Spontaneous Spinal Epidural Haematomas

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Summary
The spontaneous spinal epidural haematoma (SSEH) is a rarity, but the severe and permanent motor disability underlines its importance.

From 1957 seven cases of SSEH have been diagnosed and operated on in the National Institute of Neurosurgery, Budapest. These cases are analysed and discussed.

The clinical picture began with local pain of the spine and radicular signs but some hours or days later paraparesis or paraplegia and incontinence developed. In the discussed cases the neurological deficit progressed to complete para- or tetraplegia in 5 cases. Only 2 patients had partial spinal transverse lesions on admission. All patients underwent myelography to detect the spinal space occupying lesion and were operated on soon. Three patients recovered completely, 2 remained partly and 2 totally paralysed.

The outcome depended mainly on the timing of neurological deficiency. If the neurological signs existed less than 8 hours the patients recovered completely or fairly well while the prognosis was poor if the transverse lesion persisted longer than 24 hours.

The authors stress the importance of correct and fast decisions at the first medical examination for the outcome of this disease, because only immediate transfer to a neurosurgical department gives a chance of good recovery.

Keywords: Spontaneous; spinal epidural haematoma.

Introduction
Spontaneous spinal epidural haematomas are rare: about 270 cases have been reported in the literature since Jackson's first report in 1869. Nonetheless, the clinical importance of this problem is underlined by the severity of the neurological deficit which can be avoided by adequate diagnosis and urgent surgical intervention. In spite of the rather typical clinical picture and modern neuro-imaging techniques, the correct diagnosis is usually too late for successful surgical treatment.

Material

From 1957 seven cases of SSEH have been diagnosed and operated on in the National Institute of Neurosurgery, Budapest.

There were 5 males and 2 females, the mean age was 36 years, varying from 12 to 57. Details of the clinical data are summarized in Tables 1 and 2.

Table 1. Clinical Data I

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Anticoag. treatment</th>
<th>Prothrombin time (Quick)</th>
<th>Other disease</th>
<th>First symptom</th>
<th>Radicular pain</th>
<th>Timing of radicular pain to OP (hours)</th>
<th>Other signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>m</td>
<td>54</td>
<td>cumarin</td>
<td>89%</td>
<td>-</td>
<td>lumbar pain</td>
<td>+</td>
<td>70</td>
<td>fever</td>
</tr>
<tr>
<td>m</td>
<td>22</td>
<td>92%</td>
<td>98%</td>
<td>-</td>
<td>neck pain</td>
<td>+</td>
<td>24</td>
<td>fever</td>
</tr>
<tr>
<td>f</td>
<td>15</td>
<td>98%</td>
<td>88%</td>
<td>haemophilia</td>
<td>back pain</td>
<td>+</td>
<td>18 days</td>
<td>fever</td>
</tr>
<tr>
<td>f</td>
<td>38</td>
<td>41%</td>
<td>55%</td>
<td>myocard. inf.</td>
<td>left leg pain</td>
<td>+</td>
<td>51</td>
<td>-</td>
</tr>
<tr>
<td>m</td>
<td>12</td>
<td>98%</td>
<td>98%</td>
<td>neck pain</td>
<td>pain</td>
<td>+</td>
<td>6</td>
<td>-</td>
</tr>
<tr>
<td>m</td>
<td>57</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>pain</td>
<td>+</td>
<td>98</td>
<td>-</td>
</tr>
<tr>
<td>m</td>
<td>56</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>pain</td>
<td>+</td>
<td>7</td>
<td>-</td>
</tr>
</tbody>
</table>
In all cases the first symptom was an intensive local pain of the spine at the level of the lesion. Three of them had pain with radicular radiation.

The intervals between the development of local pain and the neurological deficit were different. Usually within hours or days sensori-motor deficit and disturbance of sphincter control occurred. This period can be surprisingly long.

One of our patients, a fifteen year old schoolgirl (V. I.) experienced two weeks from the first local back pain to the neurological signs. During that period she had slight fever. Finally she was admitted to the paediatric department of a county hospital because of numbness and weakness of the lower limbs. Examination of her lumbar CSF showed 3.08 g/l protein. Two days later she became totally paralysed below the Th 3 level, but she was transferred to our institute, only three further days later, with the diagnosis of spinal tumour.

The neurological deficit progressed to complete para- or tetraplegia in 5 cases, 2 patients had partial spinal transverse lesions on admission. The development of neurological signs takes a different period of time ranging from half an hour to three days. In the cases of complete transverse lesions this time was shorter.

Myelography was performed immediately after admission and the neurological examination. The myelogram revealed complete extradural block in 5 cases, while in 2 of them the block was incomplete. The radiomorphology of the alteration was a typical extradural extramedullary contrast block in all of our cases.

The levels of the haematomas were cervical in three, thoracic in three and lumbar in one case. The localisation of haematomas in relation to the spinal cord was dorsal in 4 patients, in 3 cases dorsal and lateral.

After the diagnosis of a space occupying lesion laminectomy and evacuation of the haematoma were performed immediately.

In our material the time that passed from admission to operation was not longer than two hours in any of the cases. The interval from onset of neurological signs to operation ranged from 6 hours to 4 days.

Concerning the risk factors there was 1 patient treated with anticoagulants, due to myocardial infarction, while a twelve year old boy was a haemophiliac.

In two cases the further microscopical examination of the removed clot showed epidural angiomatous malformation which could be related to the haematoma, while in 5 there was no detectable source of the bleeding.

The postoperative resolution of neurological signs took 24 hours to 1 month. Three patients recovered completely, 2 remained partly and 2 totally paralysed.

**Discussion**

SSEH has a doubtful natural history and pathophysiology.

It is still debated whether the source of bleeding could be the extensive epidural venous plexus or the microarteries. A sudden increase of venous epidural pressure can play a role in the formation of SSEP while 3 of our patients had physical effort just before the onset of local pain.