

## Spontaneous Thoracic Spinal Cord Herniation through an Anterior Dural Defect

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**Summary:** A 44-year-old woman was examined for progressive left lower extremity weakness and spasticity. Thoracic spine MR imaging and CT myelography showed a ventral dural defect at T7-T8 with an extradural subarachnoid fluid collection and extradural herniation of the spinal cord. Intraoperative sonography confirmed the appropriate level for dural entry and the finding of spinal cord herniation. After reduction of the herniated spinal cord, the patient experienced gradual improvement in neurologic function.

Extradural herniation of the thoracic spinal cord is an unusual cause of progressive myelopathy. Only 17 such cases have been reported in the literature (1-13). A preoperative diagnosis can be made with knowledge of the characteristic imaging appearance. Progression of the patient's neurologic deficits can be prevented and most neurologic function recovered after surgical reduction of the herniated spinal cord. We describe a case of thoracic spinal cord herniation through an anterior dural defect and illustrate the characteristic findings at MR imaging, CT myelography, and intraoperative sonography.

### Case Report

A 44-year-old woman had a history of slowly progressive left lower extremity weakness and atrophy. She was unable to run because of leg weakness and perceived incoordination. She had no pain and no evidence of bowel or bladder dysfunction. She described the left leg as more sensitive to temperature and light touch than the right leg. Physical examination revealed lower extremity asymmetry (left mid-thigh circumference 4 cm less than the right, mid-calf circumference 3 cm less than the right). Neurologic examination was remarkable for decreased strength in the left lower extremity with motor function 4+/5 in the iliopsoas and quadriceps, 4/5 in the hamstrings, and 4/5 dorsiflexion and plantar flexion of the ankle. Deep tendon reflexes were exaggerated at the left knee and ankle and her left toe was upgoing. Sensory examination revealed mild right lower extremity hypesthesia and diminished temperature sensation with a soft level at the umbilicus.

The initial MR study (Fig 1A-C) showed focal atrophy of the spinal cord at the T7-T8 level with ventral apposition of the cord to the posterior vertebral body and disk. The study was interpreted as focal cord atrophy related to prior trauma, in-

farct, or demyelinating disease. At CT myelography, the spinal cord was again noted to be attenuated at the T7-T8 level and deviated ventrally and to the left. Delineation of the dura by intradural and extradural myelographic contrast enhancement of different densities allowed the detection of the spinal cord herniating through the dura ventrolaterally (Fig 1D). Because of continuing concern about the unusual diagnosis, a high-resolution MR imaging examination was performed that was consistent with spinal cord herniation (Fig 1E).

The original axial T2-weighted image showed a questionable epidural fluid collection above and below the level of cord herniation (Fig 1F). A CT myelogram confirmed that the extradural fluid collection was in communication with the subarachnoid space (Fig 1G and H). The difference in contrast density on the CT study indicated that there was not free communication of the subarachnoid space and the extradural fluid.

A posterior approach for surgical reduction of the herniated cord was elected to facilitate reduction of the cord from the dural defect with minimal soft-tissue and osseous injury. After bilateral laminectomy at the T7-T8 level and exposure of the intact dorsal dura, intraoperative sonography was used to confirm the appropriate level for dural entry (Fig 1I). The dorsal dura was opened and the dentate ligament incised on the left. Herniation of the ventrolateral aspect of the spinal cord through a ventral dural defect was identified. Adhesions between the margins of the dural defect and a ventral nerve rootlet tethered the cord to the dura. Following dissection of these adhesions, the cord herniation was reduced, as shown in the intraoperative photograph (Fig 1J). A schematic diagram summarizes the imaging and intraoperative findings (Fig 1K).

The 6 × 15-mm ventral dural defect was closed using a fascial autograft. The dorsal durotomy defect was closed primarily. The patient had an uneventful postoperative course, with gradual improvement in neurologic function and minimal residual weakness at the 6-month follow-up examination.

### Discussion

Although extradural herniation of the thoracic spinal cord is an unusual cause of progressive myelopathy, because the imaging features are characteristic, the radiologist should be able to suggest the diagnosis preoperatively to guide the neurosurgical approach. Spinal cord herniation in the cervical spine in the postoperative patient and in the lumbar spine in post-traumatic patients has been described as well, but the history and imaging are less problematic.

Received October 29, 1997; accepted after revision March 11, 1998.

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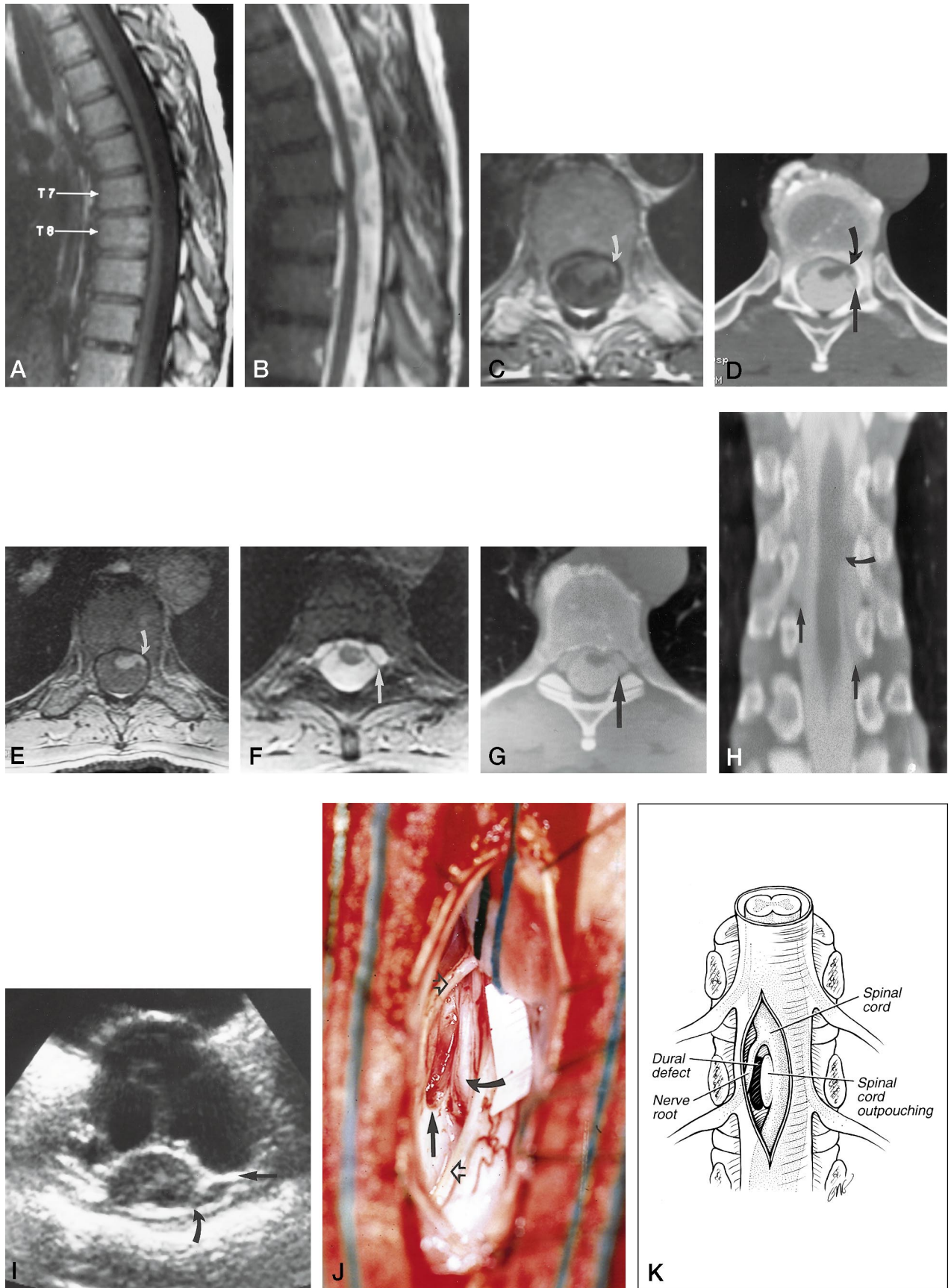


FIG 1. 44-year-old woman with slowly progressive weakness and atrophy of the left lower extremity.

- A, Initial sagittal T1-weighted MR image (500/14/2 [TR/TE/excitations]).
- B, Sagittal fast spin-echo T2-weighted MR image (3600/144/3) shows ventral displacement of the spinal cord and apparent mild cord atrophy.
- C, Initial axial T1-weighted MR image (900/18/2) shows anterior displacement of the cord with questionable thickening of the dura anteriorly, subsequently shown to be ventral cord herniation (*arrow*).
- D, Subsequent axial thoracic CT myelogram shows soft tissue extending from the anterior margin of the spinal cord through the dura (*straight arrow*). *Curved arrow* indicates cord herniation.
- E, Gradient-echo T1-weighted MR image (35/9/1) with a 20° flip angle and a 1.5-mm section thickness provides excellent delineation of the spinal cord herniation (*arrow*).
- F and G, Axial fast spin-echo T2-weighted MR image (43600/170/4) (F) and axial thoracic CT myelogram (G) immediately above the spinal cord herniation show a ventral, extradural fluid collection (extradural arachnoid cyst) (*arrow*).
- H, Coronal reformation shows the ventrolateral spinal cord herniation and the extradural fluid collection. *Straight arrows* indicate dural reflections delineating lower contrast in the extradural fluid collections from the intradural myelographic contrast; *curved arrow* indicates the inferior margin of spinal cord herniation as it traverses the dura.
- I, Intraoperative sonogram from a posterior approach depicts the spinal cord herniation, confirming the correct surgical level. (Note that right/left is reversed as compared with MR and CT studies, because of the posterior approach). *Straight arrow* denotes the highly reflective dura and the *curved arrow* indicates the spinal herniation.
- J, Intraoperative photograph from a posterior approach after reduction of the spinal cord herniation with the small dural defect (*solid straight arrow*) visible immediately above and below the nerve rootlets (*open arrows*) and lateral to the spinal cord outpouching (*curved arrow*).
- K, Schematic view through a dorsal durotomy of the spinal cord herniation after reduction from the dural defect.

The pathogenesis of thoracic cord herniation has been ascribed to a dural defect, either congenital or acquired, on the convex ventral surface of the thoracic spinal canal. The spinal cord is believed to “plug” the hole in the dura, and prolonged CSF pulsations force the cord through the dural defect. Nabors et al (14) proposed a classification of meningeal cysts, types I, II, and III to clarify and describe CSF fluid collections in the spine. Type III is an intradural arachnoid cyst (similar to intracranial arachnoid cysts), type II is a perineural cyst (including Tarlov cysts), and type I is an extradural CSF collection that communicates with the subarachnoid space, sometimes referred to as an extradural arachnoid cyst (although an arachnoid layer is frequently not found to line the fluid collection).

The extradural fluid collection associated with the spinal cord herniation described in this report and in the literature appears to be a type I cyst and probably represents a congenital defect in the dura. This dural defect allows CSF to enter the extradural space, creating a localized fluid collection sometimes referred to as an extradural arachnoid cyst. When the hole in the dura is covered by the closely applied spinal cord along the convex margin in the thoracic spine, the cord can be forced through the defect by prolonged CSF pulsations. Adhesions and distortion of the cord with possible vascular compromise over time leads to progressive myelopathy. This proposed mechanism explains the absence of idiopathic spinal cord herniation occurring in the cervical and lumbar spine and posteriorly in the thoracic spine, where the cord would not be so closely applied to a potential dural defect.

The clinical presentation in the literature of thoracic cord herniation is characteristic but nonspecific (1–13). Patients’ ages in the published cases range from 22 to 68 years, with a mean age of 43 years. A majority of the herniations described presented with a Brown-Séquard syndrome (ipsilateral hemiparesis and loss of proprioception and vibratory sensation and contralateral loss of pain and temperature sensa-

tion). A minority presented with paraparesis or bowel and bladder dysfunction. Slowly progressive deficits occurred in most patients. The female to male ratio was 1.4:1. Symptoms and cord herniation were lateral to the right in seven, lateral to the left in six, and not described in four.

The appearance of thoracic cord herniation in the literature is both characteristic and specific when adequate imaging is performed. Cord herniation was between T2 and T8 in all cases, with focal ventral deviation or atrophy of the cord over a length of 1 to 3 cm. Most reports describe an arachnoid cyst, either intradural or extradural, associated with the cord herniation. Surgical reduction of the herniated cord and primary repair of the ventral dural defect are recommended. The surgical defect in the dorsal dura may require a dural graft to provide a larger dural space, as the reduced spinal cord may be swollen and edematous. In the majority of cases reported, the patients’ neurologic deficits resolved or improved (1–9). In some cases in which symptoms persisted or worsened, the spinal cord herniation was not reduced or the dural defect not repaired (10–13).

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