Nine Cases of Nontraumatic Spinal Epidural Hematoma

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Abstract

Nontraumatic spinal epidural hematoma (NSEH) is a rare but potentially serious spinal disease, which progresses inexorably to a catastrophic neurological situation if left untreated. Nine patients (5 males and 4 females, mean age 59.8 years) with NSEH were admitted to our hospital between 1987 and 1997. Clinical data, imaging, and operative results of these patients were analyzed retrospectively. Multilaminectomy was performed to decompress the affected levels. Postoperative neurological evaluation revealed functional improvement in five patients and no significant change in four patients. The most important factors influencing postoperative recovery were the preoperative neurological condition of the patient and the interval from onset of deficit until the operation. Magnetic resonance imaging allows non-invasive and specific diagnosis of NSEH, except in the first 24 hours. Computed tomography myelography with sagittal reconstructions provides specific diagnosis in this period. Combination of these diagnostic imaging modalities allows early diagnosis. Surgical decompression within 24 hours of complete sensorimotor deficit or within 48 hours of incomplete sensorimotor deficit will achieve the optimum outcome.

Key words: spinal epidural hematoma, decompressive laminectomy, magnetic resonance imaging, myelography, neurological outcome

Introduction

Nontraumatic spinal epidural hematoma (NSEH) is a rare but serious disease. The incidence is estimated to be 0.1 patients per 100,000 patients per year. Since the first report of acute NSEH in 1869, about 336 cases have been reported. Magnetic resonance (MR) imaging has revolutionized the diagnosis of spinal cord disease because it allows visualization of soft tissues with a resolution and detail that no other method provides. The introduction of MR imaging as a spinal diagnostic method has increased the frequency of cases of NSEH from about two per year (total of 218 cases from 1869 to 1985) to approximately 10 cases per year after 1986 (total of 118 cases since 1986) (Fig. 1). No obvious cause or predisposing factor was detected in more than half of all cases. Here we review the clinical, imaging, and surgical findings in nine patients with NSEH.

Case Summary

Nine patients with NSEH, five males and four fe-

Fig. 1 Distribution of the cases of nontraumatic spinal epidural hematoma between 1869 and the present.

mals, aged from 42 to 84 years (mean 59.8 years) were admitted to our hospital between 1987 and 1997. Clinical data, imaging, and operative results were analyzed retrospectively. Patients with spinal vascular malformations or obvious coagulation disorders were not included in this study. Neurological examination was performed on admission, after sur-

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Table 1 Presenting symptoms, therapy, and clinical outcome in nine patients with nontraumatic spinal epidural hematoma (NSEH)

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age/Sex</th>
<th>Relevant prior diseases</th>
<th>Location and extent of NSEH</th>
<th>Symptoms and preoperative deficits</th>
<th>Duration of symptoms</th>
<th>MR imaging</th>
<th>Myelography</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>84/M</td>
<td>arterial hypertension</td>
<td>T7-L1</td>
<td>progressive paraparesis, hypesthesia distal to T-6, loss of sphincter control (bladder), radicular pain</td>
<td>24 hrs</td>
<td>&lt;24 hrs</td>
<td>iso</td>
<td>high incomplete block no improvement</td>
</tr>
<tr>
<td>2</td>
<td>46/M</td>
<td>—</td>
<td>T5-12</td>
<td>radicular pain, T-6 sensorimotor incomplete pain, T-7 sensorimotor complete, progressive paraparesis, hypesthesia distal to T-6</td>
<td>12 hrs</td>
<td>&lt;24 hrs</td>
<td>iso</td>
<td>high — cure</td>
</tr>
<tr>
<td>3</td>
<td>61/M</td>
<td>—</td>
<td>T6-L2</td>
<td>—</td>
<td>3 days</td>
<td>3 days</td>
<td>high</td>
<td>high no improvement</td>
</tr>
<tr>
<td>4</td>
<td>51/M</td>
<td>—</td>
<td>T11-L4</td>
<td>L-1 sensorimotor incomplete, loss of sphincter control (bladder)</td>
<td>4 days</td>
<td>4 days</td>
<td>high</td>
<td>high incomplete block no improvement</td>
</tr>
<tr>
<td>5</td>
<td>73/M</td>
<td>coronary heart disease, asprin</td>
<td>T4-L2</td>
<td>back pain, increased paraplegia, T-5 sensorimotor complete, loss of sphincter control (bladder)</td>
<td>48 hrs</td>
<td>&lt;24 hrs</td>
<td>iso</td>
<td>high complete block proximal block L-2 minimal improvement, died 3 mos later</td>
</tr>
<tr>
<td>6</td>
<td>68/F</td>
<td>asthma bronchiale</td>
<td>T9-L3</td>
<td>T-10 sensorimotor incomplete, urinary incontinence</td>
<td>18 hrs</td>
<td>&lt;24 hrs</td>
<td>iso</td>
<td>high complete block cure</td>
</tr>
<tr>
<td>7</td>
<td>54/F</td>
<td>—</td>
<td>T12-L2</td>
<td>lower back pain, L-1 sensorimotor complete, loss of sphincter control (bladder)</td>
<td>6 days</td>
<td>&lt;24 hrs</td>
<td>iso</td>
<td>high complete block slight improvement</td>
</tr>
<tr>
<td>8</td>
<td>42/F</td>
<td>—</td>
<td>T3-6</td>
<td>T-5 sensorimotor complete, radicular pain</td>
<td>31 hrs</td>
<td>29 hrs</td>
<td>high</td>
<td>high complete block improvement</td>
</tr>
<tr>
<td>9</td>
<td>49/F</td>
<td>arterial hypertension</td>
<td>T11-L4</td>
<td>T-12 sensory complete, L-1 motor complete, loss of sphincter control (bladder), radicular pain</td>
<td>8 days</td>
<td>&lt;24 hrs</td>
<td>iso</td>
<td>high complete block no improvement</td>
</tr>
</tbody>
</table>

MR: magnetic resonance.
gery, and at discharge. The clinical symptoms, localization, distribution of hematoma, clinical outcome, and other features are listed in Table 1.

NSEH was located dorsally to the spinal cord in all patients. Hematoma had occurred spontaneously without obvious etiology, although hematoma might have been caused by long-duration aspirin therapy in Case 5. There was no recollection of trauma or other factors predisposing to bleeding. Intense local back pain was the first clinical sign in seven patients. Shortly thereafter, five patients felt radicular irradiation of pain, which occurred before the loss of spinal cord function. Two patients (Cases 4 and 6) experienced neurological deficit without previous back pain. The pain was mostly diffuse over a larger part of the spine and seldom localized over the affected segments. The most frequently encountered neurological symptom was progressive sensorimotor deficit (in all patients), which developed over a period of 12 hours to 8 days (mean 71 hours). Urinary retention or loss of sphincter control was also seen in six patients.

All patients underwent MR imaging, within 24 hours (Cases 1, 2, 5–7, and 9), within 48 hours (Case 8), and on the 3rd (Case 3) or 4th (Case 4) days after the onset of symptoms. Computed tomography (CT) myelography was performed using the lumbar puncture and ascending technique in seven patients and using the lateral cervical puncture (level C-1/C-2) in two patients. There was complete block in five patients and incomplete block in two (Table 1).

Multilaminectomy was performed to decompress the spinal cord at the most affected level, identified by MR imaging or myelography. Two patients recovered completely, three remained partly, and four were totally paralyzed. MR imaging was performed within 24 hours after the onset of symptoms in Cases 5, 7, and 9 at another hospital and showed an isointense mass in the dorsal epidural space, which was only recognizable with difficulty and initially interpreted as normal in Cases 7 and 9. Consequently, Cases 7 and 9 were initially conservative.

Fig. 2 Case 5. Magnetic resonance image showing the isointense appearance of hematoma 18 hours after bleeding. Cervical myelogram showing complete block at the T-4 level. Lumbar myelogram showing complete block at the L-2 level.
ly treated, but the symptoms progressed. The patients were admitted and treated surgically after performing CT myelography in the chronic stage 6 and 8 days after the onset. The clinical outcome was poor (no improvement in Case 9 and only slightly sensory improvement in Case 7). The lower thoracic and thoracolumbar crossing segments were mostly affected.

**Illustrative Case**

A 73-year-old male (Case 5) was admitted to our neurosurgical department because of insidious development of rapidly progressing back pain, gait disturbance, and distal numbness in the lower extremities over a period of 48 hours. His history was unremarkable except for coronary heart disease, for which he had been receiving regular treatment including aspirin for 2 years. There was no obvious history of trauma.

Neurological examination revealed flaccid paraplegia. There was an absolute loss of sensation to pinprick at T-8, with a partial loss of sensation at the T-5 to T-8 levels. Patellar and Achilles reflexes were absent bilaterally, plantar responses were absent, and the anal sphincter was flaccid. T1-weighted MR imaging 14 hours before admission at another hospital had demonstrated an epidural mass dorsal to the dura, appearing as an isointense signal. CT myelography demonstrated a complete block from proximal at the superior border of T-4 to distal at the superior border of L-2 (Fig. 2). CT clearly showed the epidural mass causing complete compression of the subarachnoid space and the spinal cord from dorsal-

![Myelo-CT](image)

**Fig. 3** Case 5. Computed tomography myelogram showing an epidural mass causing complete compression of the subarachnoid space and the spinal cord from dorsally.

Emergency laminectomy at the T6–9 levels was performed 28 hours after onset of complete paralysis. A large acute partially coagulated epidural hematoma was removed from the spinal canal dorsal to the dura. The dura was seen to pulsate at the end of decompression.

Postoperatively, the patient noted a slow return of flicker movements in the plantar flexor muscles of the left ankle. The right leg remained plegic. The sensory level remained unchanged. He remained incontinent with a flaccid sphincter. He suffered acute gastrointestinal bleeding and died 3 months after the spinal decompression procedure.

**Discussion**

NSEH is a rare but potentially serious central nervous system disease, which left untreated leads to severe irreversible neurological deficits. It is a neurological and neurosurgical emergency and prompt diagnosis is imperative. The hematoma can expand rapidly and extend over many spinal segments, causing sudden spinal cord and/or cauda equina compression. NSEH includes all forms of extradural spinal hemorrhages not consequent to vertebral trauma, coagulation disorders, vertebral angiomas, or any other apparent cause. NSEH occurs in all age groups, with a higher incidence after the age of 50. The NSEH due to bleeding from vascular malformation occurs in less than 3% of cases. Clinical signs are typically sudden dorsal pain, with radicular radiation in over 50% of cases, followed by progressive sensory or motor deficits, usually within hours or days, which may progress to complete paralysis, reviewed. Postoperative recovery was affected by the level of the hematoma, the preoperative neurological condition of the patient, and the interval between onset of symptoms and surgical decompression. No obvious cause or predisposing factor was identified in more than half of the cases. The correct diagnosis was mostly too late for successful surgical treatment, which explains the poor prognosis of this disease. Rapid surgical decompression can reverse neurological deficits or halt progression, so prompt recognition of this disease can affect outcome favorably.

The source of bleeding in NSEH is still controversial, but may involve the extensive epidural venous
plexus or the small epidural arteries. A sudden increase of venous epidural pressure in the posterior internal vertebral venous plexus caused by Valsalva maneuver may be important in the formation of NSEH. There seems to be no relationship between NSEH and arterial hypertension. The involvement of vascular anomaly in the etiology of the NSEH seems to be overestimated, as the cluster-type dural artery can easily be mistaken for a vascular anomaly. The hematoma was located dorsally to the spinal cord and occurred spontaneously in all our patients, although the hematoma in Case 5 might have been caused by aspirin medication. Only two previous reports have observed an association between NSEH and aspirin. This association is difficult to judge, but aspirin has hemorrhheological effects and causes increased tendency to bleeding during and several days after administration, so a link to NSEH is possible.

MR imaging is the diagnostic choice in the subacute and chronic stages of NSEH because of its pathognomonically MR signal changes. However, the MR imaging signals changes of hematomas are isointense and confused in the first 24 hours of NSEH. CT myelography was helpful in this stage as in previous reports. In general, laminectomy or multilevel laminectomy over the most affected segments is the treatment of choice especially if the spinal symptoms are severe or progressive. Successfully conservative therapy has been reported in many cases, when the neurological condition of the patient remains stable and allows conservative nonsurgical treatment and/or when the patient’s general condition is so poor as to contraindicate surgical procedure.

Postoperatively, two patients recovered completely, three remained partly, and four were totally paralyzed. Two patients (Cases 7 and 9) were admitted and operated too late, so their clinical outcome was poor. The best postoperative recovery is achieved when surgical decompression is performed within 24 hours in patients with complete sensorimotor deficits and within 48 hours in patients with incomplete sensorimotor deficits.

References

Prior to the common use of magnetic resonance imaging, a diagnosis of spinal epidural hematoma was made by myelography with or without CT scanning. Typically the patient presented with an insidious onset of pain which rapidly progressed to weakness, usually paraplegia. Clearly by far the majority of the lesions were in the thoracic spine in our patient population. While it could occur with anticoagulation medicines such as Coumadin, that was fairly rare. Some of the patients clearly were on aspirin or NSAIDs. Some of our younger patients were vigorously exercising. One of our more dramatic case presentations has been people doing hyperextension abdominal exercises. This is where the patient lies on a table, with their feet and legs held down, and they do an exercise referred to as sit-ups or crunches where the body or thorax hyperextends and they bring their entire body up to the flexed, sitting position.

With the rather sudden and severe pain in most patients, I have been of the opinion that these are initiated by an arterial bleed. However, I have to admit I have no evidence to disprove the venous theory as a possible etiology. (The venous pressure usually is not that much dissimilar to cerebrospinal fluid pressure so it seems more plausible that an arterial vessel breaks to cause this disease.)

With the advent of magnetic resonance imaging, we know how much of a laminectomy will be needed. Prior to that, we used to do a myelogram and start removing bone until all of the clot was removed. Now with our newer diagnostic studies, we know exactly what the problem is and how far we need to do a decompression.

Trauma still remains an important etiology in this disorder. We have had several cases where senior citizens have had fractures in the limbs and rather minor fractures in the spinal column. In the fracture treatment of their limbs, once immobilized and often plated or pinned, they were placed on anticoagulants only for them to suffer a spinal epidural hematoma. This can turn an unimportant spinal fracture into a major spinal disaster, for removing the posterior elements removes some of the stabilizing bony elements. Fortunately, that sequence remains rare.

All of our data indicates that the factors influencing recovery necessitates rapid decompression within the first 24 hours in an incomplete lesion. This article, along with all of the articles quoted in the reference list, point to the same conclusion. Treatment of this disease, unless the patient is neurologically okay, is of considerable urgency if not an emergency.
sive review of the literature. As the authors pointed out, spinal epidural hematoma can occur without significant injury to the spine, causing serious neurological deficits like paraplegia or quadriplegia. Magnetic resonance imaging of high quality is the procedure of choice in making definite diagnosis of this devastating condition. Unfortunately, patients with sudden onset of back pain followed by progressive paraplegia are not always referred in time to a medical center where spinal neurosurgeons are available. Therefore, it is important for us to educate medical students and primary physicians that this relatively rare situation needs emergent surgical decompression in order to obtain reasonable recovery from the serious neurological deficits.

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