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CASE REPORT

SPONTANEOUS SPINAL EPIDURAL HAEMATOMA IN A PATIENT ON ORAL ANTI-COAGULANT THERAPY: A CASE REPORT

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ABSTRACT: Sponaneous spinal epidural haematoma (SSEH) is an uncommon cause of spinal cord compression. It may be associated with various causative factors, but in many patients, anticoagulation can be implicated. SSEH should be suspected in any patient receiving anticoagulant agents who complains of limb weakness, sensory deficits and / or urinary retention. Early diagnosis and treatment are very important for the functional recovery of the patient. Spinal magnetic resonance imaging is the most suitable neuroradiological method for early diagnosis. Although primary management is the surgical evacuation of spinal epidural haematoma via laminectomy, rare cases in which the patient is improving rapidly and progressively could be treated conservatively. A 45-yr-old woman with a spontaneous spinal epidural haematoma who was receiving acenocoumarol treatment for a mechanical mitral valve who improved with conservative management is presented in this article.

KEYWORDS: spontaneous spinal epidural haematoma, oral anticoagulant therapy.

INTRODUCTION: Spinal Epidural Haematoma is a rare entity and only few cases have been reported worldwide. It is associated with significant morbidity. Various etiological factors include idiopathic, trauma, anticoagulant therapy, epidural or spinal anaesthesia, bleeding diathesis, vascular anomalies, post operatively in spinal surgeries.[1]

Its presentation is remarkably uniform with sharp local pain followed by sensory, motor and/or bladder dysfunction.[2] Early diagnosis and decompression is the treatment modality of choice for a good result and neurologic recovery.[6] Nevertheless, there are cases worldwide wherein recovery with the conservative management was reported as in our case.[3] In patients with SSEH associated with excess use of anticoagulant therapy, immediate interruption and correction by coagulation factors (fresh frozen plasma) and vitamin K is necessary when there is suspicion of SSEH.[4] Magnetic Resonance Imaging (MRI) should be the first diagnostic tool.[5] Outcome of SSEH is inversely proportional to duration of spinal cord compression. [6] Therefore early surgical removal of clot is indicated and conservative management can be continued if rapid neurological recovery occurs.[6]

We present the following case to alert the physicians for several reasons:

Firstly, since there are many indications and widespread use of anticoagulant/thrombolytic therapies: SSEH to be suspected in a patient on such medications presenting with local radicular pain, motor, sensory, bladder disturbances.

Second, if the correct diagnosis is too late for successful treatment, permanent paralysis may set in, which is likely beyond 24hrs.[7]

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CASE REPORT: A 45yrs old female who was on oral anti-coagulant therapy following MVR with St. Judes mechanical valve after taking excess of acenocoumarol for 4 days, (total dose 24mg) presented with two day history of sudden onset rapidly progressive weakness in right lower limb followed by left lower limb in a day with urinary retention and constipation. Patient was confined to bed at the time of presentation. No history of any drug abuse. No history of trauma. No history of any other co-morbidity (hypertension /diabetes). On examination she was conscious and coherent. Power was 2/5. In both lower limbs, superficial reflexes - abdominals absent and plantars not elicitable, deep tendon reflexes knee and ankle jerk absent. Pain, touch, temperature sensations were absent below T12 level, Joint Position Sense and vibration sense were intact. After Foleys catheterization, 2litres of urine was emptied. No signs or symptoms of other system (Higher Intellectual Functions, cerebellar, autonomic, cranial nerves) involvement. No signs of any other muco-cutaneous bleeds.

MRI thoraco-lumbar spine showing T1W and T2W hyperintense lesion not suppressed on STIR noted in epidural in location extending from inferior border of D12 to superior border of L4 suggestive of EPIDURAL HAEMATOMA. PT/INR was 5. 42 initially, after holding acenocoumarol for 5days PT/INR was 1. 64. Complete blood counts were normal, platelets- 3lakhs/cu mm. In 2d-echo, gradients across the valves were good, no thrombus, prosthetic valve was functioning good, ejection fraction-60%. Acenocoumarol was stopped and FFP's were given for two days; vit k 10mg once daily was administered for 5 days. Within 24hrs of presentation patient's power in both lower limbs improved to 4/5, pain and temperature sensation improved with complete recovery of bladder function on day 8 and she was discharged restarting anticoagulants for her cardiac indication.[13]

MRI images of patient: showing hyper intensities in T1W, T2W image from D12 to L4.

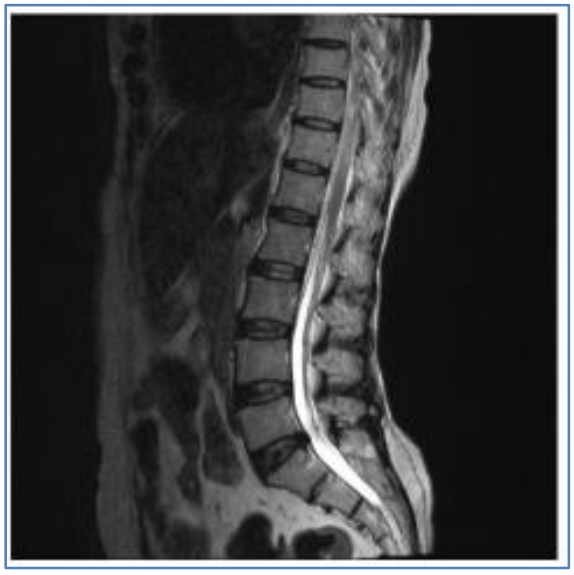

Fig. 1: T2WI

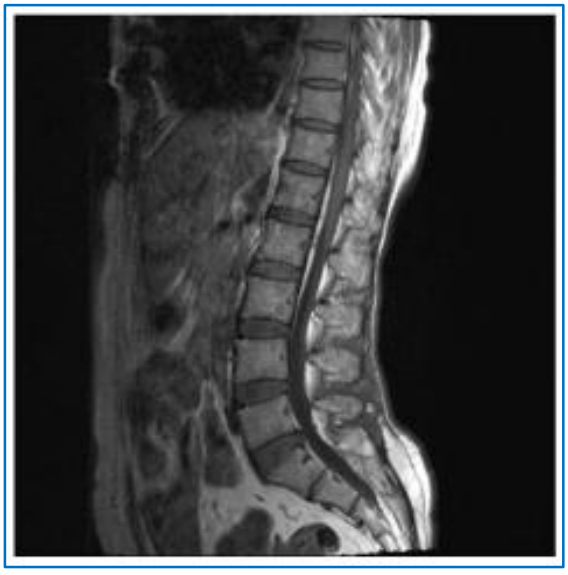

Fig. 2: T1WI

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DISCUSSION: We present a case of SSEH in a patient on anticoagulant therapy for MVR after taking in excess for 4days inspite of proper written advice by doctors regarding dosage, timing and follow-up PT/INR.

SSEH is a rare but an important cause of acute compression of spinal cord. The term spontaneous refers to haemaorrages that are not associated with trauma, tumors or vascular malformations.[8] SSEH occurs in all age groups. Cases reported from 11/2 months to 80yrs. Most cases peaking at two age groups; 15-20yrs and 45-75yrs.[1] Its reported more in males with ratio of M: F =2: 1.[1] Our case was a female who was 45 yrs old. One explanation for peak incidence in elderly is that associated factors like hypertension, atherosclerosis may contribute to it. But SSEH should be suspected in patient of any age group receiving anticoagulation.[7] Regarding most common location of haematoma, it was observed that thoraco-lumbar in adults as in our case and cervical spine in children[1] because weak vascularisation of epidural venous plexus is seen here.

SSEH occurs rarely in comparision with traumatic spinal EDH. It may be associated with various causative factors but in many patients anticoagulation can be implicated. Here we emphasize to suspect and look into if the patients have had an excess dosage complicating into bleeding/haematoma formation. Life threatening central nervous system bleeds can occur without any external muco-cutaneous bleeds.[9] In this scenario a clinician requires a high index of suspicion for SSEH. Therefore we recommend it to be suspected in patients on anticoagulant therapy with apt clinical features.

Clinical presentation is acute local radicular pain followed by motor paralysis, sensory disturbances and/or bladder disturbances below the level of hematoma. This patient had all the typical manifestations of myelopathy. There are case reports in literature where patients also presented with Brown-Sequard[12] syndrome/hemiplegia depending on extent of spinal cord compression.

Magnetic Resonance Imaging (MRI) is the diagnostic tool of choice. [5] After the advent of MRI as the diagnostic tool, there is an increase in incidence of new cases and thus leading to further necessary management.[5] Zain et. Al. suggested that may be MRI is able to diagnose cases of SSEH which are earlier missed clinically because of very early onset of recovery. In our case, T1 and T2Weighted MRI was showing hyperintense lesion in epidural space extending from T12 to L4 suggestive of Subacute SSEH.[5] Haematoma was antero-lateral in position thus sparing joint position and vibration sense.

Prothrombin time and INR ratio were high indicating a high potential to bleed including central nervous system as in our case, but there were no muco-cutaneous bleeds associated, which is very challenging to a clinician; and to have a high index of suspicion is the need. Nevertheless, there are case reports where SSEH occurred in therapeutic INR range. [7] Hence we recommend SSEH to be suspected in a patient on anticoagulation with typical presentation irrespective of PT/INR value. There may be no connection between severity of clinical presentation and severity of coagulopathy,[1] as in our case with life threatening CNS bleed (INR of 5) without any muco-cutaneous bleeds.

Theories suggesting cause of rapid recovery in a case of coagulopathy as in ours are: immediate replacement therapy[10] haematoma stays in a liquid state for a longer time which may spread easily into wide epidural space.[11]

CONCLUSIONS: Any patient on anticoagulant therapy irrespective of PT/INR if presenting with features of myelopathy, high level of suspicion regarding cord compression because of epidural bleed should be entertained. MRI spine should be performed urgently and diagnosis of SSEH to be established or ruled out for further management as prompt cessation of anti-coagulant therapy can prevent severe neurological deterioration. In our case because the patient has taken medication more than prescribed dosage with poor follow-up with PT/INR, it has led to this complication. Hence thorough patient education at the time of discharge is very much essential.

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