Myelopathy in Cervical Spondylosis with Vertebral Subluxation and Hyperlordosis

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Excessive cervical lordosis with overlapping of the lamina may contribute to the production of myelopathy in patients with spondylosis.\(^2,4\) Congenital or developmental narrowing of the spinal canal predisposes such individuals to spinal cord compression and to the additional problems created by vertebral subluxation.\(^1,6,8\) The latter results from degenerative phenomena in both the intervertebral disc and in the posterior articulations,\(^5\) and often occurs above an area of spontaneous fusion of vertebrae caused by advanced spondylosis.\(^9,10\) The infolded ligamentum flavum and dura contribute to dorsal compression of the spinal cord in hyperextension. Telescoping of the lamina often referred to as "shingling" increases this effect.\(^3\) In the presence of subluxation, the cord is pinched in extension or in flexion between the edge of the step-like defect in the floor of the spinal canal and the leading margin of the displaced infra- or suprajacent lamina.\(^7,8\)

Clinical Material

The group of eight male patients with hyperlordosis and myelopathy that we are reporting had minimal to moderate spondylosis. All had critical narrowing of the ventrodorsal diameter of the spinal canal. Retrolisthesis was present in two patients at the C4-5 interspace. Anterolisthesis was noted in three patients, at C3-C4 in one and at C4-C5 in the other two. One patient had retrolisthesis at C3-C4, anterolisthesis at C4-C5, and fusion of the C5-C6 vertebrae. The remaining two patients had stenosis of the spinal canal further compromised by dorsal intrusions of telescoped lamina and underlying soft tissues without vertebral displacement. Excessive cervico-dorsal kyphosis predisposes these patients to compensatory upper cervical hyperlordosis.

Clinical Findings and Course

A summary of the clinical findings and courses in the eight patients is presented in Table 1.

Long Tract Signs. In all patients, long tract signs with a spastic ataxic gait appeared, preceded by weakness and loss of dexterity in the arms in four. Sphincter function was intact in six patients. Hyperreflexia was present from the biceps level downward. Bilateral Babinski signs were noted in five. Fifteen patients manifested decreased vibratory appreciation and position sense in the toes. Gross atrophy of the small muscles of the hands occurred in five patients. In two, weakness was compounded by stere anesthesia, so that the hands were essentially useless. Atrophy of the shoulder girdle and proximal musculature was found in five, and fasciculations in four. The anatomical level appeared somewhat higher than that usually encountered in patients with cervical spondylomyelopathy.

Hyperlordosis. None of these patients had a clinical pattern that could be related solely to hyperlordosis, with or without pseudospondylolisthesis, although hyperlordosis of the upper cervical spine in the neutral position was seen in all patients. The excessive cervical curve was most pronounced in a patient with increased cervicodorsal kyphosis (see Case 2 reported below).

Cervical Spine Films. X-ray films in flexion and extension in patients with hyperlordosis and retrolisthesis revealed that the narrowing of the canal was greatest in extension.
while forward flexion widened the canal (Fig. 1). The step deformity in the floor of the spinal canal decreased or disappeared in flexion. The reverse occurred in patients with anterolisthesis. Vertebral dislocation of 3 to 5 mm was noted in the patients with pseudospondylolisthesis, well above normal physiological limits.7 In no patient was there a defect in the pars interarticularis. Osteoarthrits of the posterior facets was found in patients with retrolisthesis who also manifested degenerative disc disease. Fusion of the infrajacent two vertebral bodies was evident in two patients.

Spinal Fluid Protein. The CSF protein content was normal in five patients, and elevated from 60 to 104 mg% in the other three.

Myelography. Partial or almost complete block in six patients was disclosed by myelography with the head and neck in the extended position. Ventroflexion relieved the block, allowing the oil to accumulate above and below the involved level. Measurements of the ventrodorsal depth of the spinal canal were 0.7 to 0.9 cm, significantly below accepted normal values.11 The dorsal defect seen in lateral exposures presented a corresponding step-ladder pattern in the anteroposterior view. The dorsal intrusions of the lamina and ligamentous infolding contributed more to this effect than the osteophytes in the floor of the canal. The extent of the laminar decompression required could be estimated as one or more lamina above and below the total longitudinal deformity seen in the myelogram in both anteroposterior and lateral projections.

Type of Operation. Liberal laminectomy and foramenotomy permitted the cord to rise dorsally into an expanded and shorter canal, away from ventral step deformities and ridges.1,4,10 In only one patient was it considered advisable to excise the osteophytes in the floor of the canal because of the size of the intrusions. Significant nerve root adhesions and foraminal osteophytes were not found. Posterior spinal fusion was performed in two patients, one with anterolisthesis and one with retrolisthesis. Anterior fusion was subsequently performed in the patient with anterolisthesis because of increasing deformity, with eventual stabilization in a kyphotic position. Spinal fusion was

<table>
<thead>
<tr>
<th>Age</th>
<th>Duration of Symptoms</th>
<th>Spinal Anomaly</th>
<th>Spondylosis</th>
<th>Level</th>
<th>Pain, Cervico-Brachial</th>
<th>Onset, Extremities</th>
<th>Preop Deficit</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>52</td>
<td>3 yrs</td>
<td>retrolisthesis</td>
<td>slight</td>
<td>C4-5</td>
<td>+</td>
<td>arm</td>
<td>spastic gait, hyperreflexia</td>
<td>good</td>
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<tr>
<td>57</td>
<td>1 yr</td>
<td>retrolisthesis</td>
<td>slight</td>
<td>C4-5</td>
<td>0</td>
<td>leg</td>
<td>atrophy hands, spastic gait, hyperreflexia</td>
<td>good, gait improved (fusion)</td>
</tr>
<tr>
<td>60</td>
<td>6 yrs</td>
<td>anterolisthesis</td>
<td>moderate</td>
<td>C3-4</td>
<td>0</td>
<td>leg</td>
<td>atrophy hands, spastic gait, hyperreflexia</td>
<td>good, slight residual spasticity</td>
</tr>
<tr>
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<td>2 yrs</td>
<td>anterolisthesis</td>
<td>slight</td>
<td>C4-5</td>
<td>0</td>
<td>leg</td>
<td>hyperreflexia, Brown-Séquard</td>
<td>good</td>
</tr>
<tr>
<td>70</td>
<td>9 mos</td>
<td>anterolisthesis, fusion C5-6</td>
<td>moderate</td>
<td>C4-5</td>
<td>+</td>
<td>arm</td>
<td>atrophy hands, spastic gait, quadriparesis</td>
<td>fair (fusion)</td>
</tr>
<tr>
<td>62</td>
<td>3 yrs</td>
<td>anterolisthesis, anterolisthesis, fusion C5-6</td>
<td>moderate</td>
<td>C3-4</td>
<td>C4-5</td>
<td>0</td>
<td>arm</td>
<td>atrophy hands, stereanesthesia, mild spasticity, hyperreflexia</td>
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<td>38</td>
<td>4 yrs</td>
<td>upper cervical hyperlordosis, cervicodorsal kyphosis</td>
<td>moderate</td>
<td>C2-5</td>
<td>C6-7-T1</td>
<td>+</td>
<td>arm</td>
<td>old double ataxias, torticolis, atrophy hands, stereanesthesia, hyperreflexia</td>
</tr>
<tr>
<td>45</td>
<td>3 days (? )</td>
<td>hyperlordosis</td>
<td>slight</td>
<td>C3-6</td>
<td>+</td>
<td>arm and leg</td>
<td>spastic gait, tetraparesis, hyperreflexia, recent CVA</td>
<td>minimal improvement</td>
</tr>
</tbody>
</table>
Myelopathy in Cervical Spondylosis

not considered necessary in the remaining patients. Nevertheless, further observation is mandatory. Laminection included a minimum of four vertebrae. Laminection from C-2 to T-2 was performed in the patient with severe cervicodorsal kyphosis (Case 2).

All patients, except one, were operated on in the sitting position. The cervical spine was placed in a slightly flexed position in patients with retrolisthesis, opening the spinal canal to its maximum proportions. Spreading the lamina facilitated laminectomy. Flexion was avoided in patients with anterolisthesis in whom a neutral position of the spine was used. Precautions included wrapping of the lower extremities in elastic bandages and the insertion of a central venous catheter into the right atrium. A turgid, intact dural envelope was helpful in minimizing epidural bleeding by tamponade. Foramenotomy may be difficult because of crowding of pedicles and facets; it need not be extensive since foraminale osteophytes are rarely prominent in these patients.

Results of Operation. The progress of the myelopathy was halted in seven of the eight patients. Spasticity and hyperreflexia diminished and gait became more stable. Hand function improved. This was most evident in two patients with useless hands who were able to function effectively after 1 month and subsequently became independent of ancillary aid. The five patients with good functional results were employed 3 to 6 months following operation, although residual long tract signs persisted in all. The results indicate that decompressive laminectomy early in the course of the disease provides better results than delayed surgery in patients with fixed lesions.

Case reports of two typical patients, one with retrolisthesis and one with hyperlorosis, are given below.

Case Reports

Case 1. This patient was a 57-year-old laborer with a 1-year history of a spastic, ataxic gait of spontaneous onset. He had sustained a neck injury in an automobile accident 15 years previously and had been quadriplegic with recovery after 15 months.

Examination. There was atrophy of the small muscles of the hands, and a delay in micturition. Biceps reflexes were increased.

The triceps and the reflexes in the lower limbs were overactive, with ankle clonus and neutral plantar responses. Vibratory appreciation was diminished in the toes.

X-ray films (Fig. 2) disclosed retrolisthesis of C-4 dorsally over C-5, the inferior lip of the body of C-4 approaching the leading margin of the lamina of C-5, producing a scissoring or pincer effect on the cord at this level in extension. In flexion, subluxation was relieved and the normal contour of the canal restored. The myelogram showed the dorsal indentations into the canal caused by the infolding of ligaments and crowded lamina, and moderate evidence of spondylosis.

Operation. Laminection of C-4, C-5, and C-6 with spine fusion was performed in September, 1958.
Postoperative course. The patient's gait had improved significantly at the time of discharge 2 weeks later, with persistent but less spasticity. He had maintained this status when last seen 5 years later. Solid fusion had occurred.

Case 2. A 38-year-old man had double athetosis and spasmodic torticollis, secondary to corpus striatum damage in infancy. During the past 4 years, he complained of pain across his shoulders radiating into both arms.

Examination. There was shoulder girdle weakness with numbness and weakness of both hands, with atrophy. Spastic paraparesis with ataxia was present. A dermatome sensory deficit was present in the C-5 and C-6 distribution, bilaterally.

Cervical spine films (Fig. 3) disclosed reversal of the lordotic curve in the lower cervical region with an exaggerated kyphosis beginning at the C-6 segment and hyperlordosis with shingling above this area. Spondylotic alterations were noted in the lower cervical segments where little or no movement was elicited. Shingling was accentuated in extension, but was also present to a significant degree in the neutral position. The spinous processes at C-6 and C-7 were separated at their base from the underlying lamina.

Myelography disclosed extreme narrowing of the dorsal quadrants of the spinal canal at the crowded C-3 and C-4 segments.

Operation. Extensive laminectomy from T-1 through C-2 was done. The spinous processes at C-6 and C-7 were fused in a continuous sheet of bone.

Postoperative course. Partial relief of the neck and extremity pain with slight improvement in hand function was obtained, which unfortunately deteriorated after 2 months.

Discussion

All eight patients presented signs of cord compression. Cervicobrachial pain was not prominent, but weakness, atrophy, and loss of sensation in the hands was common. These changes relate more to the myelopathy than to differential compression of the nerve roots in the foramina.

Developmental Stenosis. The capacity of the spinal canal may be compromised by developmental stenosis without diminishing the size of the neural foramina. Such patients may remain asymptomatic until complications of spondylosis or vertebral subluxation

Fig. 2. Case 1. Left: X-ray film taken with the spine in extension shows retrolisthesis of C-4 dorsally over C-5, producing a pincer effect on the cord (arrows). Center: X-ray film of the spine in flexion shows subluxation relieved (arrows) and the normal contour of the canal restored. Right: Myelogram shows the dorsal indentations in the canal caused by the infolding of ligaments and the crowded lamina.
occur, abolishing the critical reserve space for the spinal cord. This results in myelopathy but not in nerve root compression.9

Shling and Subluxation. Both Penning7,8 and Crandall and Batzdorf9 described the phenomenon of shingling and subluxation producing myelopathy in patients with narrow cervical spinal canals. Isolated cases are included in other reports.1,4,6 The term "shingling" has been used to describe the x-ray and operative findings of lamina crowded beneath each other, causing pathological posterior intrusion into the dorsal segments of the spinal canal. It is difficult to evaluate the frequency of these complications, for they may not be apparent unless flexion and extension views of the cervical spine are taken in all cases. With such studies, it may be possible to evaluate the significance of retro- or anterolisthesis in causing myelopathy.7,8

Pathological Dislocation. The spinal canal is widest in the anteroposterior diameter in the position of flexion. The diameter of the stretched spinal cord in this position also diminishes but it may be held against spondylotic protrusions with resulting injury.2 In the presence of retrolisthesis, the suprajacent vertebra slides backward, compressing the spinal cord between the inferoposterior lip of the retrolisthesis vertebra and the leading anterior margin of the lamina of the vertebra below.7,8 Hyperextension increases the degree of posterior displacement, while forward flexion reduces displacement so that it may not be apparent in routine films (Fig. 1 A). The reverse occurs in patients with anterolisthesis, extension tending to minimize or correct such displacement while flexion increases it (Fig. 1 B). Spinal cord compression occurs in anterolisthesis when the spine is flexed. The distance between the posterior margin of the vertebral body and the adjacent lamina does not approach critical values unless severe dislocation occurs.

The cause of the retrolisthesis may relate to the disposition of the planes of the posterior facets, relaxation of the supporting ligaments and degenerative alterations in the articular cartilages and in the intervertebral disc.5 Narrowing of the affected interspace may not occur.7,8 An excessive dorsal or lower cervical kyphosis results in a compensatory upper cervical hyperlordosis.5 Dorsal displacement of the suprajacent vertebral segments may then occur as degenerative changes ensue. Collapse of the intervertebral disc without spondylotic fusion results in ky-

Fig. 3. Case 2. Left: Myelogram of the cervical spine in the neutral position shows extreme narrowing of the spinal canal at C-3 and C-4, reversal of the lordotic curve with kyphosis beginning at C-6, and hyperlordosis with shingling above this area. Center: X-ray film of cervical spine in extension accentuates the shingling. The spinous processes at C-6 and C-7 are separated at their base. Right: X-ray film of cervical spine in flexion showing relative fusion over the area of kyphosis with remarkable mobility in the superior segments.
phosis if the posterior articulations remain intact. Should destructive arthritis of the posterior articulations occur, forward subluxation of the vertebral body may result, similar to the pseudospondylolisthesis in the lumbar region.5

Fusion. The presence of fused vertebrae is often seen below the site of dislocation, the forces of motion being concentrated on the suprajacent interspace with resultant degenerative alterations and possible herniation of the intervertebral disc.6,10

Ligamentum Flavum. The ligamentum flavum and the posterior longitudinal ligament thicken in hyperextension and further narrow the sagittal diameter of the canal, often blocking cerebrospinal fluid flow during manometry and myelography.5,7,8 The cervical cord shortens and thereby thickens, further compromising available space.2

Diagnosis and Surgical Positioning. In patients with the ridge syndrome and retrolisthesis, the additional narrowing of the spinal canal that occurs with hyperextension may exceed critical limits during the performance of diagnostic studies and while positioning the patient for anesthesia. These abnormalities may cause the myelopathic complications that follow anterior spinal fusion, which required placing the patient’s neck in hyperextension. This may be sufficient in the presence of operative manipulation of the vertebrae and impaction of the bone graft to injure the spinal cord. To provide the maximum diameter of the spinal canal when positioning the patient for surgery, all of these factors relating to vertebral subluxation must be fully appreciated.

Shingling is accentuated by hyperextension of the spine. When complicated by retrolisthesis, the cord is compressed by a pincer or scissor-like action between the body of the dorsally dislocated vertebra and the lamina of the inferior segment. A lesser compressive effect is evident in flexion in patients with anterolisthesis. While spondylosis is present, spurring was found to be of less significance in these patients than in patients with spondylotic myelopathy uncomplicated by the above factors. Since the spinal canal is narrowed primarily by posterior intrusions of lamina and yellow ligaments, early liberal laminectomy is the treatment of choice.

Summary

We have discussed the clinical findings, surgical treatment, and results in eight patients with cervical myelopathy secondary to spondylosis and have emphasized such related factors as hyperlordosis, vertebral subluxation, pincer mechanism, and “shingling.” Seven of our patients with long-tract signs had good results from laminectomy.

References