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Case Report

Anticoagulant induced spontaneous spinal epidural hematoma, conservative management or surgical intervention—A dilemma?

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Abstract

Spontaneous spinal epidural hematoma (SSEH) is a rare cause of cord compression. SSEH with neurological deficit is an emergency situation that is commonly considered an indication for emergency surgical decompression. We describe a patient with SSEH who recovered clinically and radiologically with conservative treatment. A 25-year-old hypertensive male presented with acute onset back pain followed by asymmetrical paraparesis. He had sensory level at D9 dermatome with preserved bladder and bowel functions. He was taking anticoagulants for deep venous thrombosis of the left lower limb. Surgery was deferred because of the deranged coagulation profile. He was managed conservatively with correction of coagulopathy. After 3 days, he recovered significantly. Repeat neuroimaging revealed significant resolution of epidural hematoma. The conservative approach can be considered for selected patients who are unsuitable for early surgical intervention, those with stable neurological status [American Spinal Injury Association (ASIA) Scale E], or those in whom early recovery of function has been initiated with ASIA Scale C or D. Neurological status at presentation and suitability for surgical intervention seem to be important determinants of the type of therapeutic intervention.

Keywords: anticoagulant; conservative management; spinal epidural hematoma; spontaneous spinal epidural hematoma

1. Introduction

Spontaneous spinal epidural hematoma (SSEH) is a rare condition with an estimated incidence of 0.1 per 100,000 patients per year.[1] The etiology of SSEH is usually uncertain. The predisposing factors include vascular malformations, neoplasm, anticoagulation therapy, antiplatelet drugs, systemic hypertension, pregnancy, coronary thrombolysis, and very rarely following lumbar puncture.[2–4] In half of the cases, no predisposing factor is identified despite extensive evaluation. SSEH usually presents acutely with complaints such as neck pain or back pain, radiating pain, mild sensorimotor deficits, bladder or bowel symptoms, and even complete paralysis.[5–7] Occasionally, SSEH may present as cauda equina syndrome due to compression of spinal nerve roots.[5] Other disorders such as epidural tumor, epidural abscess, and acute disk herniation may mimic SSEH clinically. The most common presentation is acute onset back pain and radicular symptoms that may mimic disk herniation. Because SSEH may lead to severe neurological deficit and morbidity, prompt diagnosis and treatment are important. The primary treatment modality in SSEH is decompressive surgery, as a delay in intervention may result in a poor clinical outcome. Although the number of reports in the literature is increasing regarding spontaneous resolution of the SSEH, no evidence-based guideline is available on the optimal treatment of the

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SSEH.[8–15] We report a case of anticoagulant-induced SSEH that showed spontaneous rapid resolution of the SSEH on conservative management.

2. Case Report

A 54-year-old hypertensive man presented with sudden onset back pain followed by weakness of both lower limbs for 1 day. Weakness was felt more in the right lower limb compared with the left. He required the support of one person to walk due to weakness. He had a history of pain and swelling of the left lower limb 4 months previously, and was diagnosed to have deep venous thrombosis (DVT) on venous Doppler examination. Subsequently, he was placed on anticoagulant (warfarin, 7.5 mg/day) therapy. He had no history of falls or trauma to the back preceding the lower limb weakness. He denied any drug allergy or addiction. He was alert and fully oriented. The clinical examination revealed asymmetrical weakness of both lower limbs. The motor examination showed Medical Research Council (MRC) Grade 2 power proximally and Grade 3 power distally in the right lower limb. In the left lower limb, he had MRC Grade 4 power proximally as well as distally. He had American Spinal Injury Association (ASIA) impairment scale Grade C (details of the ASIA scale are shown in Table 1).[16] The sensory examination revealed the sensory level at D9 dermatome with sensory loss for pain, touch, and temperature in both lower limbs. Joint position sensations were impaired in both lower limbs. Deep tendon reflexes were suppressed in both lower limbs. The rectal tone was normal. He had no urinary complaints. There was no neurological deficit in both upper limbs.

Magnetic resonance imaging (MRI) revealed epidural hematoma in the dorsal aspect of the spinal canal extending from D3 to D8 levels, causing compression on the cord and displacing it anteriorly with secondary central canal stenosis at D5–D6 to D7–D8 levels (Figure 1). Blood investigations showed a prothrombin time of 77.3 seconds, an international normalized ratio of 5.93, and an activated partial thromboplastin time of 90 seconds. Blood cell counts, erythrocyte sedimentation time, liver function test results, and renal function test results were within normal limits. A venous Doppler of the left lower limb showed chronic DVT with partial recanalization. In view of the sensorimotor deficit with significant cord compression, neurosurgery opinion was sought and urgent decompression was recommended. As he had a deranged coagulation profile because of the anticoagulant, four units (15 mL/kg) of fresh frozen plasma (FFP) was given. Within 6 hours of FFP infusion, his weakness started to improve with one grade. Conservative treatment rather than neurosurgical intervention was considered in view of the improvement in neurological status. On Day 3 of hospital stay, he started to walk independently. Sensory deficit resolved completely. Repeat neurological examination revealed MRC Grade 5 power proximally and +4/5 distally in both lower limbs. An MRI of the spine was repeated after 5 days and revealed significant resolution of the epidural hematoma (Figure 2). After 7 days, he was again started on anticoagulant for DVT with monitoring of the coagulation profile. At 3 months of follow-up, he was independent without any neurological deficit.

Table 1
American Spinal Injury Association (ASIA) impairment scale.

ASIA scale	Degree of neurological impairment
A	Complete: no motor or sensory function preserved
B	Incomplete: sensory but no motor function preserved below the neurologic level
C	Incomplete motor function (>50%) of the key muscles below the neurologic level, motor grade < III
D	Incomplete motor function (>50%) of the key muscles below the neurologic level, motor grade ≥ III
E	Normal

3. Discussion

The underlying pathogenesis of SSEH is not clear.[2,3,17] The most important factor identified as contributing to SSEH is a hemorrhagic diathesis from either medications or disease states.[18,19] Only on rare occasions, SSEH is associated with an identifiable underlying vascular lesion. Various medications have been reported to be associated with SSEH. These include antiplatelet, anticoagulant, or thrombolytic medications, such as aspirin, warfarin, heparin, tissue plasminogen activator, and streptokinase.[20] Anticoagulants are mainly used in secondary prophylaxis of DVT, cerebral embolism secondary to lone atrial fibrillation or valvular heart disease, and cortical sinus thrombosis. Anticoagulation and/or thrombolytic therapy is also used in treatment of acute myocardial infarction.[20] In our case, the patient was taking warfarin for secondary prophylaxis of DVT. Anticoagulants, including warfarin, are used in 25–70% of patients with SSEH and are an important risk factor.[21,22] Coagulation variables are within the therapeutic range in many of the reported cases of SSEH.[21] In such cases, epidural bleeding may be initiated because of other factors. In addition to use of anticoagulant agents, the relationships with hypertension, some structural extradural anomalies, the rupture of fragile epidural veins by an adjacent herniated disk, trivial trauma, and straining are suggested.[21,23–25] Omori et al[26] pointed out the possibility of the involvement of arterial problems and cervical spondylitis.

One hypothesis suggests that elevated intrathoracic and/or intra-abdominal pressure results in rupture of the vertebral venous plexus.[27] The most frequent reported site of SSEH is the cervicothoracic region with the predominant involvement of the dorsal side of the spinal canal.[23,28] The posterior internal vertebral venous plexus in the cervicothoracic region is bigger and more convoluted compared with the anterior one. Beyond this, the posterior venous plexus is uncovered by ligamentous structures. Therefore, this hypothesis seems to be important for the underlying mechanism of SSEH.[28] Another hypothesis postulates arterial rupture as an origin of SSEH as the intrathecal pressure is greater than the vertebral venous plexus.[29] In our case, SSEH was located in the dorsal aspect of