

The unexpected weakness: A case report of spontaneous spinal epidural hematoma

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ABSTRACT

Spontaneous spinal epidural hematoma is an uncommon cause of acute spinal cord compression. It is a neurologic emergency; urgent imaging and surgical intervention prevent permanent neurologic sequelae. Here, we present the case of a 71-year-old female known to have diabetes mellitus, hypertension, and ischemic heart disease on treatment. She presented to the emergency department with sudden onset of upper back pain with chest discomfort and bilateral lower limb weakness with numbness, which was waxing and waning for 4–6 h. On examination, there were no pulse or blood pressure deficits bilaterally and neurologically power that was 3/5 in both lower limbs with decreased tone and reflexes. In view of possible myeloradiculopathy, a magnetic resonance imaging whole spine was done which showed a lesion in the posterior epidural space, suggestive of hematoma, causing compression of the posterior thecal sac. The final diagnosis was spontaneous C7-T2 epidural hematoma secondary to antiplatelet therapy.

Key words: Clopidogrel, Neurologic deficit, Spinal cord decompression, Spontaneous spinal epidural hematoma

Spontaneous spinal epidural hematoma (SSEH) is a rare condition where there is bleeding into the epidural space without known trauma or obvious cause. The incidence is as rare as 0.1/10,000 emergency department (ED) visits [1]. It may be associated with arteriovenous (AV) malformations or coagulopathies [2]. Prompt diagnosis with surgical management is necessary to prevent permanent neurological damage. Only scattered case reports and a few reviews are available on SSEH till date.

CASE REPORT

A 71-year-old female patient presents to the emergency with a history of sudden onset back pain, chest discomfort, intermittent numbness, and weakness of both lower limbs for 6 h. The backache was sudden in onset in the mid-thoracic region, stabbing in nature, and non-radiating. It was associated with symmetrical numbness and weakness of both lower limbs which was intermittent. There was no history of fever, trauma bowel, or bladder involvement, slurring of speech, or facial involvement. She was a known case of diabetes mellitus since 15 years, hypertension since 20 years, and ischemic heart disease since 8 years, on treatment for the same (Clopidogrel 75 mg OD, Atorvastatin 10 mg OD, Telmisartan 40 mg BD, and Glimepiride 2 mg + Metformin 500 mg BD).

On examination, the patient was conscious, oriented, with a blood pressure of 130/80 mmHg, pulse rate of 84 beats per minute (regular, no pulse deficit), respiratory rate of 18/min, and SpO₂ of 97% at room air. The neurological examination showed

that the tone was reduced in both the lower limbs. The power of bilateral upper limbs was 5/5, and bilateral lower limbs were 3+/5. Bilateral biceps and triceps jerks were 3+ and bilateral knee and ankle jerks were 2+. Plantar response was flexor bilaterally. No loss of pain or touch sensation in upper or lower limbs at the time of examination.

Laboratory findings included hemoglobin value of 15.6 g/dL, white blood cell count of 12,500/mm³ (normal differential count), platelet count of 308,000/mm³, prothrombin time of 11.5, international normalized ratio of 1, activated partial thromboplastin time of 34, and potassium of 3.9 mEq/L. Bedside echocardiography showed no regional wall motion abnormalities and ejection fraction of >50%. A posteroanterior chest X-ray showed no abnormality. Magnetic resonance imaging (MRI) whole spine screening showed extra-axial intrathecal lesion noted in the posterior epidural space from C7-T2 (Fig. 1), causing compression of the posterior thecal sac (Fig. 2) suggestive of hematoma.

Based on these findings, a final diagnosis of spontaneous C7-T2 epidural hematoma secondary to antiplatelet therapy (clopidogrel) was made. The patient was transfused four units of random donor platelets and operatively managed after 10 h of ED presentation by spinal decompression and hematoma evacuation. The post-operative period was uneventful. She recovered well, lower limb power improved to 5/5 and the patient was discharged after the 8 days of hospital stay. After 7 days, sutures were removed, and the patient was followed-up in the neurosurgery outpatient department and then in the physiotherapy department.



Figure 1: Sagittal T2-weighted image of the spine showing hematoma in the posterior epidural space from C7-T2 causing posterior thecal sac compression

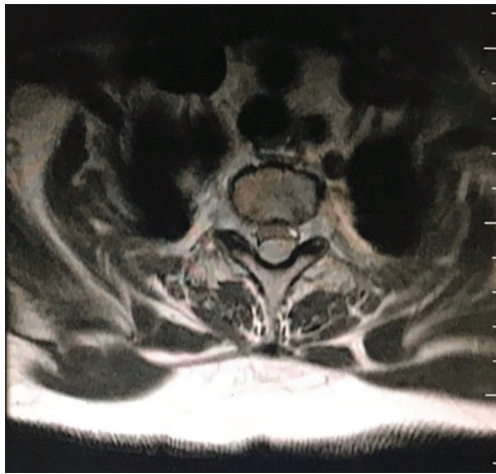


Figure 2: Axial T2-weighted image of spine showing a posterior spinal epidural hematoma with cord compression

DISCUSSION

SSEHs usually are prevalent in the fifth decade onward with a male preponderance [1,3]. They usually present as abrupt onset upper or lower back pain with neurological deficits. Early suspicion, diagnosis, and appropriate imaging are paramount to prevent permanent and devastating neurological deficits.

Our patient denied any history of trauma or injury/procedure to the back. She was on clopidogrel for cardioprotection, but otherwise denied any personal or family history of blood dyscrasias. Her platelet count was normal at the time of admission and she was evaluated for bleeding disorders which all turned negative. After ruling out all the common causes of SSEH, the patient was diagnosed to have spontaneous spinal EDH secondary to her clopidogrel therapy.

Risk factors for SSEHs are AV malformations, anticoagulants, coagulopathies, hemophilia, neoplasm, post-operative complication, or vertebral hemangioma [4,5]. About 40–60% of cases demonstrate no identifiable risk factors for the hemorrhage [5]. The source of the bleeding is postulated to be from epidural veins, arteries, and AV malformations [6]. The epidural venous system is a low-pressure system without valves and any changes in pressure can

lead to bleeding [6]. The dorsal epidural plexus is larger and more susceptible to SSEHs as it has an area of locus minoris resistentiae with minimal resistance and is not anchored as well as the ventral epidural venous plexus, hence placing it at more risk [6,7]. The areas of maximum movement along the spine are more susceptible to SSEHs, for example, cervicothoracic and thoracolumbar spines.

MRI is the gold standard investigation of choice for diagnosis. When compared to the spinal cord within 24 h from symptom onset, the hematoma typically appears isointense on T1-weighted and hyperintense on T2-weighted MRI imaging [1,5,7]. After 24 h, the hematoma often appears hyperintense on both T1- and T2-weighted images [1,7]. Chronic hematomas become hypointense on both T1- and T2-weighted images.

The treatment of choice for SSEHs typically is a hemilaminectomy or a laminectomy followed by irrigation and debridement [3,7,8]. If there is an obvious cause of coagulopathy, this should be addressed before surgical intervention [5]. The patients treated non-operatively, should be monitored with serial examinations while on strict bed rest [8].

CONCLUSION

The SSEHs are rare; however, if left untreated, the complications can be devastating. Delayed diagnosis and treatment beyond 48 h can lead to permanent neurological deficits. Our patient was a 71-year-old female with SSEH secondary to the antiplatelet therapy. ED physicians should consider SSEH after ruling out other causes of sudden onset of back pain with neurological symptoms and perform early imaging to diagnose and plan further treatment of SSEH.

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