Post-traumatic Lumbar Epidural Hematoma with Neurology:
Report of 1 Case

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The purpose of this study was to report a case with post-traumatic spinal epidural hematomas with abnormal neurologic findings, which is uncommon. A 40-year-old man presented at our clinic after a blunt trauma caused by a traffic accident in which he was a pedestrian. After admission, abnormal neurologic symptoms developed including loss of sensation and motor function in his left lower extremity. Magnetic resonance imaging demonstrated a spinal epidural hematoma with 40% canal stenosis at the L5-S1 level. Decompression including hematoma evacuation was done. Symptoms started to be reduced 18 days after operation. He was treated conservatively with medications and all symptoms resolved completely during admission and there were no further neurologic sequelae. Post-traumatic lumbar spinal epidural hematoma with abnormal neurologic findings is an uncommon condition that may present belatedly after trauma with significant neurologic compromise.

Key Words: Lumbar spine, Epidural hematoma, Neurology

Introduction

Spinal epidural hematoma (SEH) is an uncommon condition first described in the 17th century. There are over 260 case reports in the literature. The majority of these injuries are thought to occur spontaneously and to be associated with coagulopathy, vascular malformations, neoplasms, minor trauma, and pregnancy. Post-traumatic SEH with abnormal neurologic findings is very uncommon. We now report a case of post-traumatic SEH with abnormal neurologic findings in which the patient was a pedestrian injured in a traffic accident. SEH with neurologic compromise is a neurosurgical emergency that generally has been treated by emergency surgical decompression. Our SEH case is unique because of its association with blunt trauma, unilateral multiple fractures and the resolution of symptoms by surgical management.

Case Report

A 40-year-old man presented at our Emergency Department in a deeply drunken state. Initially, he did not complain of any pain or neurologic symptoms because he was inebriated. His initial vital signs were within normal limits (blood pressure, 108/69 mm Hg; heart rate, 117 beats/min; respiratory rate, 30 breaths/min; temperature, 36.0°C; 94% oxygen saturation breathing room air), and the examination was not remarkable for any deformity or tenderness. After he was awake, we redid the physical examination, and included sensory and motor deficits. He complained of tenderness over the lumbosacral spine. Neurologically, he presented with paresthesia over the left whole foot and had left foot drop. Motor power was about grade 4/5 strength of
knee flexion, 3/5 of knee extension, 4/5 of ankle plantarflexion, 1/5 of ankle dorsiflexion. But, grade 5/5 strength presented through right lower extremity and deep tendon reflexes were 2+ throughout.

A coagulation panel was normal with an international normalized ratio of 0.96 and a platelet count of 590,000.

There were no specific abnormal laboratory findings.

A plain X-ray revealed fractures involving the left inferior and bilateral superior pubic ramus, left sacral ala and spinous process of thoracic spines 2-8 level. An magnetic resonance imaging (MRI) revealed a fracture with bone marrow edema involving the left side of body and ala of the sacrum (S1-2), and hematoma in the spinal canal at the L5-S1 level (Figs. 1 and 2).

The patient was had surgery (partial laminectomy) on post-trauma day 7. L5/S1 and epidural hematoma evacuation was done. After the partial laminectomy at L5/S1, a massive hematoma with fluid was drained (Fig. 3). Operative findings included enlargement of fat tissue and ligaments in the spinal canal and hematoma formation due to
Postoperatively, we put the patient on a regimen of bed rest, serial neurological examinations, muscle strengthening exercises and pain control with nonsteroidal anti-inflammatory drugs. He remained in the orthopedic general ward unit with foot drop. A repeat MRI done on postoperative day 11 revealed removal of the hematoma in the spinal canal (Fig. 4). After postoperative day 18, motor power of left ankle was about grade 1/5 but the foot drop remained. At postoperative day 19, ankle movement in the horizontal plane was checked and at day 21, ankle movement was checked against gravity. At day 24, he started ambulation exercise. At day 27, the patient was discharged home on convalescent leave. On follow-up at 1-year and 10-months, he was able to engage in strenuous physical activity without neurologic sequelae.

Discussion

SEH is relatively rare and is seen mostly in the older literature. Groen and Ponssen [1] reported that 99% of sudden SEH occurs in the posterior region of the epidural space [1]. Localization of SEH to the lumbar area is also rare. Of the 49 cases of SEH reported by Makhham, only 5 were localized to the lumbar area [2].

The pathogenesis of SEH is not clear and risk factors have not been established. Risk factors for SEH include hemophilia A and B, coagulopathy with abnormal anticoagulants [3,4], intraspinal vascular malformation, spinal hemangioma, trauma and iatrogenic injury.

Some authors suggest the fragility of the epidural venous plexus as the cause of bleeding [3,5]. Vascular malformations were proven in some cases, but their involvement is uncertain [3]. Gundry and Heithoff [2] reported an association between epidural hematoma and rupture of a deep disc.

This led them to argue that a SEH would result from the tearing of adjacent fragile epidural veins to the annulus fibrosus or nucleus pulposus [2].

The clinical features of SEH are variable. There is severe spinal pain for some minutes to some days and paraplegia or tetraplegia [3]. MRI of a lumbar SEH has the shape of a biconvex mass [6]. The MRI signal of the mass varies, as do other hematomas [5].

The principle of differential diagnosis for SEH beneath the dura is different shape to others. On axial cutting, the mass shows a shape concave toward the marrow.

The differential diagnosis for SEH includes migration of a slipped disc, metastatic tumor, epiduritis and rarely a neural cyst [4].

In our case, functional recovery occurred after prompt surgical evacuation of the hematoma. But, some authors suggest that the rules of clinical and radiological monitoring without surgical intervention are important if minimal neurologic deficits are presented.

After a review of the literature, it appears that our case is the only post-traumatic ventral lumbar SEH with complete neurological dysfunction at the level of compression that was treated operatively with full recovery of function and with multiple fractures involving adjacent bony structures [7].

Patients with suspected SEH with neurologic dysfunction must be evaluated rapidly with computed tomography or MRI. Surgical management is determined by the surgeon based on the severity of the neurological deficits and their stability and progression. This case demonstrates an unusual injury with a unique presentation of the SEH after trauma, which was treated surgically with a good outcome.

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