Spontaneous Spinal Epidural Hematoma During Pregnancy

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ABSTRACT: Background: Spontaneous spinal epidural hematoma is a rare phenomenon that has no distinct etiology. Spontaneous spinal epidural hematoma (SSEH) during pregnancy is extremely rare. We present what we believe to be the fifth reported case of spontaneous spinal epidural hematoma associated with pregnancy in the English literature. Methods: A 31-year-old female presented with acute onset of paraplegia at 32 weeks of pregnancy. The patient had a T2 sensory level and complete paralysis of all lower extremity motor groups. Magnetic resonance imaging of the thoracic spine showed an acute epidural hematoma posterior to the thoracic spinal cord between the second and fourth thoracic vertebrae. Results: The patient was taken to the operating room where her child was delivered by cesarean section. She then underwent a posterior laminectomy and evacuation of a spinal epidural hematoma. Follow-up selective spinal angiography was negative for any vascular malformation. The patient gradually recovered lower extremity function and was independently ambulating at six month follow-up. Voluntary bowel and bladder function returned within four months but twice daily intermittent catheterization remained necessary for excessive post-void residual urine. Conclusions: Spontaneous spinal epidural hematoma in pregnancy is a rare phenomenon. It is postulated that elevated venous pressure associated with pregnancy may be a contributing factor. In the reported cases of SSEH in pregnancy most patients presented with acute symptoms, thoracic location and profound neurological deficits but, with prompt surgical treatment, generally had good long term recovery.
symptoms began as severe mid-thoracic back pain after urination. Over the next hour, the patient developed a progressive ascending thoracic sensory level accompanied by severe weakness in the lower extremities and loss of bowel and bladder function. The pregnancy had been uncomplicated with no history of hypertension. The patient had a previous uncomplicated pregnancy and delivery in the past. She was otherwise healthy and taking no medications other than a prenatal vitamin supplement. She had no significant family history and did not drink alcohol or smoke.

Examination

Motor examination at initial assessment, three hours after onset of symptoms, showed 0/5 strength in all muscle groups in the lower extremity. Reflexes in the lower extremities were absent and the plantar response was mute. Sensory examination for light touch and pin prick revealed a T2 sensory level. Proprioception was intact in the lower extremities. Her rectal tone and perianal sensation were absent. Upper extremity neurological examination was normal.

Investigations

Laboratory studies showed a platelet count of 205 $10^3$ cells/mm$^3$, partial thromboplastin time was 24 seconds and International Normalized Ratio was 1.0. Urgent magnetic resonance imaging (MRI) of the thoracic spine was performed and demonstrated an epidural mass from T2-T4 which occupied 50% of the spinal canal. Signal intensity on T1 and T2 images was consistent with acute epidural hematoma. (Figures 1 to 3)

Operation

Upon establishing the diagnosis, the patient was taken urgently to the operating room. Less than eight hours from the onset of her symptoms, under general anesthesia, a healthy 32 week child was delivered by caesarian section. The patient was then positioned prone and a T2-T4 laminectomy was performed. An acute epidural hematoma was evacuated with good spinal cord decompression.

Postoperative Investigations

The histopathological analysis revealed only fragments of clotted blood with some fibrovascular tissue but no specific evidence of vascular malformation. Spinal angiography was performed and was normal.

Postoperative course

No motor activity in the lower extremities was present for the first six post operative days. On the seventh post operative day she was able to move her toes and ankles with grade 2/5 strength of plantarflexion and dorsiflexion. By the eleventh day she developed active hip flexion. She was transferred to a rehabilitation facility three weeks postoperatively and had 3/5 hip flexion, 4+/5 ankle plantarflexion and dorsiflexion. At the time of discharge from the rehabilitation ward, two months postoperatively, she had grade 4/5 strength in most lower extremity muscle groups and was ambulating 50 feet with a wheeled walker. At four month follow-up she was voiding independently but self-catheterizing twice daily due to post-void residual urine. By six months, the patient was able to ambulate independently.

DISCUSSION

Spontaneous spinal epidural hematoma associated with pregnancy are rare. Only four previous cases have been described in the literature since 1966 and are summarized in Table 1. The etiology in most cases remains unknown and may be multifactorial. However, it has been suggested that increased venous pressure associated with pregnancy may be an underlying factor. This has been emphasized by other authors who have hypothesized that venous bleeding is the major cause of spontaneous epidural hematoma formation. As the uterus enlarges during the third trimester, these changes are maximal and may explain the increased frequency of SSEH during the final stages of pregnancy. Furthermore, as pregnancy progresses constipation may occur due to alterations in gut motility and pressure from the enlarging uterus. This may require pregnant females to strain
more often to move their bowels. Prolonged valsalva maneuvers have previously been described as a contributing factor in the development of SSEH.

After reviewing the literature, we believe that pregnant women in the third trimester are more likely to develop more severe neurological deficits associated with SSEH formation. The four patients in the literature presented at 28, 35, 37 and 38 weeks gestation. Patients present with relatively increased venous pressure and the tamponade effect that normally slows epidural bleeding would occur only after a greater volume of blood enters the spinal canal. The resultant spinal cord compression would be greater than in non pregnant patients developing a spinal epidural hematoma.

Including our case, four of five patients in the literature presented with acute onset of symptoms. All of these patients had rapid development of significant neurological deficits within several hours of symptom onset. All were found to have cord compression from the mass effect of an epidural hematoma. Steinmetz et al. described a case with a more subacute onset of symptoms that was not treated with emergent evacuation.

After reviewing a series of SSEH in non pregnant patients, Foo suggested a tourniquet affect from venous bleeding in the epidural space may be protective and allow for less urgency for surgical evacuation. We believe this may not apply to pregnant patients as vertebral venous pressure is elevated due the effects of pregnancy. This elevation may overcome any tamponade or “tourniquet” effect.

In all reported cases pain has been the initial presenting symptom. The location of pain has been predictive of the level of the hematoma. This unique feature should alert those caring for pregnant females as to the potential significance of acute axial pain during pregnancy. These patients should have a thorough neurological examination and be followed closely. If any neurologic abnormality develops, urgent referral to a spine surgeon is required. All patients should have a coagulation profile performed. An MRI should be urgently obtained to establish the diagnosis. Magnetic resonance angiography (MRA) may be performed preoperatively to assess for an arteriovenous malformation. Computerized tomography, myelography or plain x-ray imaging are less likely to be of diagnostic value.

Reviewing the literature and including our case, four of five patients presented with an upper to mid-thoracic epidural hematoma. In two previously reported cases and our case, the hematoma was posterior to the spinal cord. In one of these cases the hematoma was in the cervical spine. Two cases of thoracic hematoma were ventral to the spinal cord. It is currently unclear why there is a tendency towards formation in
the thoracic spine. In a review of 330 cases of SSEH in non-pregnant patients, Groen\cite{16} has suggested no correlation could be observed between the spinal level and postoperative outcome.

It has been described that rapid development of symptoms and severe neurological deficits at presentation are poor prognostic factors for recovery after spinal epidural hematoma formation,\cite{10, 17, 18} This is particularly true for lesions affecting the thoracic spinal cord due to smaller canal diameter and greater sensitivity to compressive lesions.\cite{18, 19} Ultimate outcome after spinal cord decompression has been shown to be related to the preoperative neurological deficit and the operative interval between onset of symptoms and decompression.\cite{16} The extent of the hematoma (number of spinal segments involved) does not appear to correlate with preoperative neurological deficit or with postoperative outcome.\cite{18} The patient described by Yonekawa et al\cite{15} had a worse outcome. This patient was noted to have a severe post operative deficit, a cervical hematoma at C4 to C6 and significant spinal cord edema at surgery.

Because of a paucity of literature on the subject, firm management guidelines cannot be established. The majority of cases in the literature presented with severe, acute neurological deficits.\cite{8, 13-15} In all cases of thoracic spinal epidural hematoma associated with pregnancy a good clinical outcome was seen with surgical treatment.\cite{8, 13-15} In one case from 1975, the outcome after cervical spinal epidural hematoma was poor.\cite{15} In patients with profound neurological deficits, aggressive surgical treatment is warranted as significant neurological recovery can be achieved with prompt decompression.\cite{2}

<table>
<thead>
<tr>
<th>Author</th>
<th>Year of publication</th>
<th>Initial Symptom</th>
<th>Weeks Gestation</th>
<th>Onset of symptoms</th>
<th>Location of hematoma</th>
<th>Neurological deficit</th>
<th>Recovery</th>
</tr>
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<tbody>
<tr>
<td>Bidzinski et al\cite{13}</td>
<td>1966</td>
<td>Pain</td>
<td>28 weeks</td>
<td>Acute</td>
<td>T2-T5</td>
<td>Complete</td>
<td>Complete</td>
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<tr>
<td>Yonekawa et al\cite{15}</td>
<td>1975</td>
<td>Pain</td>
<td>37 weeks</td>
<td>Acute</td>
<td>C4-C6</td>
<td>Profound</td>
<td>Poor</td>
</tr>
<tr>
<td>Carroll et al\cite{14}</td>
<td>1997</td>
<td>Pain</td>
<td>35 weeks</td>
<td>Acute</td>
<td>T6</td>
<td>Profound</td>
<td>Nearly complete</td>
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<tr>
<td>Steinmetz et al\cite{8}</td>
<td>2003</td>
<td>Pain</td>
<td>38 weeks</td>
<td>Subacute</td>
<td>T1-T2</td>
<td>Partial</td>
<td>Nearly complete</td>
</tr>
<tr>
<td>Kelly et al (Current study)</td>
<td>2005</td>
<td>Pain</td>
<td>32 weeks</td>
<td>Acute</td>
<td>T2-T4</td>
<td>Partial</td>
<td>Complete</td>
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**REFERENCES**


