Several cases of cervical cord compression during flexion have been reported. \(^3,5-10\) The characteristic radiological feature of this flexion myelopathy is a normal appearance on plain x-ray films and myelograms of the cervical spine in the neutral position, whereas myelography in the flexed position reveals cord compression. We describe a similar case involving the thoracic spine.

**Case Report**

This 36-year-old man had developed bilateral hypalgesia of the feet in December 1991. In April 1992, he noted the gradual onset of a gait disturbance and was admitted to our department in June 1992.

Examination. General physical examination on admission was unremarkable, but neurological examination revealed a spastic gait. Deep-tendon reflexes were normal in both upper extremities, and patellar and ankle jerks were hyperactive. Pinprick and touch sensation was diminished below the T-7 dermatome. Vibratory sensation was diminished in both lower extremities. Cranial nerve and cerebellar function was within normal limits. Plain thoracic x-ray films revealed no abnormal findings such as a narrow disc space, spur formation, a narrow spinal canal, excessive kyphosis, or subluxation. Thoracic magnetic resonance (MR) images were normal. Myelographic studies obtained in the neutral position revealed a normal spinal cord and dural sac. Computerized tomography (CT) after myelography showed anterior atrophy of the spinal cord on the right side, which was severe at the T3–4 level and mild at the upper and midthoracic vertebral levels. The spinal cord and dural sac had shifted slightly to the right at the T3–4 level. The right anterior subarachnoid space was slightly narrowed; however, the left and posterior subarachnoid space was of normal size.

Myelographic studies were performed in the flexed position to determine the cause of the cord atrophy. Myelography in the anteroposterior projection revealed extreme narrowing of the subarachnoid space from T3–6 vertebral levels, and the lateral view showed an almost complete block of the dye column between T3–4 and T8–9. It appeared that the spinal cord had been compressed between the posterior margin of the vertebral body and the posterior component of the dura (Fig. 1). After myelography, CT scans in flexion showed extreme reduction of the subarachnoid space between the T3–4 and T6–7 intervertebral levels, resulting in widening of the posterior extradural space (Fig. 2). Flexion MR imaging revealed an anterior shift of the cord and dural sac. Low-intensity areas in the enlarged epidural space indicated dilation of the epidural venous plexus (Fig. 3).

After CT examination of the thorax in the flexed position, the patient’s neurological condition worsened with spontaneous clonus of the legs. Spasticity increased and his sensory level rose from the T-7 to the T-3 dermatome. However, he was kept lying in the extended position, and returned to his previous neurological condition the next day.
Operation. On July 16, 1992, the patient underwent stabilization of the thoracic spine in the neutral position from T1–12 with posterior fusion and Cotrel-Dubousset instrumentation to avoid flexion movement. Postoperatively, his condition gradually improved. In March 1994, his neurological status was normal except for slightly hyperactive jerks of the legs. The extensive thoracic spine fusion causes him some limitation in his movements but he can work as a machine design engineer.

Discussion

Many authors\textsuperscript{1,2,4} have pointed out that an excessive degree of kyphosis or subluxation of the thoracic spine can compress the cord and cause myelopathy. In our case, there was no kyphosis, disc disease, or vertebral instability. Myelography and postmyelographic CT with the patient in the neutral position failed to reveal any lesion compressing the cord. However, myelography of the thoracic spine in the flexed position revealed blockage of the dye column and compression of the cord from T3–9. Postmyelographic CT in flexion showed marked anterior displacement of the cord and the dural sac, which resulted in widening of the posterior epidural space, and flexion MR imaging disclosed dilated epidural veins in the enlarged posterior epidural space.

Several authors\textsuperscript{3,5–10} have reported cervical flexion myelopathy. The clinical features of this condition are progressive weakness and muscular atrophy in the hands and forearms without definite sensory disturbances. Plain x-ray films of the cervical spine were normal in both flexion and extension. Myelography in the extended cervical spine failed to reveal any lesion compressing the cord; however, myelography in flexion revealed marked anterior displacement of the tense spinal cord and dural sac, which were compressed over the smooth convexity of the vertebral column by their own tension. Contact pressure

![Fig. 1. Left and Center: Myelograms in flexion showing severe compression of the subarachnoid space from T-3–6 (left) and cord compression from the T3–4 to T8–9 intervertebral level (center). Right: Myelogram in the neutral position showing no evidence of the cord compression.](image1)

![Fig. 2. Computerized tomography scans after myelography. Upper: Scans in the neutral position showing normal patency of the subarachnoid space around the atrophied spinal cord. Lower: Scans in flexion showing complete stenosis of the subarachnoid space from the T3–4 to T6–7 intervertebral disc spaces.](image2)
on the spinal cord, aggravated by axial tension due to the flexion, was considered to be the etiological mechanism of this myelopathy. Following vertebral fusion, good results were obtained.

The signs and symptoms of our patient suggested a transverse myelopathy different from cases of cervical flexion myelopathy. However, because of radiological findings similar to those in the cervical spine, this case was considered to be flexion myelopathy of the thoracic spine. In cases of localized anterior atrophy of the cord with thoracic myelopathy of unknown cause, myelographic examination in flexion is recommended.

References


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