Case Report

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Spontaneous Spinal Dorsal Epidural Hematoma in Term Pregnancy – A Case Report

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Abstract

Spinal epidural hematomas are rare, accounting for less than 1% of all spinal canal space occupying lesions.[1-3] Spontaneous spinal epidural hematoma has an incidence of 0.1 in 100,000 per year[1,4], with incidence in pregnancy being very uncommon. In this case report, we present a case of a 23-year-old primigravida at 36 weeks of gestation with complaints of sudden onset neck pain, quadriplegia and loss of sensation.

Keywords: Spontaneous spinal epidural hematoma; Pregnancy; Cervical.

INTRODUCTION

Spontaneous Spinal Epidural Hematoma (SSEH) is defined as blood within the epidural space, without a traumatic or iatrogenic cause.[1] The first case was reported in 1869 and approximately 400 cases have been reported.[5] The clinical features may vary from acute onset of neck pain to progressive paralysis, and spectrum may depend upon the speed of hematoma formation with extend of compression of the cervical cord. A search of the relevant literature revealed reports of 37 cases of SSEH during pregnancy between 1966 and 2020; 31 of these cases involved the thoracic spine .[6] The etiology and pathogenesis of SSEH is still unknown, but is considered a serious emergency that must be managed immediately to prevent permanent neurological deficit in mother and still birth.[6]

CASE STUDY

A 23-year-old primigravida at 36 weeks + 5 days of gestation, presented to outside hospital with complaints of sudden onset of severe neck pain-continuous in nature, radiating to bilateral upper limbs and associated with tingling sensation of the whole body. The patient within a course of 3 hours developed weakness of all four limbs and loss of sensation, below the upper chest (lower limb >upper limb). There was no associated bowel or bladder incontinence with these symptoms. There was no history of trauma, injuries, strenuous exercise or any other events preceding the onset of symptoms. The patient was a primigravida and had an uneventful pregnancy with regular antenatal visits. She was on iron, calcium and other nutritional supplements which were advised during the pregnancy by her obstetrician. She had no history of any coagulopathies, comorbidities and had no history of intake of any anticoagulants or antiplatelets. There was no history of similar diseases, coagulopathy or vascular malformations in the family. The patient on evaluation in outside hospital had stable vital parameters (HR-77/min,

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BP-120/70mm Hg, RR-20/min, SpO_2--99% in room air). The fetal condition was assessed using fetal doppler and found to be normal. She was evaluated by a team of obstetricians and neurosurgeons and decided to take the patient to emergency LSCS in view of neurological deficits. Emergency LSCS was done and the child was delivered safely. She was later referred to our ER for further management.

On arrival in the ER, the patient had a GCS -E4V5M6, talking in full sentences and with no signs of airway compromise. RR-20/min with a SpO_2 -97% in room air, HR- 80/min and BP was found to be 100/60 mmHg and random blood sugar -125mg/dl. Central nervous system examination showed a power of 2/5 in bilateral upper limb and 0/5 in bilateral lower limb. Deep tendon reflexes were depressed bilaterally, with absent abdominal reflexes and bilateral plantar showing no response. She had a loss of all sensations below the level of nipple, corresponding to that of T4. Other systemic examinations were within the normal limits.

Lab investigations:

EDTA whole blood:

Hb: 10.5g/dl

TC: 11060/mm^3 (DC – N78 L14 M07 E01)

Platelet: 1.81lakhs/mm^3

Coagulation profile:

BT: 2.30min

CT: 5.30min

PT/INR: 13.0/1.00

APTT: 30.2

Serum sample:

S. Urea: 33.0mg/dl

S. Creatinine: 0.50mg/dl

Sodium: 137.0mEq/L

Potassium: 3.6mEq/L

LFT: within normal limits

TSH: 1.57microIU/ml

Serology: Non-Reactive

Blood group and Rh typing: A Positive

ECG: sinus rhythm with no significant ST-T changes

2D ECHO: No RWMA, normal diastolic function, LVEF-60%

MRI cervical spine with contrast

T1 weighted image: isointense biconvex lesion outside dura matter in the posterior part extending from the C3 to C7 vertebrae seen in the sagittal plane.

T2 weighted image: hyperintense lesion seen in the sagittal plane extending from C3 to C7 vertebrae outside the dura matter posteriorly, with compression on the cord.

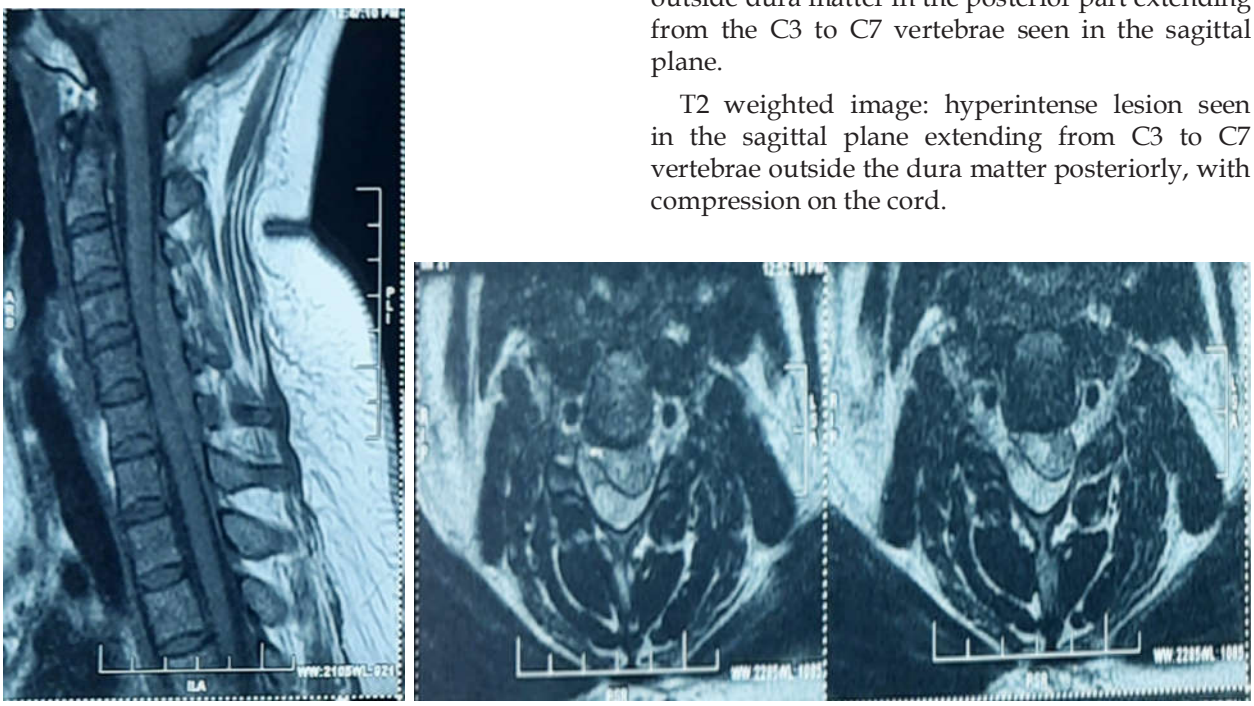
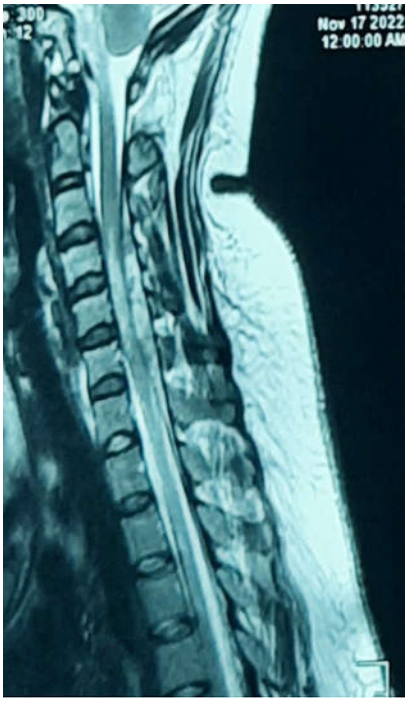


Fig. 1 and Fig 2: T1WI in sagittal and axial views respectively showing isointense lesion in epidural space in the cervical region.



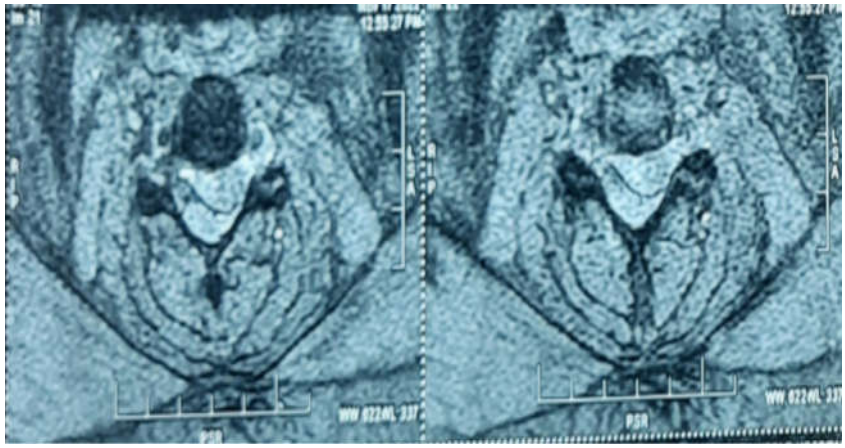


Fig. 3 AND Fig. 4: T2WI in sagittal and axial views respectively showing hyperintense lesion in dorsal epidural space in the cervical region.

The patient was admitted and evaluated. She developed features of spinal shock (vitals: PR-64/min, RR-22/min, SpO_2-90% in room air and BP-90/50mmHg). The patient was started on NORADRENALINE INFUSION (4/40 at 5ml/hr) and DOPAMINE infusion (200/50 at 4ml/hr). In view of the spinal shock, she was taken up for surgery after detailed informed consent, explaining the severity of neurological deficit and unpredictability of the time and extent of neurological recovery. The patient underwent C3-C6 cervical laminectomy, evacuation of the epidural hematoma and bony fusion under GA. At surgery, very satisfactory hematoma evacuation and cord decompression was achieved. The lateral mass and joints were decorticated and fusion was done with bone graft. The post-operative period was uneventful and the vitals improved and was extubated and tapered off from the ionotropic support. The patient underwent regular physiotherapy along with other supportive management and showed an improvement in the power (bilateral upper limb- >3/5) and return of sensation of both upper limbs and lower limbs. Histopathology examination of the specimen showed fragments of fibro collagenous tissue and blood clot with no specific pathology detected with an impression of hematoma. She was discharged on post-operative day 5 with advice on regular physiotherapy and medications (oral antibiotics, oral analgesics and other supportive and nutritional supplements).

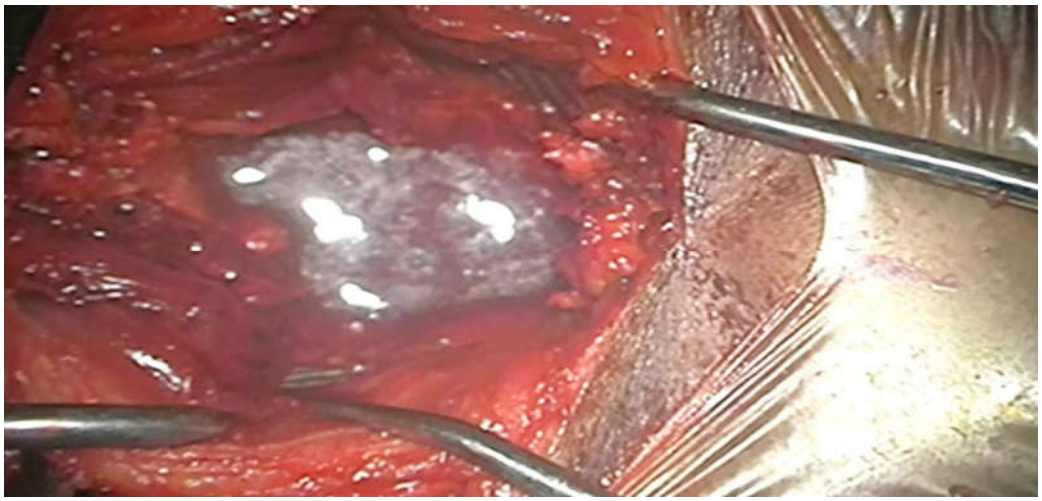


Fig. 5: Intraoperative Microscopic image of Hematoma after Cervical Laminectomy

DISCUSSION

Spinal epidural hematomas (SEH) are a rare and acute neurological condition in which the patient presents with acute severe pain at the location of haemorrhage, with radiation to extremities and rapidly develop progressive and severe neurologic deficit. [2] The etiology can be traumatic, spontaneous, iatrogenic following spinal surgery, epidural anaesthesia, lumbar puncture, any spinal arteriovenous malformation, spinal tumors and many more.[7] This was initially described in the literature by Jackson in 1869 and Bain in 1897.[6, 8, 9] Based on pathogenesis, SEH can be classified as secondary, spontaneous or idiopathic; and the term spontaneous spinal epidural hematoma is used to describe both idiopathic and spontaneous conditions.[6] Spontaneous Spinal Epidural Hematoma (SSEH) is defined as spinal epidural hematoma occurring without a traumatic or iatrogenic origin and represents 0.3% to 0.9% of all epidural space-occupying lesions with an annual incidence of 0.1/100,000 individuals. SSEH mostly affects male rather than females (1.5:1) and age group of 40 to 50 years. The frequency of the site of SSEH is thoracic, more than cervicothoracic or thoracolumbar region.[6,10,11,12,13] The incidence of posterior hematomas are more than the anterior hematomas due to the prominence of epidural venous plexus in the dorsal plane and the tight adherence of the posterior longitudinal ligament to the anterior dura .[6, 10, 11,12, 13]

The etiology of SSEH is unknown. Many precipitating factors have been reported in many literatures which includes, anticoagulant therapy[14], vascular malformation[15], bleeding disorders[16], cocaine abuse[17], hypertension, Valsalva manoeuvre[18], pregnancy.[6] A search of the relevant literature revealed reports of 37 cases of SSEH during pregnancy between 1966 and 2020, with the 1st case reported in 1966 by Bidzinski.[19] Hypothesis for the cause of SSEH is proposed to be venous bleeding taking into consideration the site of hematoma (posterior more than anterior), distribution of SSEH and the anatomical characteristics of the internal vertebral venous plexus. According to the hypothesis proposed by Bruyn and Bosma, any conditions which lead to increase in the intrathoracic/intraabdominal pressure which in turn increases the central venous pressure, can be transmitted to the valveless epidural veins, thus causing spontaneous hematomas.[20, 21] Enlargement of uterus during pregnancy leading to increased intra-abdominal pressure and compression on vena cava, resulting in the diversion of venous return and engorgement of extra dural venous plexus, along with the hemodynamic changes in pregnancy can predispose to SSEH in pregnancy.[6]

SSEH can present with a wide spectrum of symptoms, with the most common symptom being neck pain and / back pain with radicular component. The severity and progression of the disease may vary depending upon the site, amount of bleeding and degree of compression of the spinal cord. The symptoms may range from paraparesis, quadriparesis, hemiparesis to complete quadriplegia along with varying sensory and bowel and bladder involvement.[21] There are also case reports of SSEH presenting with features of spinal shock requiring emergency decompression.[22]

In patients with typical signs and symptoms, coagulation profile and appropriate imaging has to be done. The imaging modality of choice is MRI as it provides exact location, extend, age and features of cord compression or edema. MRI is the safe choice in pregnant patients to avoid radiation exposure to fetus.[6] The hematoma appears isointense to spinal cord on T1-weighted images and hyperintense on T2 weighted images taken within 24hrs of symptom onset. After 24hrs, the hematoma appears hyperintense on both T1 and T2 weighted images. Chronic hematomas become hypointense on both T1 and T2 weighted images.[1,18] On contrast enhanced MRI, enhancement may be seen in the hematoma due to hyperaemia in the dura mater and thickening of the meninges.[6,10]

The treatment of choice is surgical decompression of hematoma. But treatment may vary depending upon the extent of neurological deficits, any progress of symptoms and the time interval from onset of presentation. Conservative management is limited to those with mild or no neurological deficit and without progression of symptoms in the first 24 hours. The conservative management is also tired in patients with high risk for surgery, medically high-risk patients on anti-coagulations and in patients with a small non-compressive hematoma. [6, 10] There are increasing number of reports of individual cases managed conservatively with complete recovery. But this result cannot be extrapolated to all cases of SSEH. Conservative treatment usually leads to poor outcome, in cases where hematoma compresses the spinal cord.[21] There are also case reports where patients after recovery experienced deterioration requiring surgery.[23] Conservative treatment protocol should involve close observation of the patient in a neurosurgical unit with early repeat MRI.[21]

Conservative management would include immobilization, steroids, the treatment of coagulopathy, and percutaneous needle aspiration if symptoms progress or even if the recovery is not rapid.[6, 12]

The treatment of choice for SSEH is hemilaminectomy or a laminectomy followed by irrigation and debridement.[1,24,18] There are many factors that affect the postoperative neurological recovery, which includes the level and severity of preoperative neurological deficit and operative interval. The outcomes are much favourable in incomplete deficits as compared to complete deficits and in patients with some degree of intact sensations as compared to lack of sensory sparing .[21,25,26] Complete sensorimotor involvement, rapidly progressing symptoms, and cervical or thoracic hemorrhage are found to have a very poor prognostic value.[6,16] Studies showed that surgical decompression within 36-48 hours in patients with complete and incomplete deficits, respectively had high chances of complete recovery.[25,21,24] Though the most important prognostic indicator being the neurological status prior to the active intervention.[26,27]

SSEH in pregnancy must be treated according to the gestational age. In full term pregnancy, delivering the fetus by caesarean section in the normal supine position followed by surgical decompression in the prone position is done.[6, 10, 12 16,28] This increases the space in the abdomen and reduces the compression on the inferior vena cava. Tocolytics are recommended perioperatively to reduce the uterine contractions.[6,28] In patients with immature or nonviable fetus (less than 24 weeks of gestation), conservative management is preferred, with regular neurological monitoring, steroids for fetal lung protection and cord protection and an advice on early delivery.[6,16,30] Vaginal delivery is not preferred as uterine contraction further increase the intra-abdominal pressure and cause extension of hemorrhage. In early gestation, evacuation of the hematoma can be done.[6]

CONCLUSION

SSEH despite being a neurosurgical emergency, low incidence leads to the delay in the diagnosis of the condition and thus affects the final outcome of the cases. Urgent decompressive surgery is crucial in the prognosis of the patient's condition. Pregnancy further complicates the management of SSEH, and decision of delivery of fetus followed by surgical decompression or conservative management depends on the gestational age of the pregnancy. We hereby submit this case of spontaneous cervical epidural hematoma in term pregnancy, highlighting the investigations and management.

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