

Spinal epidural hematoma due to anticoagulant therapy: a case report and literature review

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ABSTRACT

This case report is to enlighten the awareness on spinal epidural haematoma due to warfarin. A 58-year-old man who had been on prophylactic anticoagulant therapy after aortic valve replacement since 1998, had presented with acute onset of reduced sensation and weakness in both lower extremities 3 days prior to the admission to our centre. His prothrombin time (PT) was 46.8 seconds, international normalized ratio (INR) was 5.11, and activated partial thromboplastin time (APTT) was 167.6 seconds (control, 31.3-46.1 seconds). Magnetic resonant imaging scan revealed haematoma in post epidural space at the level of Lumbar 2 and 3. He underwent evacuation and posterior instrumentation on the 6th day. Spinal epidural haematoma should be included in the differential diagnosis of progressive spinal cord and nerve root compression in patients whom are receiving anticoagulant therapy. Prompt diagnosis and early surgical decompression would positively lead to a good outcome.

Keywords: Spinal epidural haematoma, Anticoagulants, Warfarin, Spinal cord compression.

INTRODUCTION

Spinal epidural haematoma results from the rupture of fragile epidural venous plexus. Epidural venous plexus in the spinal epidural space lacks venous valves, and undulating pressure from the thoracic and abdominal cavities could impact it directly (1). Spinal epidural haematomas (SEH) are rare, most caused by trauma, anticoagulant therapy, vascular anomalies, hypertension, blood dyscrasias, epidural anaesthesia or, on the odd occasion, spinal surgery. Spinal hematoma has been described in autopsies since 1682 and had been diagnosed clinically from the time 1867. Being a rare condition and usually presented with severe neurological disorder, it could lead to permanent neurological deficit if left untreated (2).

CASE REPORT

A 58-year-old man who had been on prophylactic anticoagulant therapy (Warfarin 3mg/day) after an aortic valve replacement for valvular heart disease since 1998, presented with acute onset of reduced sensation and weakness in both lower extremities 3 days prior to the admission to our centre. The condition was precipitated by lifting a flower pot weighing 7 kg. It was associated with impaired bladder and bowel function. His neurological status was L1 American Spinal Injury Association Impairment Scale (AIS) B and he was diagnosed with cauda equina syndrome upon admission. White blood cell count was $16.8 \times 10^9/L$, Hemoglobin level was 13.9g/dL, platelet count was $338 \times 10^9/L$, Bilirubin was 19 $\mu\text{mol/L}$ (Normal, <23), albumin was 33g/L (Normal, 35-50g/L), ALT was 108 $\mu\text{mol/L}$ (Normal, <44), AST was 173 $\mu\text{mol/L}$ (Normal, 32-104). His renal profile was normal. His prothrombin time (PT) was 46.8 seconds (control, 11.4-14.2 seconds), international normalized ratio (INR) was 5.11 (therapeutic range, 2.4-4.0), and activated partial thromboplastin time (APTT) was 167.6 seconds (control, 31.3-46.1 seconds). Warfarin was withheld and he was given 4 units of fresh frozen plasma (FFP) on the day of admission

and started with a course of heparin infusion. Two units of FFP had been given on 2nd day. INR was managed to be controlled below 3 within 48 hours. Magnetic resonant imaging (MRI) scan revealed haematoma in post epidural space at the level of Lumbar 2 and 3 (Figure 1, 2). He underwent evacuation and posterior instrumentation on the 6th day. Patient was advised to undergo a 4 hourly clean intermittent self-catheterization (CISC) due to the neurogenic bladder. Although the lower-extremity neurological status had improved to L1 AIS C, the patient however still had neurogenic bladder problems upon discharge which was 2 weeks after the operation. He was advised to do a 4 hourly CISC. The patient's neurology improved to L3 AIS C a year later however he still experiences neurogenic bladder and bowel problems.

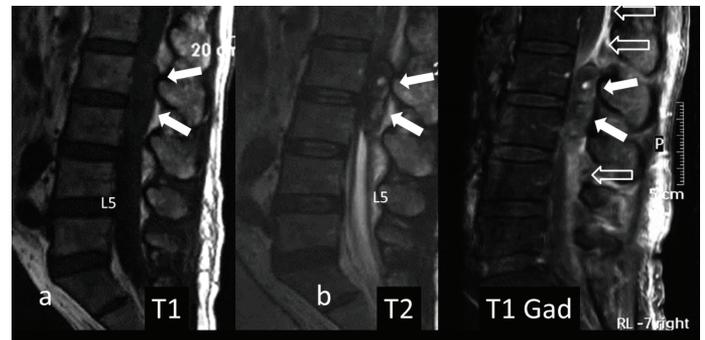


Figure 1: Sagittal MRI of the lumbosacral spine in T1 (a), T2 (b) and post contrast T1 (c) weighted images. The posteriorly located epidural haematoma (solid arrows) at L2 and L3 levels is compressing on the cauda equina anteriorly. This haematoma shows low signal on both T1 and T2 weighted images with a focal of high signal intensity on T2. There is no contrast enhancement in this haematoma. There is some reactive dural enhancement at superior and inferior to the haematoma (open arrows).

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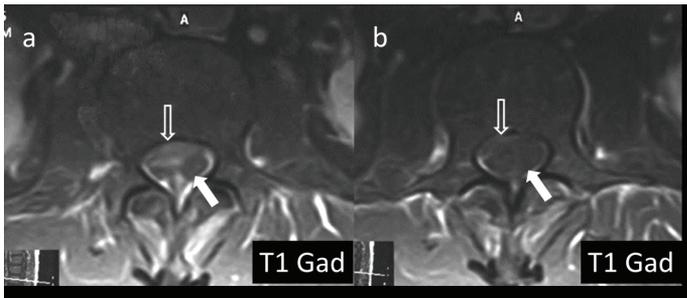


Figure 2: Sequential axial post contrast T1 weighted images just above (a) and at the level of (b) the epidural haematoma showing anterior displacement and compression of the cauda equina (open arrows) by the epidural haematoma (solid arrows).

DISCUSSION

The sites of predilection of spinal epidural haematoma are the cervicothoracic and thoracolumbar junction involvement with an average of 3.8 spinal segments (3). Intense, knife-like pain at the location of the hemorrhage can be an initial presenting symptom which can lead to progressive paralysis below the affected spinal level. Motor and sensory deficits with neurogenic bladder and bowel were the main presenting symptoms in our case. Symptoms of meningitis, disturbances of consciousness, and epileptic seizures were the presenting symptoms of Subarachnoid hematoma which can be misled as cerebral hemorrhage. This occurrence was common among the age group between 55 and 70 years old with male preponderance. The severity of the preoperative symptoms and timing of surgical decompression determined the chances of neurological recovery (2). Gelabert et al reviewed 8 cases of Spontaneous spinal epidural hematomas. The median of the patients' age was 63.5 years with female preponderance. Acute vertebral pain was the most common presenting symptom followed by cord compression syndrome (4). MRI was the investigation of choice as it can readily distinguish spinal epidural haematoma from other lesions (2,5). Spinal epidural haematoma was isointense in T1-weighted images and hyperintense in T2-

weighted images on MRI (4). Tomarken believed that low-molecular-weight heparin agents should be prescribed with caution as it can lead the rise to a significant risk of spinal hematoma which was catastrophic to the patient (6). Early surgical decompression of haematoma in patients with less severe preoperative symptoms will contribute to the increment to the high chances of complete recovery. Thus, timely clinical recognition of cord compression due to spinal hematoma led to prompt diagnosis and early effective intervention resulting in positive outcome (2,4). Delay in surgery for more than 6 hours reduced the probability of recovery (7). Lawton et al reported that improved neurological outcome was noted in patients who had surgery within 12 hours of the onset of neurological symptoms (8). It was therefore essential to recognize the relatively typical clinical presentation of spinal hematoma in an opportune manner to allow correct diagnostic and therapeutic measures to be taken to maximize the patients' chances of complete recovery (2). Prompt diagnosis and clot evacuation before severe spinal cord compression and subsequent ischemic necrosis favors good prognosis. Spinal cord subdural hematoma should be considered as one of the differential diagnoses of progressive spinal cord and nerve root compression in patients receiving anticoagulant therapy. Prompt diagnosis and early surgical decompression would positively lead to a good outcome (9). As prolonged period of spinal cord ischemia due to hematoma collection led to permanent spinal cord damage, prompt diagnosis and clot evacuation was mandatory to achieve good prognosis and functional recovery (10).

In our case, patient had a good neurological recovery however, neurogenic bladder and bowel problems still remained after a year. This could possibly be due to the fact that patient presented to the hospital a few days subsequent to the onset of problems which delayed surgical decompression.

Although the spinal cord compression due to spinal epidural haematoma resulting from over warfarinisation is not an uncommon entity, this problem should be listed first in the mind when patients on warfarin develop neurology, as prompt diagnosis and timely evacuation is the mainstay of treatment to improve the clinical outcome and patients' quality of life.

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