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CASE REPORT

Spinal Spontaneous Epidural Hematoma

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Spontaneous spinal epidural hematoma is a rare but significant condition of spinal cord compression. Urgent decompressive surgery is generally indicated to prevent serious permanent neurological deficits and improve outcome. We encountered a case of an 83-year-old woman who sustained sudden onset of severe back pain, followed by progressive weakness and numbness over bilateral lower limbs. Magnetic resonance imaging of thoracic and lumbar spine demonstrated an epidural mass extending from T9 through L4, causing spinal cord and thecal sac compression. Emergent decompressive surgery was performed, and epidural hematoma was diagnosed postoperatively; the patient had significant improvement of neurologic deficits. The relevant literature is also reviewed.

Key words: Anti-platelet, decompressive surgery, spinal epidural hematoma

INTRODUCTION

Spontaneous spinal epidural hematoma (SSEH) is uncommon, and etiology remains obscure.[1] The typical symptom of SSEH is a sudden onset of severe back or neck pain, followed by symptoms and signs of rapidly evolving nerve root and spinal cord compression. The duration between symptom onset and accurate diagnosis is very important for SSEH because therapeutic outcome depends on the delay between diagnosis and surgical decompression. Management of SSEH remains a challenge for physicians. Here, we report a case with SSEH of thoracic and lumbar spine and reviewed the relevant literature.

CASE REPORT

An 83-year-old female was brought to the emergency department (ED) with the sudden onset of severe sharp low back pain, numbness, and weakness of both lower limbs while she talked on the phone at home. She fell and then above symptoms developed rapidly within 2 h. She couldn't stand up without assistance. There was no history of head and spinal trauma, smoking, drinking nor family history of vascular problems. She had left internal carotid artery stenosis (80%) and hypertension under regular medications, and she took different kinds of anti-platelets each time for 8 years (including aspirin, dipyridamole, ticlopidine, clopidogrel). On examination, the patient was alert, orientated and afebrile with a blood pressure of 123/77 mmHg. She had an unremarkable head, neck, chest, cardiovascular and abdomen examination. Neurological examination revealed the muscle power exhibited grade 1/5 in left lower limb and 0/5 in right lower limb. Decreased sensation below the T10 dermatome. Her reflexes of lower limbs were decreased. Acute urinary retention developed later, and Foley catheter was indwelled at ED. Laboratory investigation including complete blood count, chemistry panel, coagulation profile and bleeding time were all within normal limits. Magnetic resonance (MR) imaging demonstrated extensive posterior spinal epidural mass from T9 through L4 causing compression of the spinal cord and thecal sac. The mass was relative isointense on T1-weighted images and mild hyperintense on T2-weighted images. After injection of gadolinium, no apparent enhancement was seen within the mass [Figures 1 and 2]. Based on the rapid progression of clinical presentation, normal laboratory data, and imaging findings, infection and neoplasm were excluded. An epidural hematoma of thoracic and lumbar spine was highly suspected. Emergent decompressive laminectomy from T9 to L3 was performed. On operation, an epidural hematoma was discovered and evacuated. Pathologic report revealed hemorrhage without neoplasm or vessel malformation. After operation, muscle strength recovered well with 5/5 over left lower limb and 4/5 over right lower limb.

DISCUSSION

Spontaneous spinal epidural hematoma is a rare spinal emergency in the emergent department. The incidence of

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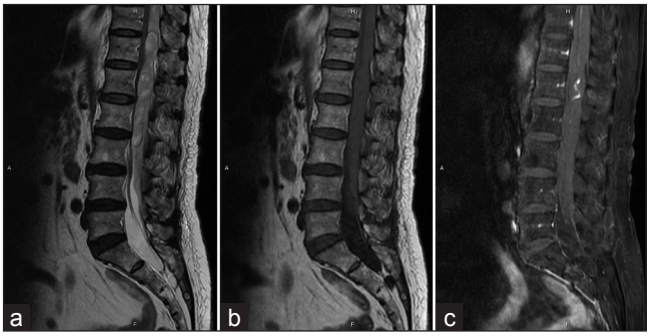

Figure 1: Magnetic resonance imaging of the lumbar spine. (a and b) Sagittal T2-weighted image and T1-weighted image revealed a heterogenous hyperintensity and isodensity posterior epidural mass extending from T9 to L4, respectively. (c) After injection of gadolinium, sagittal T1-weighted image revealed no enhancement

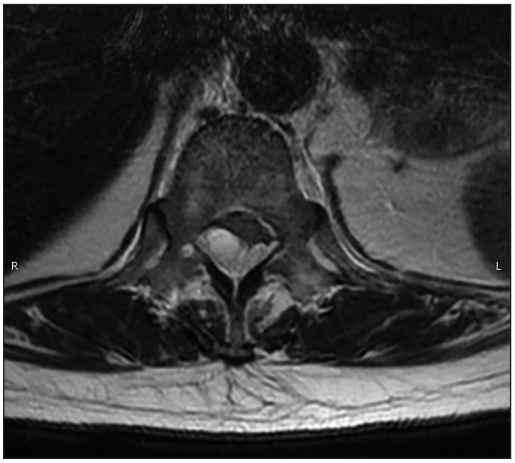

Figure 2: Magnetic resonance images of the T12 axial T2-weighted image show a predominantly hyperintense posterior extradural mass that compressed the spinal cord

SSEH has been estimated at 0.1 patients/100,000 individuals and represents <1% of spinal space-occupying lesions.[2] SSEH accounts for 40-50% of spinal epidural hematomas.[3] The sex ratio (male:female) is 1.5:1, and most SSEHs occur in patients between the ages of 50 and 80 years.[4] The etiology of SSEH remains unknown and has been associated with hypertension, anticoagulant therapy, increased intrathoracic and intraabdominal pressure (Valsalva maneuver such as straining, sneezing, lifting, or whooping cough), pregnancy or labor, and vascular malformation, long-term anti-platelet usage.[5-7] Although the exact pathogenesis of SSEH is unclear, bleeding from posterior epidural venous plexus is the most possible origin.[8] Sudden increasing pressure from the thoracic or abdominal cavity would result in abrupt increases in intravenous pressure. The prior local pooling within valveless, thin-walled epidural veins ruptured following the fluctuations of the pressures. Recent cases series showed 54% of SSEH patients reported a straining-associated event during the initial attack.[5] However, some researchers have considered spinal epidural artery as a rupture vessel because the pressure in venous plexus is lower than that in the epidural space and rapidly deteriorating neurological deficit clinically.[9] Further studies are needed to clarify the precise pathogenesis of SSEH.

The clinical presentations of SSEH are characteristic of sudden onset of severe neck and/or back pain often radiating to corresponding dermatome. Motor and/or sensory deficits caused by compression of nerve roots and spinal cord follow and progress within several hours depending on the levels of lesions. Despite the characteristic syndrome of SSEH, there may be a long delay between the onset of symptom and the development of neurologic deficits. If there is a delay in treatment, progressive spinal compromise can lead to permanent neurologic deficits or even death.[1] After the introduction and general usage of MR imaging, the early and accurate diagnostic capabilities have been improved than before. The early MR images of acute SEH reveal isointense or hypointense on T1-weighted images and hyperintense on T2-weighted images. After injection of gadolinium, peripheral enhancement of the lesion is mostly found, and central enhancement is occasionally found.[10]

Early decompressive laminectomy with evacuation of hematoma is considered as the best treatment for SSEH.[5] Conservative treatment is still an important option of treatment in some selective patients with mild and rapidly spontaneous recovery symptoms.[11] The outcome predominantly depends on the level and extent of the lesion, severity of the neurologic deficits, and the interval between onset of symptom and surgical decompression. In Shin's series, the outcome is good when patients with an incomplete injury were operated on within 12 h. Even patients with complete neurological deficits can improve if the surgical operation is performed within 24 h.[12]

Our case indicates that long-term use of anti-platelets can induce SSEH. Although there have been only seven reported cases of aspirin-related SSEH,[7] physicians should be aware of this rare but serious complication. Early decompressive laminectomy with evacuation of hematoma was undertaken, and the outcome was optimal.

CONCLUSION

Spontaneous spinal epidural hematoma is a rare but disabling or even fatal condition. SSEH associated with anti-platelet agents is even rare. Early diagnosis and management improve the prognosis but remain clinically challenge. Urgent spinal MR imaging is essential to confirm the diagnosis, and decompressive laminectomy with evacuation of hematoma is strictly indicated.

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