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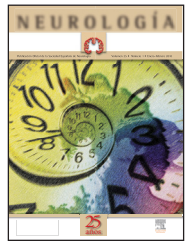

ORIGINAL ARTICLE

Spinal haematomas: the spinal aplopexy

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KEYWORDS
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Abstract
Introduction: Spinal haematomas (SH) are a rare pathology. They can produce a rapid and irreversible neurological deterioration.
Patients and method: In this retrospective study, we review 8 of SH treated in our centre over the last five years. Data collected were: age, sex, predisposing factors, clinical status, radiological features, treatment and outcome.
Results: Five patients were female and three male. Age ranged between 13 and 81 years. Five patients were hypertensive. Four had a coagulation disorder. In three patients SH occurred after physical effort. One case appeared after a lumbar puncture and another was secondary to intramedullary cavernous angioma. All patients presented with pain followed by neurological symptoms. Four cases were epidural, two subdural and two were intramedullary. All epidural SH and one subdural SH were located dorsal to medulla. Five patients were operated on due to progressive neurological deterioration. Only one of them showed neurological improvement. Patients who were not operated on had a better neurological status and they improved spontaneously. Two of them were discharged without neurological symptoms.
Conclusions: There is controversy over physiopathology of SH. The triggering mechanisms are unknown. The vessel (artery or vein) and the anatomical compartment in which SH arise are also unknown. Prompt diagnosis and urgent surgical treatment are needed when the patient has neurological symptoms. In these cases, the most important prognostic factor is the preoperative neurological status.

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PALABRAS CLAVE
Apoplejía espinal;
Hematoma epidural espinal;
Hematoma subaracnoideo espinal;
Hematoma subdural espinal

Hematomas espinales: la apoplejía espinal

Resumen
Introducción: Los hematomas espinales (HE) son una afección muy poco frecuente, que puede causar un deterioro neurológico rápido e irreversible.
Pacientes y método: Presentamos un estudio retrospectivo con 8 casos de HE tratados en nuestro servicio en los últimos 5 años. Se analizaron: edad, sexo, factores predisponentes, situación clínica, características radiológicas, tratamiento y evolución.
Resultados: Los pacientes eran 5 mujeres y 3 varones. Las edades oscilaron entre 13 y 81 años. Cinco pacientes eran hipertensos; 4 presentaban alteraciones de la coagulación; en 3, el hematoma apareció en relación con un esfuerzo físico; en 1, tras una punción lumbar y en 1 fue secundario a un cavernoma intramedular. El inicio clínico fue en todos los casos con dolor, seguido de la instauración de síntomas neurológicos. Cuatro eran epidurales, dos subdurales y dos intramedulares. Todos los HE epidurales y uno subdural eran posteriores a la médula espinal. Cinco pacientes fueron intervenidos por deterioro neurológico progresivo. Sólo 1 presentó mejoría clínica. Los casos no intervenidos estaban en mejor situación neurológica y mejoraron espontáneamente, y 2 de ellos fueron dados de alta sin secuelas.
Conclusiones: Los HE son procesos de fisiopatología controvertida. Se desconocen los mecanismos que los desencadenan, así como los vasos (arteriales o venosos) y el compartimento anatómico en que se originan. Se precisa un diagnóstico precoz y un tratamiento quirúrgico urgente cuando hay síntomas neurológicos progresivos. En estos casos, el factor pronóstico más importante es el estado neurológico preoperatorio.

Introduction

Spinal haematomas (SH) are rare clinical entities which can cause rapid neurological deterioration that is often irreversible if not diagnosed and treated promptly. Jackson is credited with the first case of clinically diagnosed SH, published in 1869 under the title "*Case of Spinal Apoplexy*"[1]. The first successful surgically evacuated case was published in 1911[2]. In recent years, we have witnessed an increase in cases diagnosed. This is due to advances in diagnostic techniques and the increase in life expectancy, as well as the number of patients following anticoagulation therapy[2]. Nevertheless, the low incidence is surprising when compared with that of intracranial haemorrhage.

The aim of this study was to draw attention to this condition, analyse the different pathophysiological theories in existence and to publish our experience with these types of processes.

Patients and method

We conducted a retrospective study of 8 cases of spinal haematomas diagnosed and treated at our neurosurgery department in the last 5 years. We excluded postoperative spinal haematomas and those secondary to direct trauma to the spine from this study.

Through the review of hospital records, we analysed the following variables: age, gender, personal history, initial symptoms, neurological clinical development, haematoma location and extent, treatment followed and results obtained. Three of these cases were published as clinical cases[3-5].

Results (table 1)

The patients were 5 women and 3 men. The ages ranged between 13 and 81 (average 51.6) years, 5 were hypertensive and 4 patients presented coagulation disorders: 2 patients (cases 5 and 6) received acenocoumarol (Sintrom®) for atrial fibrillation; 1 (Case 4) was being treated with low molecular weight heparins and triple platelet antiaggregation (aspirin, clopidogrel and glycoprotein IIb/IIIa inhibitors) due to a recent acute myocardial infarction; and 1 (Case 8) was a haemophiliac child. Case 5 appeared after a lumbar puncture: a 74-year-old woman with atrial fibrillation (AF) and chronic renal failure who was admitted for a study of chronic adult hydrocephalus; during her admission, acenocoumarol was replaced by enoxaparin (Clexane® 60 mg/24 h) and an infusion test was performed by lumbar puncture. The test was negative and she was discharged from hospital; 8 days later, she was readmitted for paraplegia with sensory level. In 3 patients there was a history of some form of physical exertion. In the 2 youngest (13 and 16 years), the haematoma appeared after playing football, but they did not report any direct trauma (cases 3 and 8). Case 2 appeared after a Valsalva manoeuvre was performed due to a coughing fit. Case 7 was secondary to an intramedullary cavernoma. The clinical onset for all cases started with

Table 1 General characteristics

	Age and gender	Aetiological factors: Predisposing factor	Aetiological factors: Trigger factor	Initial symptoms	Neurological symptoms and exploration on admission	Location	T1-T2 (MRI)	Treatment (time of symptom onset-surgery)	Prognosis
Case 1	81, F	HT, cervicarthrosis	?	Dorsal pain	Paraplegia Sensitive level	C7-D8 Posterior Epidural	Iso-hetero	Laminectomy (10 h)	No improvement: paraplegic on discharge
Case 2	63, F	HT	Stress	Dorsal pain	Paresthesias both hands	C7-D7 Posterior Epidural	Iso-hetero	Medical	Spontaneous improvement: paresthesias in left hand
Case 3	16, M	?	Stress	Cervical pain	Monoparesis 3/5 in left arm	C3-C5 Posterior Epidural	Hyper-hypo	Medical	Spontaneous improvement: asymptomatic on discharge
Case 4	60, F	HT anticoagulation	?	Dorsal pain	Paraparesis 1/5 in less than 12 h	D9-L1 Posterior Subdural	Iso-hetero	Laminectomy (12 h)	No improvement: paraplegic on discharge
Case 5	74, F	Anticoagulation HT, CRF	Lumbar p.	Dorsal pain	Paraplegia Sensitive level Sphincters	D12-L3 Anterolateral Subdural	Iso-hetero	Laminectomy (8 days)	No improvement: death
Case 6	70, M	Anticoagulation HT	?	Dorsal pain	Paraparesis 1/5 Sensitive level Sphincters	D2-D4 Intramedullary	Iso-hyper	Laminectomy (4 days)	No improvement: paraplegia on discharge
Case 7	36, F	Cavernoma	?	Dorsal pain	Paraparesis 4/5	D11-D12 Intramedullary	Iso-hetero	Medical	Spontaneous improvement: asymptomatic on discharge
Case 8	13, V	Hemofilia A esfuerzo		Dorsal pain	Paraparesia 4/5	D5-D6 Posterior Epidural	Iso-hyper	Laminectomy (6 h)	Improvement: asymptomatic on discharge

C: cervical; D: dorsal; Hetero: heterosignal; Hypo: hyposignal; HT: hypertension; CRF: chronic renal failure; Iso: isosignal; L: lumbar; F: female; Lumbar p.: Lumbar puncture; MRI: magnetic resonance imaging; T: time; M: male.

pain, followed by the establishment of neurological symptoms. In all cases the imaging diagnostic was performed using magnetic resonance imaging (MRI): 4 were epidural, 2 subdural and 2 intramedullary. All epidural SH were posterior to the thecal sac and the spinal cord: 1 cervical (C3-C5), 2 cervicodorsal (C7-D8 and C7-D7) and 1 dorsal (D5-D6). One subdural SH was posterior to the spine (D9-L1) and another was anterolateral to it (D12-L3). The 2 intramedullary SH affected the spinal cord (D11-D12 and D2-D4). Five patients underwent surgery, 3 of them before 12 hours from symptom onset. Only the youngest patient improved clinically. He presented a 4/5 paraparesis at the time of surgery. In all cases that were not operated on, the pain and neurological symptoms disappeared and imaging controls showed a progressive resolution of the haematoma. Two of them were discharged without neurological sequelae.

Discussion

SH can be classified as epidural, subdural, subarachnoid or intramedullary, according to the affected anatomical compartment. Of these, the most common are those with epidural location, which are 3/4 SH[2,6,7]. The rest, especially the intramedullary, are very infrequent[2].

Age and gender

SH are more common between the fifth and sixth decades of life, with a peak incidence between 55 and 70 years[2,8]. Two of our patients were 13 and 16 years old, which is a very rare situation because spontaneous SH are very rare in people under 20[2].

Most series show a predominance of males over females, a trend that is more pronounced in epidural SH (2:1 ratio) than in subdural SH (with a ratio of almost 1:1)[2]. In our series, there was a slight predominance of females over males (5/3), although this fact was not significant due to the low number of cases.

Aetiological factors

SH have been associated with numerous factors, including intracranial surgery[9], trauma and lumbar punctures[3,10], coagulation disorders[2,4,5,8,11,12], Valsalva manoeuvres[2], sudden movements[7], hypertension and arteriosclerosis[2], advanced age[2], arthrosis[7], etc. However, these factors are very common in the general population, while the incidence of SH is very low. It is currently believed that there are predisposing factors (hypertension, advanced age with fragile vessels, arteriosclerosis, coagulation disorders, etc.) on which trigger factors act (physical effort, sudden movements beyond the limits of tolerance of vessels, Valsalva manoeuvres, lumbar punctures, etc.), and bleeding is generated only when both coincide in the same patient[2]. Coagulation disorders alone would thus be unable to cause SH, unless they appeared in combination with other circumstances, such as lumbar punctures, physical exertions, sudden movements, Valsalva manoeuvres, etc.[8]. In fact, prothrombin time is not related to SH severity or frequency[2].

Idiopathic SH are the most common and account for 40% in some series[2]. Among secondary SH, the most common are those related to coagulation disorders and lumbar punctures[2,8,11-13]. In our series, 4 patients (50%) presented abnormal coagulation. In 2 of these, no trigger factor for the haematoma was identified. In the other 2, 1 appeared after a lumbar puncture and 1 was related to physical exertion.

Direct trauma to the spine rarely causes SH with spinal cord compression[2,14]; such trauma is more common in children aged 1-2 years due to neck fragility in relation to head weight. Tumours and vascular malformations often cause subarachnoid or intramedullary SH[2]. The spinal tumours that most often originate SH include ependymomas, neurinomas and gliomas[2].

Physiopathology

Classification of spinal haematomas according to the anatomical compartment affected

The pia mater is the innermost, finest meningeal layer, which adheres closely to the spinal cord and spinal roots. The arachnoid is composed of two layers: a compact, laminar, outer one in contact with the dura mater, and an inner one formed by conjunctive trabeculae. The dura mater is the outermost membrane, thick and rugged, and consists of a set of concentric lamellae rich in collagen fibres[9]. These membranes create three anatomical areas: epidural, subdural and subarachnoid, in which blood can accumulate and give rise to haematomas with their same name.

Both the epidural and subarachnoid spaces are real spaces that exist in physiological conditions. The subarachnoid space lies between the arachnoid and the pia mater and contains cerebrospinal fluid (CSF), which circulates freely. The subdural space, however, is a virtual space that does not exist in normal conditions and is occupied by a tissue of elongated, neurothelial cells with numerous branches and that define small lacunar spaces, with amorphous material[11,15,16]. The subdural space appears only when the neurothelial cells are broken and fracture planes are formed which join the lacunar spaces. The epidural space is delimited by the dura mater and the periosteum of the vertebral spinal canal. It contains epidural fat that mechanically protects the spinal cord. Covered by this fat, there is a venous plexus that runs along the entire length of the epidural space. The veins of this plexus have very thin walls and lack venous valves.

The spinal vessels of larger calibre are in the epidural space, the width of the dura mater and the subarachnoid space[11]. Throughout the width of the arachnoid and the subdural space (dura mater-arachnoid interface), there are only capillaries and venules[11,16].

Epidural haematomas

Neither their pathophysiology nor the source of bleeding is known. There are two theories: arterial and venous. According to the latter[17,18], epidural SH are caused by a sudden rise in intrathoracic or intra-abdominal pressure (coughing, sneezing, Valsalva) that is transmitted directly to the epidural venous plexus veins and break them. The

venous theory is based on the fact that this plexus is more developed dorsally, and is smaller in the ventral side of the dural sac, where it is covered in part by the posterior longitudinal ligament[14,17,18]. This would explain why most of the epidural SH are dorsal to the thecal sac. This theory has been questioned because the pressure in the epidural veins, especially in the cervical region, is very low and less than intrathecal pressure; a venous bleeding would therefore not be able to progress and compress the spinal cord[6]. The arterial theory[19,20] argues that the source of bleeding is the radicular arteries that accompany the nerve roots within the epidural space. A sudden and forceful movement, especially in people with spondylarthrosis, would cause these vessels to stretch and break[7]. This would explain why epidural SH are often initiated with radicular-type pain, their alleged association with hypertension and the fact that the clinical course is acute and brusque[2]. On the other hand, in none of our cases (nor in most of those published[2]) has a broken artery been observed during surgery, although the evacuation was very early. In contrast, we could observe profuse epidural venous bleeding in all epidural haematomas we operated on. This result coincides with those of the majority of published cases[21]. For this reason, the venous origin of haematomas is more widely accepted[2].

Subdural haematomas

The pathophysiology of subdural SH is scarcely known[22-24]. It is not known which vessels originate the haematoma[23] or whether it starts in the subdural or subarachnoid space, as it is very common to observe the blood spread and occupy both spaces[8,21,23]. Some authors therefore prefer to speak of intradural extramedullary SH[23]. The vessels of the subdural space are very rare and of small calibre[11,15,16], so it is believed that the bleeding could start in the subarachnoid space[21,25,26]. The rupture of these vessels would cause a secondary arachnoid rupture, and the blood would then reach the subdural space[21,23,26]. This would explain the fact that some cases start with symptoms similar to those of subarachnoid haemorrhage (headache and stiff neck)[2,26].

In one of the subdural haematomas in our series (Case 5), we observed during surgery how the blood collected in the subdural space extended to the spinal subarachnoid space. The patient was an anticoagulated woman who had undergone a diagnostic lumbar puncture. The origin of the haematoma may have been the direct puncture of the vessels of the inner layers of the dura mater or subarachnoid space[11]. It could also be explained by CSF hypotension, which would make the arachnoid slide on the dura mater, which in turn would tear neurothelial cells and small vessels[11]. This effect would be enhanced by anticoagulation.

Subarachnoid haematomas

These are the haematomas in which the arachnoid remains intact and contains the haematoma separated from the subdural space[2,8]. They are considered as characteristic lesions and independent of subdural haematomas[8]. They are believed to arise from a sudden increase in pressure in the lumen of radicular vessels, arterial or venous, in the subarachnoid space. If the bleeding originates in that area, it is more difficult for a localised clot to form by the fibrinolytic effect of the CSF and the dilution of the blood itself. However, if the bleeding is very fast or if some factor impedes CSF flow (arachnoiditis, spondylosis, herniated disks, etc.), the growth of the clot is favoured[8,23]. In our series, there were no cases of pure subarachnoid SH.

Intramedullary haematomas

These are usually secondary to an underlying condition, such as tumours or vascular malformations. Of the 2 cases in our series, 1 was secondary to an intramedullary cavernoma and 1 appeared in a patient with hypertension and anticoagulation.

Clinical manifestations

Most SH usually have a sudden, sharp (ictal) course. They generally appear with dorsal spinal pain, depending on the affected level, followed by symptoms of spinal cord compression. Numerous neurological deficits have been described: sensory and motor alterations, sphincter dysfunction, Brown-Séquard syndrome[2-8,11,13,14,21,23,27,28], among others. In all our patients, SH started with pain and all cases presented motor deficits, except for one patient who only referred paresthesias in both hands.

Epidural SH usually have a similar but more abrupt course than subdural SH[2]. Subarachnoid SH can take two clinical forms: as typical SH, with pain and symptoms of spinal cord compression; or similar to a brain condition with headache, vomiting, meningism and alterations in the level of awareness.

Approximately 75% of subdural SH cases develop the entire spectrum of clinical manifestations before 72 h. However, subacute and chronic forms have also been described[2,27], especially in the lumbar region, where the epidural space is wider and the cauda equina offers better support for the mass effect than the spine[27].

All cases in our series had an acute evolution, except for Case 5, which began with spinal pain at 6 days after the lumbar puncture; the neurological symptoms appeared 2 days later.

Diagnosis

The existence of a spinal haematoma should be suspected in any patient who presents pain and symptoms of spinal cord compression. In computed tomography (CT), epidural SH appear as hyperdense lesions with a biconvex lens shape[6,7]. The subdural cases often take the form of a half moon[12-14]. Subarachnoid SH are more diffuse lesions without net limits[2]. In the case of the subacute, they can be isodense and be overlooked.

Magnetic resonance imaging (MRI) is the diagnostic method of choice (figs. 1 and 2). The signal from the blood depends on age, and its behaviour is similar to that of cerebral haematomas[26,29]. In the first 24 h, they appear as isointense lesions on T1 and hyperintense on T2, due to the presence of oxyhaemoglobin. On the second and third days, they present a hyposignal in both T1 and T2. Subsequently, the signal increases in both sequences due to the appearance of methaemoglobin[26,29-31].

The majority (75%) of epidural SH are dorsal to the thecal sac[2,6,7], since the anterior side of the spinal dura mater is

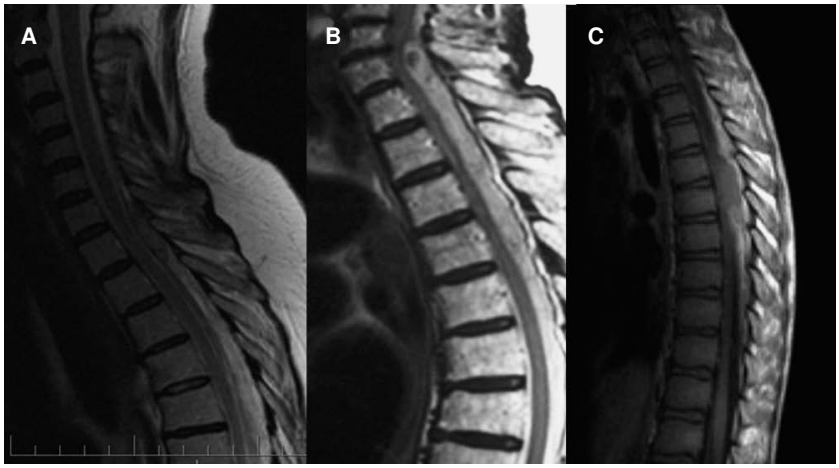

Figure 1 A: Case 1, MRI scan in sagittal section, T2 sequence; extensive epidural lesion with areas of hypersignal and hyposignal that displaces and compresses the spinal cord. B: Case 2, sagittal section in T2 showing a lesion posterior to the thecal sac, with a heterogeneous signal, typical of blood in the acute phase. C: Case 8, sagittal image in T1 showing a D5-D6 dorsal lesion, isointense to the spinal cord, with epidural morphology, compressing the spinal cord at that level.

very adhered to the periosteum of the vertebral canal and the posterior part of the longitudinal ligament[14]. The anterior location is more common in subdural haematomas than in epidural[14]. Subarachnoid also tend to be dorsal to the spine[8]. Only one of the SH in our series was anterolateral to the spinal cord.

The most affected levels are the dorsolumbar and cervicodorsal joints[2,8,22]. However, this distribution varies with age: they are more frequent in the cervical and upper dorsal regions in children and young people, while they are more common in the dorsolumbar region between 46 and 70 years[2]. Our series shows similar results, and the most frequent haematomas were the cervicodorsal and dorsolumbar.

The differential diagnosis between epidural, subdural and subarachnoid SH can be difficult. The epidural cases usually have a biconvex lens shape and more defined boundaries and affect 2-4 vertebral bodies[6,7,13,22,29]. The subdural are usually shaped as half moons, with less defined boundaries, and affect a larger number of spinal segments[13,14]. At times, the MRI does not suffice to distinguish a subarachnoid from a subdural hematoma[8,32], and the definitive diagnosis can only be reached through surgery.

The appearance of abnormal vessels in the conventional sequences should make us suspect there is an underlying vascular lesion. Magnetic resonance angiography (TRICKS sequences) should be performed in these patients, and the diagnosis should be confirmed with a spinal angiography[2].

Treatment

Patients with no symptoms of spinal cord compression can be managed with conservative treatment[2,6,12,29]. In patients with neurological alterations, especially if they are progressive, the treatment of choice is surgical drainage of the haematoma before the alterations become irreversible[2-4,14,23,24], unless the poor general condition of the patient contraindicates the surgery[2-8,11,13,20,23] or there is a progressive clinical improvement, as in our case. The injuries are usually dorsal to the spinal cord, so the most commonly-used surgical approach is the posterior; the haematoma is drained by a laminectomy, with or without dural opening, depending on the location of bleeding. Five of our patients were operated on, all through a posterior approach: 4 presented a severe motor deficit (paraplegia or paraparesis 1/5); the other (Case 8) was operated on due to progressive paraparesis 4/5, the MRI revealing a dorsal lesion that occupied nearly half of the spinal canal in the axial sections.

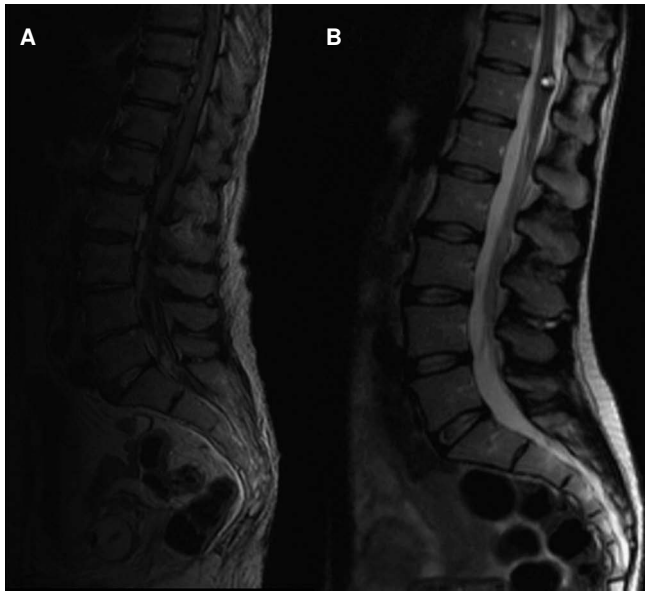

Figure 2 A: Case 5, MRI scan, sagittal section in T2 sequence showing a lesion of heterogeneous signal, located in a ventral position to the thecal sac. B: Case 7, T2 sequence; intra-axial lesion that expands to the medullary cone, with a hyperintense central area surrounded by a peripheral zone of hyposignal.

Patients who are not operated require close clinical monitoring to detect changes in neurological condition. It is essential to carry out image controls to monitor clot evolution[2,6]. In the 3 patients in our series who were not intervened, the MRI showed spontaneous resolution of the haematoma.

Prognosis

This is largely influenced by the location of the blood. Epidural haematomas have a better prognosis than subdural or subarachnoid, since in them the blood is in direct contact with the spinal cord[8,22]. Thus, 50% of epidural SH[2] and only 26.8% of surgically treated subarachnoid SH[8] recover completely. Only half of patients with operated subdural haematomas can carry out independent life after surgery[33], and total mortality reached 30%[34].

The most important prognostic factors are preoperative neurological condition[2-7,23,30,33,35,36] and rapid surgical decompression after onset of neurological symptoms[2-4,6,7]. A full recovery was shown in 65.9% of patients treated within 12 h of symptom onset[2]. Cervical and dorsal haematomas have a worse prognosis than lumbar, since at this level the spinal canal is wider and there is no spinal cord[24,33]. Age, speed of onset of neurological symptoms or the number of segments affected do not appear to have prognostic significance[2,33].

In our series, the most important prognostic factor was the preoperative neurological condition: none of the patients with severe motor deficits (paraplegia or paraparesis 1/5) experienced any improvement, regardless of the speed with which they were intervened. In contrast, all our patients with mild neurological symptoms (intervened or not) improved. The number of bodies affected was not decisive: Case 2 had a very large haematoma occupying eight vertebral bodies and was discharged with slight paresthesia in one hand.

Conclusions

SH are rare clinical entities, despite the frequency of the aetiological factors with which they have been related. Their pathophysiology remains unknown, as do the pathogenic mechanisms that trigger them. They can produce rapid, irreversible neurological deterioration, so early diagnosis and treatment are essential.

Conflict of interests

The authors declare no conflict of interests.

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