Cervical flexion myelopathy: a “tight dural canal mechanism”

Case report

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The authors describe a case of flexion myelopathy due to specific morphological changes of the cervical cord, termed a “tight dural canal mechanism.” The patient was an 18-year-old man with progressive weakness and muscle atrophy of the left arm. Neuroradiological examination revealed that the lower cervical cord was compressed during flexion of the cervical spine, but that there was no disc disease or cervical vertebral instability. The cord compression was attributed to a pinching mechanism by the posterior border of the vertebral body and the posterior component of the dura on flexion.

KEY WORDS • flexion myelopathy • myelography • tight dural canal

Several authors have reported morphological changes in the spinal cord related to the onset of symptoms in cervical disc disease, spinal canal stenosis, or a combination of these disorders. Intramedullary changes due to cervical disc disease have also recently been described. However, most of these reports deal with morphological or pathological changes of the cervical cord associated with extension, and little has been presented about the changes that take place on flexion. We have recently treated a case of impairment of the lower cervical cord related to flexion of the neck, resulting in neurological symptoms. There was no evidence of cervical disc disease, excessive kyphosis, or subluxation of the vertebral body.

A previous case of specific morphological changes on the patient assuming a flexed position was reported in 1983 by Mii. We describe a similar case with a different pathogenesis.

Case Report

This 18-year-old male student had first developed weakness of the left arm at the age of 16 years. Weakness and atrophy gradually progressed over the ensuing 2 years, and he was admitted to our department in June, 1984.

Examination. General physical examination on admission was unremarkable. Neurological examination disclosed muscle atrophy and weakness involving the left dorsal and palmar interossei, the thenar and the hypothenar, the extensor and flexor digitorum communis, and the extensor and flexor carpi radialis and ulnaris muscles. Fasciculations were observed in the same muscle groups, predominantly in the dorsal interossei muscles. Irregular, fine, and tremulous movements were present in the fingers of the left hand when the patient attempted to extend them; this was probably due to muscle weakness and atrophy. Deep-tendon reflexes were normal in both upper extremities, and the patellar and ankle jerks were slightly hyperactive. The pinprick and touch sensation was slightly diminished in the distribution of the C6–T1 dermatome of the left arm, but temperature, position, and vibration sensation were normal. No muscle weakness or atrophy was observed in the right arm or either leg. Cranial nerve and cerebellar function were within normal limits.

Plain cervical x-ray films revealed no abnormal findings such as narrow disc space, spur formation, a narrow spinal canal, excessive kyphosis, or subluxation. However, a mild degree of scoliosis was demonstrated. Myelographic studies in the neutral and extension positions were normal except for mild atrophy of the lower cervical cord on the lateral view. Lateral myelographic studies in the flexed position, however, revealed that
the entire dural canal was shifted to the front of the spinal canal between C4-5 and C-7 and the posterior epidural space was widened (Fig. 1). As a consequence, it appeared as if the spinal cord had been pinched between the posterior margin of the vertebral body and the posterior component of the dura.

Computerized tomography (CT) after myelography showed slight atrophy of the cord at the level of the lower cervical vertebrae, more severe on the left; however, the cord was still sufficiently thick, and the subarachnoid space was fully open. The CT scans showed that the spinal cord was compressed forward to the front of the spinal canal and became flattened from the C4-5 to C-7 vertebral level on flexion, more profoundly on the left (Fig. 2). Delayed CT 6 hours after intrathecal injection of contrast medium revealed a high-density area within the cord due to the inflow of the contrast medium at the C5-7 vertebral level, a region that appeared to correspond to the anterior horn of the left side of the cord. Furthermore, enhanced CT studies showed a mild enhanced effect in the posterior epidural space which had been enlarged on flexion (Fig. 3). This seemed to indicate the presence of an epidural venous plexus in the enlarged epidural space. Based on these findings, we intended to perform an anterior fusion of the lower cervical vertebrae; however, the patient refused surgery. No progression of his neurological symptoms has been noted during the 2-year follow-up period.

Discussion

Many authors have pointed out that, on flexion of the neck, the cord can be pressed hard against the posterior aspect of the vertebral body by an excessive degree of kyphosis of the cervical vertebrae or by an abnormal alignment such as severe subluxation.2,3,6,8,9 Recently, Miً3 reported a case of flexion myelopathy caused by differential growth of the length of the cervical vertebral column and the spinal cord (the cervical cord was shorter than the cervical vertebral column). This disorder was termed an "overstretching mechanism." In that case, the primary evidence of such an imbalance was a myelographic visualization of the shadow of the root sleeve which was lifted toward the head on flexion. In the present case, such a finding was not obtained.

The radiological findings in our case may be explained by the following factors: 1) there was no attachment between the dura and the yellow ligament associated with fibrous trabeculation, thus the posterior component of the dura freely shifted forward on flexion; 2) the elasticity of the dura per se was decreased by some unknown cause; and 3) the relative shrinkage.
Cervical flexion myelopathy

of the dural canal had occurred as a result of uneven growth of the spine and dura. As the vertical distance of the posterior part of the cervical canal is normally extended on flexion, the posterior component of the dural canal is likely to become tense, shift forward, and press on the cord. In these circumstances, it is understandable that the physiological curving in flexion of the cervical vertebrae is most marked at the C-5 and C-6 levels; therefore, the pressure is most severe at these levels. Among the factors mentioned above, it is likely that the first factor is acquired as a result of the second or third factors, or is congenital in nature due to abnormal development of epidural venous plexuses.

We have tentatively referred to the mechanism of pressure on the spinal cord caused by such conditions of the dura as the “tight dural canal mechanism on cervical flexion” and have termed the neurological symptoms due to such conditions “flexion myelopathy.” The major neurological signs and symptoms in this case (namely, weakness and atrophy of the left forearm and hand muscles with fasciculation) can be attributed to the impairment of the left anterior horn of the cord, demonstrated by the collection of contrast medium on delayed CT myelography. The causes for flexion myelopathy vary widely and include such conditions as subluxation, excessive kyphosis, and overstretching mechanism, but a tight dural canal mechanism should be considered as another possible mechanism for impairment.

References


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