Spontaneous Thoracic Spinal Cord Herniation
—Case Report—

Takahiko EGUCHI, Hiroshi YOKOTA, Yuji NIKAIDO, Misato NOBAYASHI, and Toshikazu NISHIOKA

Department of Neurosurgery, Osaka-Minami National Hospital, Kawachinagano, Osaka

Abstract

A 54-year-old female presented with spontaneous thoracic spinal cord herniation manifesting as chronic progressive motor weakness in both legs. Spastic paraparesis (4/5) and pathological reflexes such as ankle clonus were noted. She also had mild bladder dysfunction but no bowel dysfunction. She had no sensory disturbance, including tactile and pinprick sense. Magnetic resonance (MR) imaging revealed that the atrophic spinal cord was displaced into the ventral extradural space at the T4-5 intervertebral level with markedly dilated dorsal subarachnoid space. Computed tomography obtained after myelography showed no evidence of intradural spinal arachnoid cyst. She underwent surgical repair of the spinal cord herniation via laminectomy, and spinal cord herniation through the ventral dural defect was confirmed. Postoperative MR imaging revealed improvement of the spinal cord herniation, but her symptoms were not improved. Spontaneous spinal cord herniation is a rare cause of chronic myelopathy, occurring in the upper and mid-thoracic levels, and the spinal cord is usually herniated into the ventral extradural space. Early differential diagnosis from intradural spinal arachnoid cysts is important for a satisfactory outcome.

Key words: spinal cord, herniation, thoracic spine, arachnoid cyst

Introduction

Spinal cord herniation is a pathological condition in which the spinal cord is displaced out of the dura through a dural defect, and is classified as spontaneous, iatrogenic, and traumatic according to the cause of the dural defect.5) Spontaneous spinal cord herniation is extremely rare, with only 21 cases confirmed by surgery.1,3–14) We treated a patient with spontaneous herniation of the mid-thoracic spinal cord through a dural defect manifesting as chronic spastic paraparesis.

Case Report

A 49-year-old female with paraparesis presented with motor weakness in her left leg beginning 10 years previously. She had been healthy with no history of previous trauma. The motor weakness had gradually spread to both legs over the following 8 years. The paraparesis had slowly become worse over the last 2 years. She was referred to several clinics, but no cause was identified, despite magnetic resonance (MR) imaging of her thoracic and lumbar spine. She could not walk up stairs in March 1998. She was admitted to our hospital on April 10.

The patient had mild spastic paraparesis (4/5), predominantly on the left. Exaggerated knee and ankle jerks and ankle clonus were also noted, but Babinski reflex was absent. Her bilateral quadriceps muscles were atrophic. She had mild bladder dysfunction but no bowel dysfunction. She had no sensory disturbance, including tactile and pinprick sense. Vertebral radiography showed no abnormalities. MR imaging demonstrated the displacement of the spinal cord outside the dura at the T4-5 intervertebral level (Fig. 1). Myelography and computed tomography (CT) immediately after myelography revealed that the severely atrophic cord was displaced ventrally and to the left at the T4-5 intervertebral level (Fig. 2). The dorsal subarachnoid space at the same level was markedly dilated, but the absence of filling defect or retention of contrast medium excluded subarachnoid cystic lesions, although...
delayed CT was obtained 6 hours after myelography. Laminectomies of the T4-5 levels were performed with the patient prone and under general anesthesia. The dura and arachnoid were opened in the midline. The atrophic spinal cord was displaced anteriorly after cutting the dentate ligament. No abnormal findings suggesting arachnoid cyst or arachnoiditis were noted. Careful retraction of the spinal cord revealed that the anterior part of the spinal cord was herniated through the dural defect (Fig. 3). The adhesion between the spinal cord and the opening of dural defect was mild, so the spinal cord was released from the dural opening by intradural blunt dissection, and part of the herniated cord was reduced intradurally. The size of dural defect was 7 mm in the transverse direction and 20 mm in the longitudinal direction. Extradural dissection revealed no other abnormalities, such as dural duplica-
Fig. 3 Intraoperative photograph showing that the spinal cord (SC) is displaced extradurally through the ventral dural defect (thick arrow), but no evidence of other disorders, such as intradural arachnoid cyst or dural duplication. Arrow indicates herniated tissue. Left side of the photo shows caudal direction, and right side rostral direction.

Fig. 4 Postoperative magnetic resonance images showing improvement of the displacement of the spinal cord. left: Sagittal T1-weighted image, right: sagittal T2-weighted image.

tion and extradural arachnoid cyst, except the dural defect. The dural defect was repaired using a GoreTex patch and the dorsal dural incision was closed with running sutures.

The paraparesis mildly deteriorated after the operation, although postoperative MR imaging revealed improvement of the spinal cord herniation (Fig. 4). Her paraparesis had gradually improved, but then deteriorated over the 3 months after surgery. Secondary arachnoid cysts due to arachnoiditis were noted during follow-up MR imaging. The patient underwent a second surgery for the dissection of arachnoiditis and wide dural plasty, but the paraparesis did not improve, and she is confined to a wheelchair.

Discussion

The 22 cases of spontaneous spinal cord herniation are described in Table 1. The age at diagnosis was 36 to 71 years (mean 50.8 years) in nine males and 13 females. The lesions were located between the T-2 and T7-8 intervertebral levels, with no lesions outside the upper and mid-thoracic region. The directions of the herniations were ventral (usually ventrolateral) in 20 cases and lateral in the other two cases. The dural abnormalities consisted of dural defects in 20 cases and dural out-pouches in two cases. Dural duplication was associated with the dural defect in four cases.

Various hypotheses for the pathogenesis of spinal cord herniations have been proposed. The consensus is that a preexisting dural defect or dural out-pouch is necessary for the development of spinal cord herniation.1,4,5,10,11,13 Two conditions are necessary for the spinal cord to herniate through the dura: the dural defect leading to an extradural arachnoid cyst and the defect situated on the concave side of the spinal curvature (ventrally in the thoracic spine).5 Such physiological kyphosis at the mid-thoracic level may be important for the pathogenesis of spontaneous cord herniation.1,5,10,11 Physiological pulsatile cerebrospinal fluid movement may gradually push out the cord after it adheres to the aperture of the dural defect.1,5,10,11 The dorsal intradural arachnoid cyst may push out the cord through the ventral dural defect,4 but most cases including ours were not associated with dorsal intradural arachnoid cysts.1,3,7,8,11,12 The dorsal arachnoid cysts in all these cases may be secondary.10 There was no evidence of ventral epidural arachnoid cyst in our case nor in some previous cases.1,3,7,8,10,11 Therefore, there is some doubt about the necessity for the preexisting ventral extradural arachnoid cyst. Cases of spontaneous intracranial hypotension have recently been reported, caused by ruptured spinal extradural meningeal cysts, but without association with spinal cord herniation.3 Moreover, symptoms suggesting intracranial hypotension have not been described in cases of spontaneous spinal cord herniation.1,3,14

All cases of spontaneous spinal cord herniation presented with gradually progressing myelopathy, persisting for a period of 0.8 to 12 years (mean 5.0
### Table 1  Summary of reported cases

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Age/Sex</th>
<th>Level</th>
<th>Location</th>
<th>Type of myelopathy</th>
<th>First presenting</th>
<th>Suffering period (yrs)</th>
<th>Surgical procedure</th>
<th>Result</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wortzman et al. (1974)</td>
<td>63/M</td>
<td>T-7</td>
<td>ventral</td>
<td>Brown-Séquard</td>
<td>sensory</td>
<td>2</td>
<td>repair/biopsy*</td>
<td>improved</td>
<td></td>
</tr>
<tr>
<td>Masuzawa et al. (1981)</td>
<td>36/M</td>
<td>T4-5</td>
<td>lateral</td>
<td>Brown-Séquard</td>
<td>motor</td>
<td>0.8</td>
<td>repair</td>
<td>excellent</td>
<td>DO, EA</td>
</tr>
<tr>
<td>Oe et al. (1990)</td>
<td>61/M</td>
<td>T-4</td>
<td>ventral</td>
<td>transverse</td>
<td>motor</td>
<td>10</td>
<td>repair</td>
<td>unchanged</td>
<td>IA, Dup</td>
</tr>
<tr>
<td>Isu et al. (1991)</td>
<td>43/F</td>
<td>T5-6</td>
<td>ventral</td>
<td>sensory dissociation</td>
<td>sensory</td>
<td>1</td>
<td>unrepair</td>
<td>improved</td>
<td>EA, IA</td>
</tr>
<tr>
<td>Tronnier et al. (1991)</td>
<td>45/F</td>
<td>T2-3</td>
<td>ventral</td>
<td>sensory dissociation</td>
<td>motor</td>
<td>1.7</td>
<td>unrepair</td>
<td>unchanged</td>
<td>EA, IA</td>
</tr>
<tr>
<td>Nakazawa et al. (1993)</td>
<td>43/F</td>
<td>T-2</td>
<td>ventral</td>
<td>Brown-Séquard</td>
<td>sensory</td>
<td>5</td>
<td>repair</td>
<td>excellent</td>
<td>Dup</td>
</tr>
<tr>
<td>Kumar et al. (1995)</td>
<td>39/F</td>
<td>T4-5</td>
<td>ventral</td>
<td>Brown-Séquard</td>
<td>sensory</td>
<td>3</td>
<td>repair</td>
<td>excellent</td>
<td>Dup</td>
</tr>
<tr>
<td>Borges et al. (1995)</td>
<td>38/M</td>
<td>T7-8</td>
<td>ventral</td>
<td>Brown-Séquard</td>
<td>sensory</td>
<td>2</td>
<td>repair/biopsy*</td>
<td>excellent</td>
<td>EA</td>
</tr>
<tr>
<td>Sioutos et al. (1996)</td>
<td>68/F</td>
<td>T-7</td>
<td>ventral</td>
<td>Brown-Séquard</td>
<td>sensory</td>
<td>12</td>
<td>repair*</td>
<td>excellent</td>
<td></td>
</tr>
<tr>
<td>Miura et al. (1996)</td>
<td>48/F</td>
<td>T-7</td>
<td>ventral</td>
<td>Brown-Séquard</td>
<td>sensory</td>
<td>8</td>
<td>repair</td>
<td>improved</td>
<td></td>
</tr>
<tr>
<td>Hausmann and Moseley (1996)</td>
<td>57/F</td>
<td>T-6</td>
<td>ventral</td>
<td>Brown-Séquard</td>
<td>sensory</td>
<td>8</td>
<td>repair</td>
<td>improved</td>
<td></td>
</tr>
<tr>
<td>Takahashi et al. (1997)</td>
<td>57/M</td>
<td>T-7</td>
<td>ventral</td>
<td>Brown-Séquard</td>
<td>motor</td>
<td>0.8</td>
<td>repair</td>
<td>unchanged</td>
<td></td>
</tr>
<tr>
<td>Uchino et al. (1997)</td>
<td>61/F</td>
<td>T-7</td>
<td>ventral</td>
<td>Brown-Séquard</td>
<td>motor</td>
<td>2</td>
<td>repair</td>
<td>unchanged</td>
<td>IA, EA</td>
</tr>
<tr>
<td>Present case</td>
<td>54/F</td>
<td>T4-5</td>
<td>ventral</td>
<td>paraparesis</td>
<td>motor</td>
<td>10</td>
<td>repair</td>
<td>unchanged</td>
<td></td>
</tr>
</tbody>
</table>


years). Twelve cases initially presented with sensory disorder including pain or sensory dissociation and 10 cases presented with unilateral motor weakness in the leg. Most cases presented as Brown-Séquard type myelopathy at the time of diagnosis. These clinical features are in agreement with the presence of ventrolateral dural defects in which the anterior funiculus was herniated first. In our case, no sensory dysfunction was noted at diagnosis. The size of dural defect in our case may be larger than that of previous cases. We believe that the bilateral lateral funiculi herniated at the early stage. The 7 mm transverse diameter of the dural defect is the largest described.

The clinical symptoms of spinal cord herniation are progressive, unless surgically repaired. The posterior approach was selected in 19 of the 22 cases. The transthoracic approach was selected in the other three cases, under a preoperative misdiagnosis of thoracic disc herniation. In five cases, the repair of the dural herniation was unsuccessful, and in three cases the herniated tissue was resected as biopsy. Only 13 patients had favorable surgical result, and the others had unsatisfactory surgical outcomes. In our case, secondary arachnoiditis was important in the deterioration of the symptoms, as in a previous case.11) Cases which were not repaired resulted in poor surgical outcome as expected, and cases which presented as motor weakness, particularly over longer periods, also had unfavorable surgical outcomes. We emphasize that early treatment results in a good surgical outcome, so diagnosis of spontaneous cord herniation in the early stage of herniation is important. At the present time, non-invasive MR imaging can clearly demonstrate extradural spinal cord herniation, but only in the advanced stage. MR imaging obtained in the early stage of this disorder may reveal a strong resemblance to the findings of dorsal intradural arachnoid cysts, which are more familiar.
to us. Differential diagnosis with intradural arachnoid cysts can be based on a sharp notch in sagittal MR imaging of the dorsal spinal cord outline because of the focal kinking of the cord in cases of herniation, whereas dorsal intradural arachnoid cysts consist of smooth margins.11) We agree that this case is progressive, but may not be in an early stage. We suggest that dorsal subarachnoid space dilation on the thoracic level with cord displacement close to the anterior dura on MR imaging of patients presenting myelopathy, particularly with sensory dissociation, indicates spinal cord herniation. We should not exclude transdural herniation of the spinal cord in the presence of a dorsal intradural arachnoid cyst, because spinal cord herniation may be associated with secondary dorsal arachnoid cyst.

References


Address reprint requests to: H. Yokota, M.D., Department of Neurosurgery, Nara Medical University, 840 Shijo-cho, Kashihara, Nara 634-8522, Japan.