Cervical myeloradiculopathy caused by arthrotic hypertrophy of the posterior facets and laminae

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Five patients with typical cervical myeloradiculopathy caused by focal cervical spinal stenosis are presented. Dorsal intrusions into the spinal canal by hypertrophied apophyseal joints and thickened laminae resulted in cord and nerve root compression. Minor spondylotic changes were present in the floor of the spinal canal. Laminar decompression with foraminotomy and facetectomy relieved the patients of their symptoms. An anterior approach should not be considered in the management of this disorder. Our findings of severe apophyseal arthrosis with lesser degrees of associated spondylosis are similar to those described in anatomical studies by other authors. While uncommon, myelopathy caused by dorsal compression of the spinal cord and nerve roots deserves specific mention so that therapy can be directed to the proper quadrants of the spinal canal wherein the significant pathology is located.

KEY WORDS • cervical myeloradiculopathy • stenosis • apophyseal arthrosis • dorsal compression • cervical spine

CERVICAL spondylotic myeloradiculopathy has invariably been related to cervical stenosis wherein all available space has been compromised by osteophytes arising from the vertebral bodies and the uncinate processes. Little attention has been directed toward similar effects caused by dorsal intrusions into the spinal canal and foramina by apophyseal arthritic changes except for a single brief case report. Five additional cases observed during the past 2 years indicate that an awareness of the effects of arthrosis is mandatory in the surgical management of the myelopathy usually attributed to spondylosis.

With our present myelographic methods of studying patients with cervical myelopathy, conventional anteroposterior exposures cannot differentiate between dorsal and ventral intrusions. Only lateral flexion and extension examination with a full column of oil can identify clinically important changes in the dorsal quadrants of the cervical spinal canal that relate to cord compression and radiculopathy.

Little investigative effort had been expended in evaluating the effects of cervical apophyseal arthrosis on neural compression until Holt and Yates, in a cadaver study, showed injury to nerve roots and ganglia.
FIG. 1. Case 1. Left: Anteroposterior view of the cervical spine shows advanced apophyseal arthrosis at C3-4 with lesser changes at C4-5, most evident on the right (arrows). Center: Lateral view. Arrows point to the thickened, fused laminae of C2-4 with normal spaces between the laminae above and below. Right: Oblique view demonstrates the degenerative changes in the facets at these levels (arrows). While there is evidence of spondylosis at lower levels, the canal is not stenotic (see Fig. 2). The spurs projecting into the foramen in the oblique view were not present in the canal or foramen exposed at surgery and are presumed to be further lateral on the vertebral body. Hence, in this exposure, they may project over the foramen without actually being foraminal in location.

caused by enlarged facets encroaching on the posterior part of the intervertebral foramen. Telescoping, a common complication of apophyseal arthrosis, further reduced the transverse diameter. The posterior roots were particularly vulnerable to compression at their junction with the ganglia. The anterior roots, in contrast, rarely showed evidence of injury in the 120 cases these authors examined. Microscopically the curves impressed into the ganglion reflected the direction of bone compression with flattening and crowding of neurons around concave margins, fibrosis, and degeneration of neurons and nerve fibers.

Hadley\(^3\) also demonstrated compression of the nerve roots in the intervertebral foramen secondary to apophyseal hyperplasia. Friedenberg, et al.\(^2\), in a dissection of 41 cervical spines, noted that the most severe apophyseal joint changes were present at the high cervical levels, findings similar to those we encountered in our patients. At the C-5 and C-6 levels, there was an increased incidence of degenerative changes in the discs and joints of Luschka. A poor correlation was found between the changes seen radiographically and the results obtained from dissection of the apophyseal joints. In only 12 of 37 joints with anatomical changes were the alterations evident in plain films. They confirmed observations of Holt and Yates\(^4\) demonstrating severe degeneration of apophyseal joints in the presence of normal discs.

Payne and Spillane\(^5\) observed that the commonest cause of narrowing of the intervertebral foramina was uncovertebral osteophytes and narrowing of the adjacent intervertebral discs. Some intervertebral foramina in their group of 70 specimens did, however, show narrowing caused by osteophytes projecting from the inferior articular facets. The anterior roots often avoided compression by lying in a niche under a deformed uncus. The changes in the apophyseal joints were by no means an inevitable accompaniment of old age, and were frequently unaffected in cervical spondylosis. As in the patients described by Holt and Yates,\(^4\) these five patients showed marked evidence of apophyseal arthrosis with lesser abnormalities in the vertebral bodies. Changes in the posterior elements were more
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evident in the upper cervical region as compared to the spondylotic changes, which were more apparent in the lower cervical segments, suggesting that an independent stress-related mechanism caused these alterations.2,4

Case Reports

Case 1

This 53-year-old man had a 15-year history of recurrent neck pain. During the past 2 months, he noted increasing weakness and wasting in the muscles of his upper extremities, most evident in the deltoid and biceps, greater on the right. He had fasciculations and considerable loss of dexterity. His gait had become spastic and unsteady.

Examination. Reflexes were hyperactive in both upper and lower limbs, with unsustained clonus. Plantar responses were neutral. Sensation was intact.

X-ray examination of the cervical spine showed severe arthrosis involving the upper cervical segments, especially at C3-4, where hypertrophy of the posterior facets caused dorsal narrowing of both intervertebral foramina at this level (Fig. 1). Myelography disclosed partial block at the C3-4 level, with a prominent indentation into the dorsal quadrants of the spinal canal caused by apophyseal arthrosis. There were minimal changes in the floor at this level (Fig. 2).

Operation. Laminectomy of C-2, C-3, C-4, and C-5 was performed, with wide foraminal decompression at the C3-4 and C4-5 levels where the facets were markedly hypertrophied. The thickening of bone extended medially into the laminae, which were partially fused at these levels (Fig. 3). The nerve roots showed extensive perineural scarring at C3-4 and to a lesser degree at C4-5. Osteophytes in the floor of the canal were small and were not disturbed.

The dura was almost transparent and no abnormalities were noted through the membrane which expanded to fill the opening created by laminectomy and facetectomy. Little or no epidural fat was present. There was considerable thickening of the yellow ligament at C3-4 and C4-5.

Postoperative Course. The patient tolerated surgery well. Steady improvement followed with loss of fasciculations, return of power to both biceps and deltoids, and resolu-

FIG. 2. Case 1. Posteroanterior (left) and lateral views (right) in the myelogram show a partial block dorsally in the spinal canal, beginning below the arch of C-2 and maximal at C3-4, caused by the advanced apophyseal arthrosis and thickened, fused laminae.

FIG. 3. Case 1. A: Operative findings showing extensive arthrosis involving the posterior facets and laminae of C-3 and C-4 on the right extending into adjacent structures above, below, and medially. B: Decompression has included laminectomy of C-2, C-3, C-4, and C-5, with extensive foraminoctomy on the right. Facetectomy at C3-4 completely exposes the C-4 nerve root through the foramen. Neurolysis and excision of foraminal osteophytes completed the procedure. Arrows indicate the dorsal indentation of dura and nerve roots exposed by excision of the involved laminae and facets.
FIG. 4. Case 2. Upper: The initial myelogram shows a transverse defect at C3-4 caused primarily by shingling and the dorsal intrusion of thickened laminae and arthrotic facets removed at surgery (arrows). Lower: Repeat study 1 year later shows a fully expanded dural sac with dorsal migration of the spinal cord and no evidence of olisthesis or further spondylotic degeneration.

Case 2

This 63-year-old woman complained of difficulty in walking of 2 years' duration, with discomfort spreading across her shoulders down her upper extremities. She gradually lost the useful function in both hands. Her symptoms had increased in severity with a rapid deterioration in gait during the 3 months before presentation. Delay in urination was noted at this time.

Examination. Her gait was wide-based and spastic. She was unsteady in the Romberg position. Her reflexes were hyperactive throughout, including the biceps reflexes. Plantar responses were neutral. There was a sensory level to pinprick in the mid-cervical dermatomal area with poorly defined borders. Vibration and position sense were diminished in her lower extremities. Atrophy of the small muscles of her hands was prominent and fine consecutive movements were poorly performed.

X-ray films of the cervical spine disclosed hyperlordosis with shingling and severe arthrosis of the posterior facets, most evident at C3-4. Myelography disclosed marked indentation dorsally, most severe at C3-4 and C4-5, where a partial block was evident. A small step deformity was noted ventrally (Fig. 4 upper).

Operation. Laminectomy confirmed the severe arthrosis of the posterior facets, laminar thickening, and partial fusion at C3-4, and to a lesser degree C4-5. The canal was sharply indented conforming to the myelographic changes. Laminectomy from C-2 through C-7 permitted dorsal migration of the cord and removed the intruding masses posteriorly. Partial facetectomy completed the decompression at C3-4 and C4-5.

Postoperative Course. Return of function of both hands was gratifying and sustained. The patient was relieved of the major discomfort in her neck, and ambulation improved, her gait becoming stable after 3 months. Sphincter function returned to normal. This was maintained for 1 year, after which her walking deteriorated. A second myelogram showed no dorsal indentations or stenosis, with posterior migration of the spinal cord into an enlarged canal (Fig. 4 lower). A computerized tomogram disclosed evidence of generalized brain atrophy with a dilated ventricular system, Sylvian fissures, and cortical sulci. It was believed that her gait disturbance was cerebral in origin.

Case 3

This 55-year-old machinist had complained of intermittent cervical pain for 10 years. For 6 months he had noted unsteadiness in walking and was aware of numbness of the thumb and index fingers and over the radial aspect of his forearms.

Examination. Moderate restriction of cervical movement was most evident in hyperextension. Both deltoid and biceps muscles were atrophied with greater weakness prox-
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imally. The left hand was weak and dexterity diminished. His gait was spastic and unsteady. Reflexes were sluggish in his upper extremities, and hyperactive below. Plantar responses were in flexion. There were no sensory alterations.

X-ray examination showed extensive degenerative changes involving the posterior articular facets, maximal at C4-5 but also quite evident at C3-4, with lesser changes at C5-6. Spondylosis was minimal. Myelography showed a partial block at C4-5 with lateral defects at C3-4 and C5-6.

Operation. Laminectomy of C-3, C-4, C-5, and C-6 was performed, with unroofing of the lateral recesses and foramina. The major change dorsally was hypertrophy of the left posterior articular facets at C3-4 and C4-5, associated with thickening and fusion of the neural arches. Considerable perineural and peridural scarring was evident. There was no epidural fat. Minimal scarring was present in the floor of the canal.

Postoperative Course. Sustained improvement, especially in motor power, was noted early, and weakness resolved after 3 months. While reflexes remained sluggish or absent in the upper extremities, long tract signs in his lower extremities disappeared. Gait became normal and he returned to work. His only difficulty was in maintaining his neck in forward flexion for long periods.

Case 4

This 63-year-old truck driver had noted progressive weakness and loss of dexterity in both upper extremities, greater on the right side for 6 months. During the 3 months before admission, his gait had become increasingly unsteady. He was unaware of any change in sensation or in sphincter function. There was no history of injury and only minimal complaints of cervical discomfort.

Examination. Neurological evaluation disclosed an absent biceps reflex on the right, 2+ active on the left, with hyperactive triceps reflexes bilaterally. Reflexes in both lower extremities were hyperactive with sustained clonus. His plantar responses were neutral. His gait was spastic and ataxic. There was weakness in both deltoid and biceps muscles, with atrophy of the intrinsic muscles of his hands, more severe on the right. He had a vague sensory level to pinprick over the lower portion of his body extending to the mid-dorsal region. Vibratory appreciation was decreased in his toes; his position sense was intact.

X-ray examination showed spondyloarthrotic changes in the cervical spine. The posterior articular facets were severely arthrotic at the C4-5 level and on myelography, a partial block was evident here with a prominent dorsal defect. Lesser changes were apparent at the C3-4 level. While defects causing stenosis in the myelographic column were circumferential, the most prominent intrusions into the spinal canal were dorsal.

Operation. Laminectomy of C-3, C-4, C-5, and C-6 was performed, with wide unroofing of the lateral recesses and foraminotomy, the latter most extensive at C4-5. Enormous overgrowth of the posterior articular facets was present at C4-5, spreading to include the adjacent laminae which were thickened and partially fused, corresponding to the major myelographic defect. There was reactive scarring about the nerve roots at the C4-5 level, with lesser changes at the interspace below. Minor spurring was present in the floor of the canal and foramina. No epidural fat was present.

Postoperative Course. There was rapid improvement postoperatively. The patient’s gait became more stable, and his lower extremities less spastic. Motor power and atrophy improved first in the shoulder girdles and subsequently in both hands where dexterity returned to normal. The only residual deficit after 3 months was a slightly spastic gait.

Case 5

This 81-year-old man developed increasingly severe ataxia 4 months before hospitalization. He did not complain of pain, but was aware of weakness of both hands, more severe on the right side.

Examination. Biceps reflexes were sluggish, and all reflexes below this level were overactive. Plantar responses were extensor. There was atrophy of the small muscles of the right hand with additional paresis of the biceps and deltoid muscles. No sensory changes were observed. X-ray examination of the cervical spine showed spondylosis, and arthrosis of the posterior facets at C4-5 on the right side. The anteroposterior view in the myelogram showed a transverse defect at
C4–5 but, on the lateral view, there was a large indentation into the dorsal quadrants of the canal caused by a major intrusion of hypertrophied lamina and facets.

Operation. There was a fusion of the right laminae at C-4 and C-5, with overgrowth that included the arthrotic articular facets. The extradural compression was corrected by means of laminectomy and foraminotomy that extended from the lower half of C-3 through C-5. Facetectomy was necessary at C4–5 where peridural and perineural scarring was conspicuous. The floor of the canal was not remarkable.

Postoperative Course. The patient's gait improved, and after 3 months minimal dysfunction remained evident with slight spasticity in his lower extremities and only minor weakness in the left upper limb. He died 5 years after surgery of unrelated causes.1

Discussion

The ages of our patients varied from 53 to 81 years. Symptoms were present for 2 to 6 months in four patients, and for 2 years in one. Trauma played no causative role. One side was usually more involved than the other. Pain in three patients was never disabling, although it was present for many years before the onset of myelopathy. In all patients, weakness was present in the upper extremities, involving both proximal and distal muscles, with atrophy and loss of dexterity. Sensory changes were of lesser severity than the motor abnormalities. Sphincter function was rarely involved. Long-tract signs in each patient were manifested by a spastic, ataxic gait, hyperreflexia, and neutral or minimally abnormal plantar responses.

Both clinically and on x-ray examination the level of involvement was primarily at C3–4 and C4–5. Roentgenograms showed severe arthrosis of the posterior articular facets and involvement of the lamina with lesser changes on the vertebral bodies. These alterations were easily missed on the initial plain films. The myelograms, however, were distinctly positive in the lateral exposures where the defects in the oil column were prominent posteriorly with minor abnormalities in the floor of the canal.

Operation consisted of decompressive laminectomy with removal of the thickened and often fused laminae. Liberal foraminal decompression at times required excision of varying portions of the posterior articular facets, neurolysis and excision of foraminal osteophytes. Results in all five patients were gratifying with improved function in both upper and lower extremities; the residual deficits were primarily persistence of hyperreflexia and varying spasticity. No evidence of instability was encountered.

The anterior surgical approach is useless if relief of dorsal compression of neural elements is to be achieved.

References

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