

# Spontaneous Spinal Epidural Haematoma During Pregnancy

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## Abstract

Spontaneous spinal epidural haematoma (SSEH) is rare in pregnancy and only five cases have been reported. We present a case of a 25-year-old female G<sub>1</sub>P<sub>0</sub> (gravida one, para zero) who at 31 weeks of gestation developed progressive descending quadriplegia and numbness upto the level of C4. MRI of the spine was performed, which demonstrated a cervical epidural evolving hematoma/abscess extending from C3 to C7 vertebrae. For which spinal decompression with spinal laminectomy was performed with removal of an epidural haematoma. The patient's neurological function subsequently improved. The diagnosis and management of spontaneous epidural haematoma in pregnancy is presented with a review of the literature.

## Introduction

Spontaneous spinal epidural haematoma (SSEH) is uncommon. To our knowledge there are only five reports of such haemorrhage occurring during pregnancy. We describe a patient who developed SSEH at 31 weeks of gestation and required surgical treatment. The diagnosis and management of this condition in pregnancy is presented, with a review of the literature.

## Case Report

A 25-year-old G<sub>1</sub>P<sub>0</sub> (gravida one, para zero) female, presented at 31 weeks of pregnancy with acute flaccid quadriplegia. Patient presented with a sudden onset of a severe pain between her shoulder blades, radiating to neck and head. The patient then developed sudden weakness in both the upper limbs which rapidly progressed to involve both lower limb and numbness in both upper limb and lower limb within half an hour of onset. Examination revealed flaccid quadriplegia with sensory loss up to approximately C4 level. Patient developed urinary retention for which she was catheterized. She had constipation also. The rest of her physical examination was unremarkable. Laboratory investigations were normal. Platelets count was within normal limits, bleeding time and clotting time were normal. Her serum biochemistry – blood sugar fasting and post prandial, urea, creatinine, SGOT, SGPT, serum bilirubin, alkaline phosphatase were normal. Serum prothrombin time was 12.0 seconds and control was 12.0 seconds with INR 1.04. Antinuclear antibody test was negative, rheumatoid factor was negative, anti-double stranded DNA antibody was negative, HIV 1 and 2 was negative. Urine routine and microscopy was normal. Electrocardiogram was normal. MRI of the spine was performed, which demonstrated cervical epidural evolving hematoma/abscess extending from C3 to C7 vertebrae.

There was a 7 x 1.6 x 0.6 cm fluid intensity lesion with few hypointense foci on T2W images along the right posterolateral aspect of the cervical cord extending from the level of C3 vertebra to C7 vertebra. It extended for a minimal distance along the nerve roots in to the right neural foramina. It was causing displacement and compression of the cervical cord (maximum at C5 vertebral level). It showed rim enhancement on the post-contrast scan

consistent with epidural haematoma/abscess (figures 1 and 2).

The spinal cord was normal in signal intensity. The cervical vertebrae revealed normal marrow signal intensity. The intervertebral disc spaces were normal. There was no abnormal pre or paravertebral soft-tissue mass. There was compression of the adjacent spinal cord caused by haematoma but without obvious signal changes within the spinal cord.

The patient was subjected for spinal decompression for which spinal laminectomy from C3 to C6 vertebrae was performed which demonstrated an epidural haematoma. Spinal decompression was performed 30 hrs after the onset of symptoms. During operation there was no vascular malformation. Histopathological examination of the surgical specimen confirmed hematoma and did not reveal any neoplastic process or vascular malformation.

In follow-up at 15 days post-surgery, the patient had shown good recovery with no neurological deficit. Thirty days following surgery the patient delivered normal vaginal delivery which was uneventful. The baby was female child weighing 1.5 kg.

## Discussion

SSEH represents a rare spinal emergency, with a frequency of less than 1% of spinal space-occupying lesions.<sup>1</sup> Pregnancy related epidural haematoma is an even more rare entity and only five documented cases have been reported.<sup>3-5</sup> Two of these reports pre-date routine use of MRI.<sup>3</sup> The first symptom of SSEH is usually severe localized back pain, often with a radicular component. The onset of pain is occasionally related to minor straining such as defecation, lifting, coughing or sneezing, but in majority of cases the onset of pain is spontaneous. Signs of spinal cord and nerve root dysfunction appear rapidly and may progress to paraparesis or tetraparesis, depending on the level of the lesion. Cases of Brown-Sequard syndrome have been described.<sup>4</sup> In the lumbar spine, the epidural haematoma may mimic an acute disc herniation. There have been reports of concomitant lumbar disc herniation and epidural haematoma. The most important aetiologies of SSEH are clotting disorders, which may be acquired (anticoagulant therapy, haematological malignancies) or congenital (haemophilia). Vascular malformations are rarely responsible for SSEH (4% in a series of 158 cases and 6.5% in a series of 199 cases).<sup>2</sup> Other less common predisposing factors for SSEH include: systemic lupus erythematosus, ankylosing spondylitis, rheumatoid arthritis, Paget's disease, disc herniation and hypertension. In about

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Fig.1 : Sagittal T<sub>2</sub> weighted spinecho sequence [Repetition Time (TR)-4270/Echo Time (TE) 80] reveals posterior epidural lesion that is hyperintense to the cord. H – Head, P – Posterior.

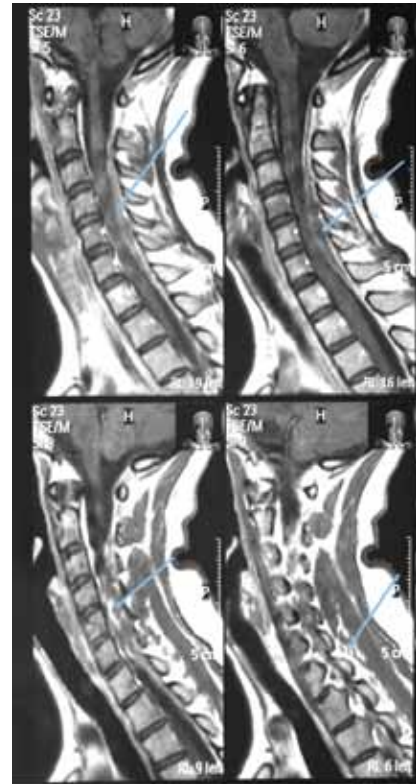


Fig.2 : Sagittal T<sub>1</sub> weighted image [Repetition Time (TR-500)/Echo Time (TE-10)] reveals posterior epidural lesion that is hyperintense to the cord. H – Head, P – Posterior

40–50% of cases, however, no underlying cause can be identified.<sup>2</sup>

The most widely accepted hypothesis is that of venous bleeding. Epidural veins are valveless and are situated in the low pressure epidural space. These veins are unprotected from sudden increases in intra-abdominal or intrathoracic pressure (as in the Valsalva manoeuvre), leading to rupture and haemorrhage.<sup>2</sup> It has been proposed that an increase in venous pressure in the epidural space, in association with the haemodynamic changes of pregnancy, may predispose to rupture of a preexisting pathological venous wall.<sup>3,5</sup> The epidural venous plexus is mostly prominent in the thoracic spine. The SSEH most often is located in the thoracic and cervicothoracic region followed by the thoracolumbar location and extends over a few vertebral body levels.<sup>1</sup> The SSEH is usually posterior or posterolateral to the thecal sac.<sup>1</sup>

Rapid diagnostic evaluation is essential to minimize delay in treatment of spinal epidural haematoma.

Currently, MRI is the diagnostic method of choice for spinal emergencies because it allows rapid, non-invasive evaluation of large parts of the vertebral column and the spinal cord in all planes.<sup>6</sup> MRI will provide information about location, extent of the haematoma, as well as the degree of cord compression. MRI is also helpful in determining the age of the haematoma.

The chronological characteristics of an MRI of a spinal epidural haematoma are similar to those seen with intracranial haemorrhage. In the hyperacute stage (first 6 h), the spinal epidural haematoma appears as isointense to the spinal cord on T1 weighted images, and mildly hyperintense and heterogeneous on T2 weighted images, as a result of the presence of intracellular oxyhaemoglobin. In an acute stage (7–72 h) the haematoma is still isointense on T1 weighted images, and becomes hypointense

on T2 weighted images, due to presence of intracellular deoxyhaemoglobin (which causes T2 shortening). As the concentration of methaemoglobin increases, the haematoma becomes hyperintense and homogeneous on T1 and T2 weighted images. A T2 weighted gradient-echo sequence, which shows hypointensity of the blood products, may be added to the standard MR sequences. MRI may show enlarged serpiginous areas of low signal corresponding to flow voids in abnormal vessels seen in vascular malformations, however those vessels may be compressed by the haematoma itself.

Spinal angiography is routinely performed in some centres when an epidural haematoma is found. Others reserve spinal angiography for cases in which MRI or myelography indicates presence of a vascular malformation.<sup>6</sup>

Conventional myelography, and later CT, used to be the main diagnostic modalities for diagnosing epidural haematomas. Myelography and CT myelography may show an epidural lesion with partial or complete spinal block but is not specific, invasive, and may worsen the clinical status. Conventional CT may diagnose an epidural haematoma, but may give false-negative results if the haematoma is isodense to the thecal sac or the spinal cord, and if the image quality is affected by artefacts (often seen in upper thoracic region).<sup>1</sup>

Urgent surgical decompression is the treatment of choice for SSEH causing acute compromise of cord function. There have been a number of reports in which SSEH was treated nonoperatively with good outcome, mainly haematomas localized at the cauda equina level and with mild neurological deficit. In the case of SSEH in full-term pregnancy, the fetus should be delivered first by cesarean section, with urgent spinal decompression to follow. Since venous pressure increases with

uterine contractions, the haemodynamic changes of labour may result in further haemorrhage.<sup>5</sup> The critical factors for recovery after SSEH are the level of pre-operative neurological deficit and the operative interval.<sup>6</sup> In complete pre-operative sensorimotor loss, surgery within 36 h of onset of symptoms correlates with favourable outcome.<sup>6</sup>

## Conclusion

Although SSEH is a rare cause of spinal cord compression, it is essential that the diagnosis is made as early as possible to enable full recovery. MRI plays an especially important diagnostic role. Surgery needs to be performed as rapidly as possible, because the interval between onset of symptoms and surgery, together with the pre-operative clinical status, determine the clinical outcome.

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