Spontaneous Thoracic Spinal Epidural Hematoma Causing Paraplegia

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ABSTRACT

Spontaneous spinal epidural hematoma is a rare emergency. The exact etiology is unknown. A 60-year-old woman arrived at the emergency department with back pain, followed 20 minutes later by acute paralysis of both legs. The initial magnetic resonance image (MRI) showed a spinal epidural hematoma extending from T-1 to T-12, with compression of the thoracic spinal cord. Emergency right hemilaminectomy (ten levels) revealed an epidural hematoma with compression of the spinal cord. The case was discharged from the hospital on the seventh post-operative day with a slightly unsteady but improving gait. This case is unique as it extends to T-1 and T-12. The presenting symptoms, etiology, pathophysiology, differential diagnosis, and treatment of this unusual lesion are reviewed.

KEY WORDS: Thoracic spinal epidural hematoma, magnetic resonance imaging, paraplegia.

ÖZ


ANAHTAR SÖZÜKLER: Magnetik rezonans görüntüleme, torakal spinal epidural hematom, parapleji.
INTRODUCTION

We describe a case of spontaneous thoracic epidural hematoma with surgical decompression. A wide range of underlying diseases, such as coagulation disorders (2, 26) and vascular malformations may be the cause (13). Association with acetyl salicylic acid intake or chronic renal disease have has been reported (3, 4, 21, 26). No underlying cause can be identified in up to 40% of the cases (2, 9, 15, 17, 24). The fragile spinal veins, especially the valveless epidural venous plexus, are accused of being the site of structural weakness. Congestion that precedes rupture of the veins is a potential pathophysiological mechanism (17). The usual therapy is surgical removal of the hematoma (2, 7). We report a case of thoracic spinal epidural hematoma associated with acute paraplegia, which was treated with decompression and followed by magnetic resonance imaging (MRI) documentation of complete resolution within 30 days.

CASE REPORT

A 60-year-old woman presented to the Emergency room complaining of the acute onset of paraplegia. She had been experienced progressive weakness in both lower limbs, and, on presentation to the Emergency Room 30 minutes later, was found to be paretic and completely paralyzed in both legs with an upper thoracic sensory level at T1. Her only medication was an antiplatelet drug only for 3 days. There was no history of hypertension, trauma, or bleeding dyscrasia. There was no history of constitutional symptoms. MRI showed a large, spontaneous, epidural hematoma in thoracic region. A posteriorly-located hematoma and cord compression were shown on sagittal MRI. The laboratory examinations including prothrombin time, partial thromboplastin time, platelet count, and liver function tests, were unremarkable.

A surgical decompression with right T-3 to T-12 hemitotal laminectomy was performed (approximately 13 hours after the initial event) and the patient improved symptomatically (Frankel B). There was no bleeding during the surgical procedure. This was confirmed by a physical examination that showed paraparesis (not complete paralysis as before), with the same sensory level of T-2. Radiological examinations were obtained promptly, and the results (see below) were consistent with thoracic epidural hematoma. The patient was discharged to her home with a complete neurological recovery and complete radiological improvement of the epidural hematoma.

Radiological investigations

Magnetic resonance imaging (MRI) with and without gadolinium enhancement of the thoracic cord was performed using T-1 and T-2 weighted imaging (TR, 650 MS; TE 30 MS; excitations: 1). The MRI study confirmed the presence of a lesion compressing the cord posteriorly extending from T-3 to T-12. The lesion had an isointense signal on T1-weighted and a hyperintense signal on T2-weighted images, and did not enhance after the injection of gadolinium (Fig 1,2,3).

DISCUSSION

Figure 1: Sagittal noncontrast-enhanced spin echo T1-weighted image of the thoracic cord, showing an isointense signal of the lesion compressing the cord posteriorly extending from T1-T12. It did not enhance after the injection of gadolinium.

Figure 2: Sagittal fast spin-echo T2-weighted image of the thoracic cord, demonstrating an hyperintense epidural hematoma compressing the cord posteriorly along the thoracic spine.
Nontraumatic spinal epidural hematoma was described as early as 1869 by Jackson (13). Since that time, many spontaneous spinal epidural hematoma cases have been reported. The standard therapy has been prompt evacuation of the hematoma, usually with good neurological recovery. Spontaneous spinal epidural hematomas include all forms of extradural spinal hemorrhages not consequent to vertebral trauma, coagulation disorders, vertebral angiomas, or any other apparent cause (4, 7, 21, 23, 26). Franscini et al. (10) reported a case of spinal epidural hematoma that was related to single high-dose aspirin intake and an additional platelet glycoprotein Ia/IIa deficiency. The characteristic clinical onset is sudden dorsal pain, with radicular radiation in over 50% of the cases. Intense local pain, in or over the spine, is followed by progressive sensory or motor deficits, usually within minutes or hours (seldom days), which may progress to complete paralysis (2, 3, 6, 17).

The incidence of spontaneous spinal epidural hematoma is higher in men. It tends to occur more often in the cervical segments in patients younger than 40 and the lumbar region in patients over 40 (11). Some authors suggest that congestion followed by rupture of the spinal venous system is the primary event (3, 14, 16, 22, 25). Beatty and Winston (4), on the other hand, suggest that an arterial source of bleeding originating from the extensive network of epidural arteries better explains the precipitous neurological deterioration seen clinically. Proof of either theory has been difficult to obtain because of a lack of angiographic documentation of the lesion as well as inconsistent explanations of the cause of the surgically removed clot. No cause is evident in most cases, even after open surgical exploration and removal of the clot (24).

This patient had been treated with an antiplatelet drug for three days and presented initially with impaired thrombocyte aggregation, which returned to normal values after the patient discontinued the antiplatelet drug. Irrespective of the cause of the hematoma, surgical treatment is usually performed in patients presenting with progressive signs of spinal cord compression (12, 18). The outcomes for these patients depend on the time interval between the onset of symptoms and the surgical therapy (19). Surgical treatment seems to be unnecessary when the clinical symptoms are not progressive or when early recovery is observed (20).

The therapy of choice for a spontaneous spinal epidural hematoma has been prompt surgical evacuation of the hematoma by laminectomies; usually with favorable results. In general, the earlier the operative evacuation, the better the neurological outcome (18, 19). In spite of this fact, some spontaneous resolutions have been reported with good outcome (1, 7). The authors of this report do not attempt to challenge the prudent general principle that prompt operative evacuation should be the standard treatment. Instead, they try to demonstrate that in the unusual case in which the patient’s rapid neurological deterioration is followed by early and sustained neurological recovery, confirmed by radiological resolution of the lesion, nonoperative therapy may be appropriate. Multilevel thoracic spinal epidural hematoma could be treated by removal of every other hemilamina of the involved space and evacuation of the hematoma. Total laminectomies could prove to be hazardous. Boukobza et al. (5) reported 11 cases of spinal epidural hematoma. Five of these patients were treated conservatively. Follow-up MRI on three patients revealed the disappearance of the hematoma after 6 days in one patient and after 2 months in two patients.
The first treatment choice for our patient with paraplegia was surgical decompression. She presented with a history typical of a spontaneous spinal epidural hematoma and showed clinical evidence of spinal cord compression at the lower T1 level. These clinical features can resemble those of an acutely ruptured thoracic disc, epidural neoplasia, transverse myelitis, a dissecting aortic aneurysm, congenital cysts, spondylitis, or an infection such as an epidural abscess (8, 20) but, the clinical history, radiological findings, and clinical evolution of this patient’s condition left no doubt in the authors’ minds that this was a hematoma. Furthermore, the results of an MRI [the modality of choice for the diagnosis of spinal epidural hematomas (20)] showing an isointense signal on T1-weighted images and the absence of gadolinium enhancement were consistent with our diagnosis.

CONCLUSIONS

If a patient is stable, improving neurologically, or at significant medical risk, a nonoperative course may indeed be appropriate. Individualization of therapeutic regimens to fit the specific clinical circumstances is prudent. Spontaneous spinal hematoma is a rare event that is usually thought to require immediate surgical therapy. MRI is the diagnostic tool of choice for the detection of spinal epidural hematomas.

References