Low-pressure headaches and spinal cord herniation

Case report

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Almost 40 cases of spontaneous transdural spinal cord herniation have been reported in the literature. These patients often present with gait disturbance and sensory changes, and their condition is diagnosed as Brown–Séquard syndrome. The pathogenesis of this condition has remained poorly understood. In particular, there is no agreement whether the dural defect is acquired or congenital. In the reported case, a 21-year-old man presented with a 3-year history of intermittent low-pressure headaches consistent with intracranial hypotension. Eventually, the headaches resolved but he developed myelopathy due to a spinal cord herniation. In this case, the authors hypothesize that the progressive spinal cord herniation through a spontaneous dural tear sealed the site of the cerebrospinal fluid leak, causing the resolution of headaches.

KEY WORDS • spinal cord herniation • cerebrospinal leak • headache • dural repair
In most cases, the spinal cord is anteriorly displaced at the level of herniation, typically between T-2 and T-8, and has an almost pathognomonic appearance on sagittal MR imaging. Axial MR imaging and CT myelography demonstrate the anterolaterally displaced spinal cord at the level of herniation. Ewald, et al., have reported a case in which the patient became symptomatic before the abnormality could be demonstrated on MR images. In such a case, they recommend CT myelography as the most sensitive method for early detection. The most common misdiagnoses are intradural posterior arachnoid cysts. Cine MR imaging is helpful in excluding these cysts.

Although the pathogenesis is unclear, various theories exist. Isu, et al., have postulated that a posterior intradural arachnoid cyst may displace the spinal cord anteriorly. We found no such structure during this operation, nor during our two other unreported cases, as mentioned by other authors. In this present case, it is possible that the spinal cord occluded the initial dural defect and that CSF pulsations caused additional cord herniation through the defect. Most spinal cord herniations occur in the thoracic spine because of its normal kyphosis and the cord’s physiological anteroposterior movement secondary to cardiac and pulmonary actions. This is also a common location for spontaneous dural tears presenting with intracranial hypotension. The clinical course in our case supports this theory.

The cause of the dural defect is not known. Several authors have implicated an inner layer defect of the duplicated anterior dura as the cause. In our case, the defect had clear dural margins through which the cord and arachnoid had herniated. The margins were smooth and regular. After reducing the herniation, the epidural space and posterior longitudinal ligament were clearly visible. Several authors have similarly described finding the hernia sac as an “epidural arachnoid cyst” or as “flimsy fibrous tissue.” We hypothesize that initially there may be a small rent or weak section of dura. Because of CSF and spinal cord pulsations, the small rent enlarges. In this case, the arachnoid tore leading to CSF extravasation and position-related headaches. Eventually, the cord herniated through the defect (Fig. 1). This stopped the CSF extravasation and related headaches but led to spinal cord compromise and myelopathy.

Clinically, most cases present with a Brown–Séquard syndrome. Our patient also suffered from position-related headaches. Masuzawa, et al., reported a patient with a history of frequent headache. To our knowledge, this is the first reported case in which there was a definite history of position-related headaches. After surgery, our patient experienced significant recovery of gait, bladder, and sexual function. Repeated MR imaging 3 months postoperatively revealed anatomical reduction of the spinal cord. Surgical reduction promises a high possibility of recovery and prevention of further deterioration. The herniated spinal cord needs to be reduced using meticulous microsurgical techniques. The surgeon then should repair the dural defect primarily or using a dural graft. Watanabe, et al., recommended simple enlargement of the inner dural defect and reported similar results as those achieved after a primary repair.

References

Fig. 1. Sagittal (left) and axial (right) MR images revealing the site of the spinal cord herniation (arrows).
Spinal cord herniation and headache


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