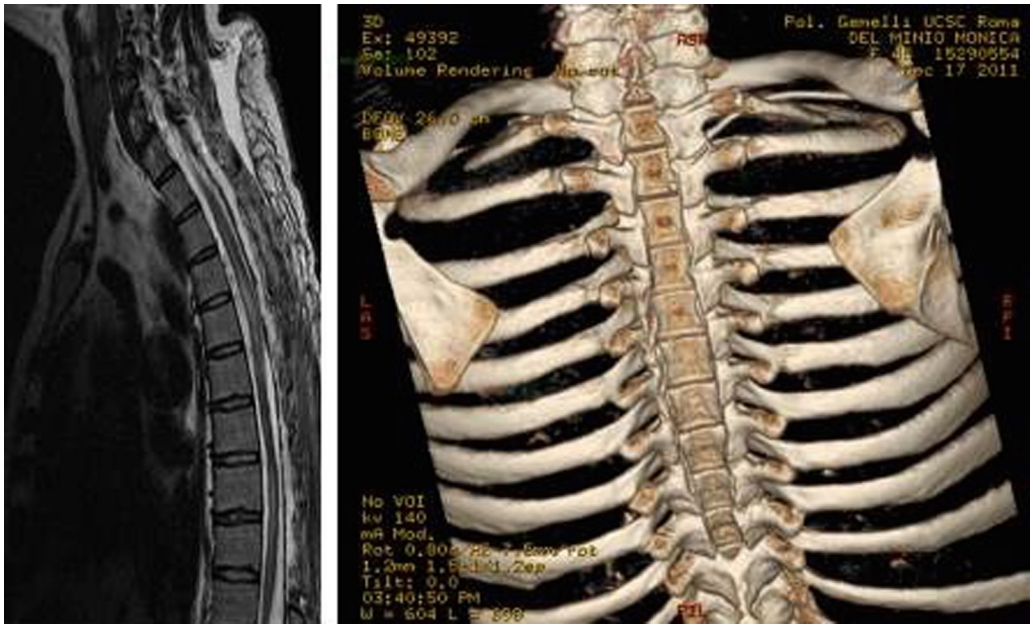

Fig. 2. (Left) Sagittal T2 MR reconstruction (late follow up) showing T1–T10 cord decompression along with diffuse lesional pattern signal from Th3 to Th 10. (Right) 3D CT scan reconstruction showing the extension of laminectomy (C7) Th1–Th 10.

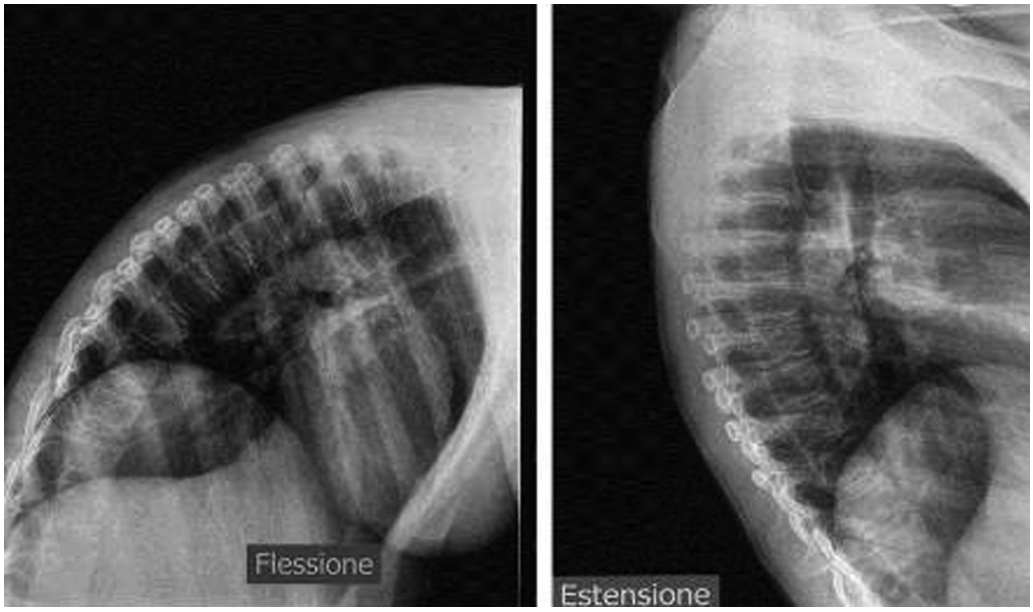

Fig. 3. Dynamic (Right Flexion–Flessione Left Extension–Estensione) Thoracic spine X-ray exam excluding postoperative instability.

initial symptoms are usually vague and the hematoma is difficult to identify until the patient displays symptoms of cord compression hours or days after the onset of pain. Ascending numbness, radicular paresthesia, bowel and bladder dysfunction and progressive paraparesis can be prodromic to permanent neurologic deficits or even death [2,3,15].

3.3. Diagnosis

Although in the past computed tomographic myelography (CT) was largely used for SSDH diagnosis, nowadays MRI is the gold standard imaging modality for recording and recognizing the temporal changes of the hemorrhage, thus facilitating both monitoring of treatment. Indeed the MRI findings of SSDH vary based on the clot, age and oxygenation: within the first 24 h after symptom onset, the hematoma shows isointensity on T1WI and hyperintensity on T2WI. After 24 h, it appears as a high signal on T1WI and as a low signal on T2WI. After injection of gadolinium, peripheral enhancement of the lesion is found frequently; otherwise the central enhancement is found only occasionally. The early MRI findings of our case confirmed the hypothesis of the onset of the SSDH within 24 h. Although T2 hyperintense signals suggesting intramedullary edema are frequently related with a poor prognosis, our patient presented a good postoperative recovery. The effects of the primary mechanical injury and the development of a secondary injury are frequently coexisting, but the vascular impairment seems to be the epiphenomenon leading to the final outcome. Possible reversal of the differential pressure resulting in slow vascular drainage of the cord could explain the secondary vascular anomalies as well as the damage of the neural tissue as observed in our case. The

Table 1
Review, 26 cases of SSDH reported in literature.

Author	Year	Age, sex	Hematoma location	Bleeding cause, risk factors	Preop neuro deficit	Angio	Treatment (spinal)	Recovery
Swann [22]	1984	46, F	TL junction	Unknown	Transient mild paraparesis	Yes	Lumbar puncture	Complete recovery
Kalina [10]	1995	60, F	T7–S2 anterior	Unknown, polycythemia vera	Mild paraparesis	No	Conservative	Complete recovery
Kang [11]	2000	49, F	T5–L3, anterior	Unknown	Transient mild paraparesis	No	Conservative	Complete recovery
Küker [14]	2000	81, M	Mid T spine	Unknown	Paraparesis (M3/5)	No	Surgery	Complete recovery
Küker [14]	2000	56, F	Thoraco-lumbar	Unknown	Paraparesis (M1–3/5)	Yes	Surgery	Good recovery
Kirsch [2]	2000	47, M	T4–L5, antero-lateral	Unknown	Paraparesis	Yes	Laminectony T11-L1	Improved
Kirsch [2]	2000	42, M	CCJ–L3, around SC	Unknown	Paraplegia	No	Laminectomy T2-5	No recovery
Kirsch [2]	2000	34, M	T1–4, around SC	Unknown	Only pain and paresthesia	Yes	Conservative	Complete recovery
Yamada [23]	2003	38, F	T1–7, anterior	Unknown	Mild paraparesis	Yes	Conservative	Complete recovery
Konitsiotis [13]	2003	60, F	T3–L5, anterior-lateral	Unknown, essential thrombo-cythaemia	Only pain	No	Conservative	Pain subsided
Cha [6]	2005	72, F	T3–T6, posterior-lateral	Unknown, aspirin + low molecular heparin	Paraplegia	Yes	Laminectomy T3-5	No relevant recovery
Kyriakides [15]	2007	44, M	T2–T6, anterior	Unknown	Paraplegia	No	Laminectomy T2-6	Subtotal recovery
Kim SD [12]	2008	48, F	T1–4, mainly anterior	Unknown	FMDParaplegia	No	Laminectomy T1-4	No recovery
Ozdemir [21]	2008	50, M	T4–T8, anterior	Unknown	Paraparesis (M3–4)	No	Laminectomy T4-6	Complete recovery
Kakitsubata [3]	2009	66, M	T11/12, anterior-lateral	Unknown	Only pain	No	Conservative	Pain subsided
Oh [20]	2009	59, F	C3–C6, posterior-lateral	Unknown	Left-sided hemiparesis	No	Conservative	Complete recovery
Badge [5]	2009	78, F	T3–T12 posterior	Anticoagulant therapy	Neuro-deficit in lower limb	No	Laminectomy L5	Good Recovery
Panciani [18]	2009	79, F	C5–T6	Unknow	Paraplegia and urinary retention	No	Conservative	Improvement
Payer [19]	2010	59, M	T2–T9 anterior	Anticoagulant therapy	Acute paraparesis, sphincter dysfunction	No	Conservative	Complete recovery
Dampeer [8]	2010	68, M	T6–T7 anterior	Anticoagulant therapy	Paraplegia with paresthesia, urinary retention	No	Laminectomy T6-7	Improved
Alpoim [4]	2011	57, F	T4–T9	Anticoagulant therapy	Dorsal pain, paresthesias and paraparesis	No	Laminectomy T4-5	Complete recovery
Na-rae Yang [24]	2011	55, F	C2–T6	Hypertension, diabetes	Back pain and progressive paraplegia	No	Conservative	Complete recovery
Na-rae Yang [24]	2011	38, M	C6–T5 antero-lateral	Unknow	Chest and back pain, acute urinary retention	No	Conservative	Complete recovery
Haji Mohd Yasin [9]	2012	Unknow	Unknow	Warfarin and fluoxetine	Acute neurological abnormalities of the limbs	Unknown	Unknow	Unknow
Panciani [17]	2013	79, F	C5–T6	Unknow	Paraparesis, anesthesia from mammillary line, sphincter dysfunction	No	Delayed surgery: T5 hemilaminec-tomy	Significant improvement
Chung [7]	2014	66, F	C7–T4	Unknow	Headache of sudden onset and neck stiffness	Yes	Conservative	Improvement

major involvement of the central grey matter, compared to the less compromised peripheral white matter, as seen on the axial MRI imaging, could be explained by the greater sensitivity of the more metabolically active grey matter to venous congestion due to the AVDF.

Spinal angiography is generally considered to be the gold standard for demonstrating spinal artery aneurysms, arteriovenous malformations, spinal dural arteriovenous fistulas or other pathologies that can cause spinal subarachnoid hemorrhage. Nevertheless a clear infarction or hemorrhage are extremely rare especially in cases of spinal DAVF [25]. In our case spinal angiography had not been performed for two reasons: the unavailability of angiographic room in emergency and a rapid progression of symptoms; therefore we preferred to remove the cause of spinal cord compression promptly, although the cause of bleeding was not assessed previously.

By reviewing the literature, angiography has been used to rule out vascular malformations and was performed in 6 of the 26 cases

described in the literature; it still remains a case to case decision based on availability, degree of neurosurgical emergency and suspicion level of vascular malformation (see Table 1).

3.4. Treatment and follow up

Among the similar cases reported in the literature (12/26 patients: 46.15%) nonoperative treatment may be justified in presence of minimal neurologic deficits. In presence of major deficits, or a rapidly deteriorating clinical and radiological (CT, MRI) patterns, patients are usually likely to benefit from drainage or surgery [18]. [10,18,20,23]. In such cases, early decompressive laminectomy with evacuation of hematoma are considered the best treatment for SSDH, as performed in our case (Table 1). On the other hand a case of a 79-year-old female undergone a surgical evacuation of a spontaneous SCSH one year after the diagnosis has been described [17]. She presented with a severe paraparesis and showed a considerable improvement in sensory-motor performances after surgery. Consequently the treatment of spontaneous SCSH is not well defined and universally accepted. Early surgery is mandatory in cases presenting with severe deficits and an aggressive approach should be considered as a viable option in cases of spontaneous SSDH even after a long lasting spinal cord compression [17].

Although the outcome predominantly depends on the clinical status and the levels of the lesion, our case showed a satisfactory late follow up, despite he presented severe neurological deficits and he underwent 10 levels laminectomy; such finding seems related to the short interval (within the 24 h) between the onset of the symptoms and the surgical decompression [27–29]. Moreover it is also known that patients with paraplegia and bowel and bladder dysfunction present the poorest prognosis regardless of surgical or conservative treatment [18,27–29].

3.5. Levels laminectomy

Our case is the first reported in the literature harbouring 10 levels thoracic SSDH treated with conservative 10 levels laminectomy; consequently posterior fixation was not performed because unnecessary. In adult patients conservative laminectomy at thoracic level does not necessarily affect spinal stability, since thoracic chest "per se" supports local stability. Conversely, post-laminectomy kyphosis is expected in most of pediatric patients and up to 100% when it is performed at cervical level [30].

We did not decide to perform laminectomy at alternate levels because in this way a complete access to the hematoma and its total removal, could not been completely allowed. Furthermore we did not perform neither laminoplasty nor posterior fixation since the intrinsic stability of the thoracic chest does not require anatomic reconstruction of the posterior elements at this level and the operative time would be excessively prolonged for an urgent procedure. Surprisingly thoracic kyphosis has been reported as complication after laminoplasty [31].

4. Conclusion

- Fast clinical and neuroradiological identification along with urgent (within 24 h from the onset of the symptoms) surgical management are mandatory in SSDHs in order to achieve satisfactory clinical results.
- Extensive operative strategy can be necessary.
- Ten levels thoracic laminectomy is safe and effective, as long as conservative. No postoperative instability occurs in 10 levels thoracic laminectomy since articular processes are spared.

Conflict of interest statement

None.

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None.

Consent

We have obtained written consent from the patient before starting the study.

Author contributions

All authors of this paper have directly participated in the planning, execution, or analysis of this study. All authors of this paper have read and approved the final version submitted.

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