Spontaneous spinal epidural haematoma associated with aspirin intake

Oh J Y L, Lingaraj K, Rahmat R

ABSTRACT

Spontaneous spinal epidural haematoma (SSEH) is rare. No identifiable cause is found in the majority of cases, while anticoagulation accounts for up to 17 percent of cases. Aspirin-associated SSEH, however, has rarely been described in literature. A 62-year-old man on prophylactic aspirin presented with symptoms of acute cord compression due to spinal epidural haematoma that was confirmed on magnetic resonance imaging. An emergency decompression laminectomy was performed ten hours after the onset of his symptoms. No vascular anomaly was detected. Our patient recovered well and regained full motor and sensory function. Aspirin is unlikely to be the direct cause of SSEH but may predispose to it, with the underlying cause being a locus minoris resistentiae, consisting of a network of weakened epidural veins. Early diagnosis and treatment are essential for a successful outcome.

Keywords: acute spinal cord compression, aspirin-associated spontaneous spinal epidural haematoma, aspirin complication, cord compression, spinal emergency, spontaneous spinal epidural haematoma

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INTRODUCTION

Spontaneous spinal epidural haematoma (SSEH) is an uncommon condition. In the majority of cases, no identifiable cause is found, while other factors such as anticoagulation, arterivenous (AV) malformations, pregnancy and trauma have been implicated.[1] Aspirin-associated SSEH is not so common. We report a case of SSEH in a patient receiving prophylactic aspirin.

CASE REPORT

Our patient, a 62-year-old Chinese man, was woken up in the early hours of the morning by the onset of low back pain. This was followed by progressive bilateral lower limb weakness and numbness, as well as urinary retention, for which he was subsequently seen at our emergency department within four hours. There was no history of trauma, although he was involved in strenuous lifting the day before. Relevant past medical history included hypertension and ischaemic heart disease, for which a coronary artery bypass graft was performed in 2004. The postoperative course was uneventful and he was placed on aspirin 100 mg and atenolol 50 mg daily. There was no history of anticoagulant therapy or bleeding diathesis.

Clinical examination four hours after the onset of symptoms revealed paraplegia of both lower limbs (Medical Research Council grade 0) with a sensory level at the L1 dermatome. Reflexes were depressed. Rectal examination showed a lax anal tone, with decreased perianal sensation. There was no neurological deficit in the upper limbs. Haematological investigations, such as platelet count and coagulation profile, were within normal limits. Magnetic resonance (MR) imaging of the thoracolumbar spine revealed an epidural haematoma posterior to the spinal cord, extending from the T9 to T12 vertebral levels. The dimensions of the collection were noted to be largest at the T10 level, where it measured 6 mm in depth and 10 mm in width. Cord compression was noted at this level (Figs. 1 & 2). No abnormal signal changes were seen.

An emergency decompression laminectomy was performed from T8 to T12 vertebral levels, ten hours after the onset of his symptoms. Intraoperatively, bleeding was seen to be arising from a dural vein at the site of the haematoma, which was evacuated and haemostasis was achieved. No vascular anomaly was detected. The patient made an excellent recovery after surgery and regained full power (Medical Research Council grade 5) in two weeks. He was able to ambulate without assistance and was discharged soon after. The indwelling catheter was removed four weeks postoperatively, after urological investigations revealed normal bladder function.

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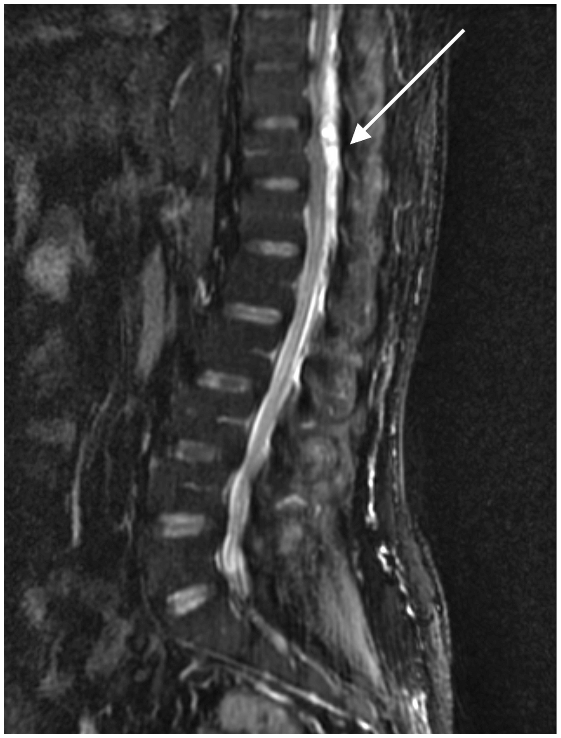

Fig. 1 Sagittal T2-W MR image shows an epidural haematoma at the T9–T12 levels.

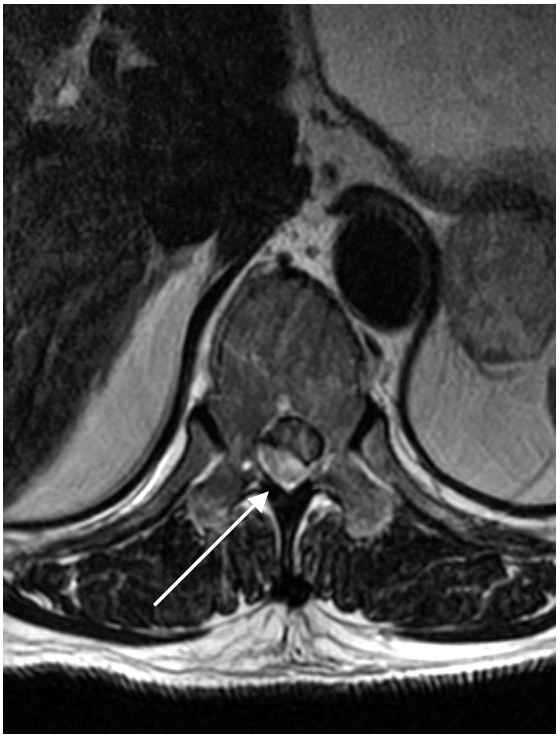

Fig. 2 Axial T2-W MR image shows the epidural haematoma is largest at the T10 level, causing cord compression.

DISCUSSION

Spontaneous spinal epidural haematomas are rare. No identifiable cause is found in the majority of cases, while anticoagulation accounts for up to 17% of cases. Other factors, such as AV malformations, pregnancy and trauma, have also been implicated.[1] To the best of our knowledge, aspirin-associated SSEH has only been reported twice in literature. Locke et al described the first case where a healthy 36-year-old man developed an acute spinal haematoma at the T5–T6 levels after taking aspirin (1,500 mg in two hours) for indigestion.[2] Following this, Heye reported a 79-year-old man presenting with paraplegia from a T10 haematoma while taking prophylactic aspirin (250 mg/day).[3] In both cases, there was no prior history of trauma or anticoagulation. Platelets counts were also within normal limits. However, Locke did find that the bleeding time in his patient was significantly prolonged by up to three times. This test more accurately reflects platelet dysfunction, though it is not routinely performed.

An interesting case to suggest a possible link between aspirin and SSEH was reported by Rose et al,[4] whose patient developed SSEH after ingestion of an excessive amount of garlic (2,000 mg daily). Garlic has antibiotic and fungicidal effects that accelerate fibrinolysis and inhibit thrombocyte aggregation. These properties are similar to those of antiplatelet preparations, such as aspirin. Clopidogrel, another antiplatelet preparation, has also been found to be associated with SSEH.[5] Nevertheless, some authors believe that factors such as antiplatelet agents, anticoagulant therapy and pregnancy are unlikely to be the direct cause of spinal epidural haematomas, given the high prevalence of these factors in the general population and the overall low incidence of spinal epidural haematomas. Rather, they postulate that the underlying cause of spinal epidural haematomas is a network of weakened epidural veins or a "locus minoris resistentiae" that ruptures upon transmission of increased intrathoracic or abdominal pressure.[1,6] This hypothesis is supported by the observation that most spinal epidural haematomas are situated dorsally where the epidural plexus is larger as compared to its ventral counterpart. In addition, SSEHs are commonly found at the cervicothoracic and thoracolumbar junctions, where extensive networks of venous convolute are found. In our case, the patient gave a history of heavy lifting prior to the onset of symptoms. This may have contributed to an increase in intrabdominal and thoracic pressure, leading to the rupture of a weakened venous plexus, as alluded to earlier.

SSEH may present with sudden onset back pain, quadriplegia and bladder dysfunction, depending on the extent and location of the lesion. The diagnostic investigation of choice is MR imaging, which gives

information on the spine and spinal cord in multiple planes. Axial images assess the degree of spinal cord compression, while sagittal images show the extent of the haematoma. The treatment of choice is surgical evacuation and neural decompression. Isolated cases of SSEH being successfully treated conservatively have been reported, but in these cases, the patients showed improving neurological signs at presentation.[7,8]

The rapid and complete neurological recovery seen in our patient reflects the importance of early diagnosis and treatment. Various authors have shown that the time to surgical decompression is an important predictor of a positive outcome.[9] Groen and Van Alphen demonstrated that patients who were treated within 48 hours had a significantly better chance of complete recovery that those treated after this critical time period (54% vs. 26%).[10] In conclusion, SSEH is a rare condition, in which aspirin may be considered a predisposing factor. The underlying cause may be a locus minoris resistentiae, consisting of a network of weakened epidural veins. Early diagnosis and treatment are essential for a successful outcome.

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