Cervical myelopathy due to spondylosis

Case report

JOHN C. HAWKINS, III, M.D., F. YAGHMAI, M.D.,
AND R. ARTHUR GINDIN, M.D.
Departments of Pathology (Neuropathology) and Surgery (Neurosurgery), Medical College of Georgia, and the Veterans Administration Hospital, Augusta, Georgia

The authors report the case of a patient with cervical myelopathy who was examined at autopsy 2 years after a second anterior cervical fusion by Cloward's technique. The clinical course and pre- and postoperative myelograms are presented. Theories as to the etiology of myelopathy are discussed. This case demonstrates chronic changes that seem to implicate a vascular theory but not the specific vessel or vessels. The mechanism of improvement following the Cloward procedure is not explained by the pathological slides.

KEY WORDS • cervical spinal cord injury • ischemic myelopathy • spinal cord compression • spondylotic myelopathy

Previous autopsy reports of spondylotic cord lesions have been limited to patients who had acute trauma and died in the immediate postoperative period from air embolism, or were nonoperative cases. We are presenting a patient with myelopathy who required anterior cervical fusion at two levels using the Cloward technique. He died 2 years after the second operation from an unrelated cause. The changes seen in the cord did not exactly conform to those said to be indicative of a vascular etiology or to those described with compression injury.

Case Report

This man, in 1968, at the age of 44 years, noted some weakness in his legs. In July, 1969, he began having difficulty initiating micturition. In December, 1969, he had further weakness in his legs and began using a cane when he walked. In February, 1970, he had nocturnal urinary incontinence and shooting pains in his extremities. On March 2, 1970, while attempting to go down the steps of his porch to the ground, a distance of approximately 2 feet, he landed on his knees. After this accident, he complained of low-back pain and increased weakness in his extremities.

First Admission. On admission on March 3, 1970, his strength was 3/5 bilaterally in the upper and lower limbs. He had marked hyperreflexia, 4+ bilaterally, with knee and ankle clonus and Hoffmann and Babinski signs on both sides. He had decreased vibration sense to the iliac crests and decreased position sense in the lower extremities. After some difficulty initiating voiding, he voided 150 cc and was found to have a residual of 60 cc of urine. On March 10, 1970, a myelogram (Fig. 1) demonstrated restriction of the canal from C-3 to C-7 with marked narrowing by an anterior bar at C3-4, but no defect...
J. C. Hawkins, III, F. Yaghmai and R. A. Gindin

posteriorly. The cerebrospinal fluid (CSF) protein was 185 mg% with no cells. On March 30, 1970, a Cloward procedure was performed at C3–4. At operation approximately 3 mm of herniated disc was found behind the posterior longitudinal ligament impinging on the dura, to a greater extent on the left than the right. The patient’s postoperative course was uncomplicated. By April 22, 1970, he could walk with the use of a walker and had little trouble initiating micturition. He could do finger-nose-finger and heel-to-shin tests but did demonstrate spasticity.

On June 6, 1970, the patient had continued numbness of the arms and legs. He denied difficulty with urinary or bowel control or impotence. He still walked with the aid of a walker. No fasciculations were seen. Motor strength was 4/5 (good) bilaterally in the upper and lower extremities.

Second Admission. On October 10, 1970, the patient again presented with the complaint of inability to support his body weight. A myelogram (Fig. 2) revealed a bar at C5–6, much more prominent than the previous study had indicated. The CSF protein was 190 mg% and there were no cells. On November 2, 1970, a Cloward procedure was performed at C5–6. Bilateral spurs were resected. The postoperative course was without complications. The patient left the hospital on November 25, 1970, using crutches as a walking aid; he had persistent hyperreflexia and spasticity.

Third Admission. On January 25, 1971, the patient stated that he could not walk. Examination revealed 4/5 strength in the lower limbs. He had good rectal tone, no residual urine, sustained ankle clonus, and loss of position and vibratory sense. The CSF protein was 110 mg% with no cells. A myelogram demonstrated absence of the previous bars (Fig. 3).

The patient was discharged on February 19, 1971, after intensive physical therapy. He could walk with the help of a cane.

On April 2, 1971, the patient was seen in the clinic. He had a broad-based, shuffling

Fig. 1. Preoperative myelogram. Left: Anteroposterior view demonstrating multiple defects. Upper Right: Lateral view illustrating the narrowness of the spinal canal and the largest defect at C3–4. Patient in slight extension. Lower Right: Patient supine in neutral position. No defect is seen from C-2 to C-6.
Cervical myelopathy due to spondylosis

Patient had only gross movements of the hands, and could not feed himself. Strength overall remained 4/5 in both upper and lower extremities. Pinprick sensation was intact.

Fourth Admission. On October 10, 1972, the patient was admitted with a fever of 104°F. A lumbar puncture revealed a protein of 96 mg% and no cells. He was treated for bacterial endocarditis, but died on October 13, 1972, despite attempts at resuscitation.

Postmortem Examination. The spinal cord was removed via an anterior approach and care was taken to prevent introducing ar-
artifacts. It was fixed in 10% buffered formalin and later cut in multiple transverse sections. Paraffin sections of hematoxylin and eosin, luxol fast blue, Bodian, and trichrome stains, and also frozen sections stained for fat were studied. The posterior half of the cord at C-4 through C-7 showed a long-standing cystic cavitating lesion with gliosis similar to the lesion of an old infarction. This resulted in collapse of the cord and decreased anteroposterior diameter. Sparse foamy macrophages were still seen dispersed through the lesion. The lesion had extended slightly anteriorly to involve the base of the anterior horns, otherwise the cells of the anterior horns were normal in morphology and population at all levels. Secondary tract degeneration was seen in the corticospinal tracts below the C-7 level, and in the posterior columns above C-3. The degeneration was noticed in axons and their myelin sheaths with resulting gliosis. There were no corticospinal tract changes proximal to the main lesion (Fig. 4). Moderate leptomeningeal fibrosis (C3-8) and recent microinfarction (C-8) were also noted. No discernible changes in vessels, including the anterior spinal artery, or hemosiderin pigments within or without macrophages were seen.

Discussion

In the following discussion, we compare the pathological changes seen in the spinal cord in our case with those reported by others in cases of cervical spondylosis examined at autopsy.

In 1952, Bedford, et al., reported a case of cervical myelopathy. The topography of the lesion was “not consistent with a vascular lesion,” according to the authors. The lesion of the cord was noted in both gray and white matter with diffuse loss of nerve cells and gliosis. There was marked flattening, but relatively little demyelination was noted at the points of flattening. The pia mater was believed to be thicker and more cellular than normal, and there was prominent demyelination in the lateral columns and in the anterior portion of the posterior columns.

Mair and Druckman, in 1953, reported four cases of myelopathy that presented changes which they ascribed to reduction of blood supply in the distal distribution of the anterior spinal artery. The injury primarily involved the anterior horns, the lateral columns, and the anterior part of the dorsal horns. The lesions were not of uniform severity. The less damaged portions of the
Cervical myelopathy due to spondylosis

Gray matter exhibited chromatolysis and neuronophagia, while the more severe lesions showed loss of nerve cells, gliosis, and even cavitation. The changes in the white matter ranged from swelling of the myelin and axons and microglial proliferation to destruction of myelin and axons and glial scar formation. Small cavities occurred also in the white matter as a result of tissue necrosis. The histological changes were similar to those of ischemia. The authors postulated compression of the anterior spinal artery on the surface and in its ramifications throughout the cord. The distal ramifications were the most affected since the compression force would exert a more marked obliterative effect upon vessels that are subjected to this force over a longer distance.

In 1960, Wilkinson reported several cases of cervical spondylosis and myelopathy. Among these were two cases of rather chronic myelopathy, one of insidious onset and the one seemingly related to two traumatic incidents. The descriptions of the cord are somewhat less detailed than the other cases. Crescentic deformation of the cord, and some degeneration in the posterior columns above the lesions and below in the lateral columns were mentioned. Wilkinson noted that several degenerative changes may lead to lesions in the cord and nerve roots. There was liability of the cord to damage as a result of injury to the spondyloïtic spine through interference of blood supply, but no thrombosis of the anterior spinal artery was found.

The case presented here was similar to that of Bedford, et al., in that no pattern of injury consistent with a single vessel etiology could be found, but marked infarction and tract degeneration was seen. Our case shows changes in the watershed area supplied by both the anterior and posterior spinal arteries, but also demonstrates severe lesions in the posterior columns.

The mechanism of the etiology of myelopathy secondary to cervical spondylosis is an issue much discussed but not clearly resolved. Taylor, in 1953, postulated that the ligamentum flavum bulged forward, causing compression of the cord between the thickened ligamentum and the osteophyte bar during extension of the neck. Later, he suggested that radicular artery occlusion caused the myelopathy. More recently, Breig, et al., Hukada and Wilson, and Turnbull have shown that changing from flexion to extension may cause changes in the antero-posterior and transverse diameter of the cord, causing stretching and coiling of the vascular supply and supposedly putting the anterior column and posterior columns out of the reach of these short penetrating vessels.

Our findings are consistent with chronic ischemia in this area. They do not support anterior spinal artery occlusion alone, nor acute minor trauma as the etiology for the myelopathy of cervical spondylosis.

Summary

This case demonstrates chronic changes in the cervical cord that seem to best fit a vascular theory, but do not implicate the exact vessel or vessels. The mechanism of improvement following the Cloward procedure is also not demonstrated.

Acknowledgments

I wish to thank Ms. Jackie Walton who typed the manuscript and Mr. Thomas Lanier who prepared the illustrations.

References


Address reprint requests to: R. Arthur Gindin, M.D., Section of Neurosurgery, Medical College of Georgia, Augusta, Georgia 30902.