Thoracic spinal stenosis: experience with seven cases

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The authors report seven cases of thoracic spinal stenosis caused by thickening of the laminar arch and facet joints. Claudication was a prominent clinical feature of this disorder. Motor and sensory abnormalities were found in all cases with either segmental or peripheral distribution. Diagnosis was best made from the computerized tomography scans. Treatment consisted of extensive posterior decompression including medial facetectomy, resulting in satisfactory improvement in five of the seven patients. A review of the literature regarding this disorder is presented.

KEY WORDS • spinal stenosis • thoracic spine • myelopathy

Thickening of the posterior spinal elements leads in some cases to compromise of the spinal canal and its neural structures. This phenomenon is well recognized as a disease entity, particularly in the cervical and lumbar spine; however, it has rarely been reported in the thoracic spine in the absence of metabolic disorders or a history of trauma. We present seven cases of thoracic myelopathy caused by thoracic spinal stenosis, and discuss their symptoms, radiological findings, and treatment.

Summary of Cases

Seven patients with thoracic spinal stenosis were operated on at our institutions during the 8 years between 1978 and 1986. A summary of their clinical presentation is presented in Table 1. These patients ranged in age from 38 to 68 years (average 48 years).

Symptoms

All the patients complained of motor and sensory deficits in their legs during the clinical course. These symptoms were described variously as fatigue, weakness, tightness, heaviness, cramping, or a "pins-and-needles" feeling, and were often experienced simultaneously in both lower limbs, usually more pronounced in one than the other. Sometimes these symptoms appeared first in one leg and later spread to the other. These complaints were usually brought on by physical exercise or by maintaining the same position for a long time, and were relieved by rest or a change in posture. Three patients had a dull aching pain in their low-back area, which usually did not radiate into the legs, as sciatica does. Incontinence of urine was intermittently observed in one patient. The duration of symptoms ranged from 5 months to 6 years (average 2.8 years).

Physical Signs

All seven patients had weakness of the legs. Knee and/or ankle jerks were normal in one patient, diminished or absent in three, and exaggerated in three. Three patients had muscular atrophy: two in both thighs (Cases 4 and 7) and one in the calf (Case 2). The straight-leg raising maneuver was normal in five patients and restricted in two. Sensation was impaired in all modalities, but in a variable distribution; sensory deficit was segmental in four cases and of a stocking type, suggesting that the lesion was of peripheral origin, in three cases. From these neurological findings it was difficult to predict the vertebral level affected.

Neuroradiological Findings

The anteroposterior diameter of the spinal canal at the affected level varied from 10 to 13 mm on plain thoracic spine x-ray films. However, neither routine thoracic spine films nor polycycloidal tomography demonstrated calcification of the ligamentum flavum. Myelographic findings included a partial extradural defect in five cases and complete obstruction in the other two, but failed to indicate the etiology. The myelographic block was at the T10-11 interspace in one case, at T11-12 in five, and at T9-11 in one. In all seven patients, plain computerized tomography (CT) dem-
TABLE 1
Clinical summary of seven cases with thoracic spinal stenosis

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Symptoms</th>
<th>Duration of Symptoms</th>
<th>Myelographic Findings</th>
<th>Treatment*</th>
<th>Ligamentum Flavum</th>
<th>Follow-Up Period</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Thickness</td>
<td>Calcification</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>68, M</td>
<td>back pain, motor &amp; sensory deficits, urinary incontinence</td>
<td>2 yrs</td>
<td>incomplete (T10-11)</td>
<td>L(T10-11), F(T11-11)</td>
<td>4 mm yes</td>
<td>6 yrs</td>
<td>recurrence after initial relief</td>
</tr>
<tr>
<td>2</td>
<td>48, M</td>
<td>motor &amp; sensory deficits</td>
<td>3 yrs</td>
<td>incomplete (T11-12)</td>
<td>L(T10-12), F(T11-12)</td>
<td>3 mm no</td>
<td>5½ yrs</td>
<td>some relief of symptoms</td>
</tr>
<tr>
<td>3</td>
<td>42, M</td>
<td>motor &amp; sensory deficits</td>
<td>1 yr</td>
<td>complete (T11-12)</td>
<td>L(T9-12), F(T11-12)</td>
<td>3 mm yes</td>
<td>5 yrs</td>
<td>relief of symptoms</td>
</tr>
<tr>
<td>4</td>
<td>43, F</td>
<td>motor &amp; sensory deficits</td>
<td>1 yr</td>
<td>incomplete (T9-11)</td>
<td>L(T9-L1), F(T10-11)</td>
<td>4 mm yes</td>
<td>5 yrs</td>
<td>relief of symptoms</td>
</tr>
<tr>
<td>5</td>
<td>46, M</td>
<td>back pain, motor &amp; sensory deficits</td>
<td>6 yrs</td>
<td>complete (T11-12)</td>
<td>L(T10-L1), F(T11-12)</td>
<td>2 mm yes</td>
<td>4½ yrs</td>
<td>recurrence after initial relief</td>
</tr>
<tr>
<td>6</td>
<td>52, M</td>
<td>back pain, motor &amp; sensory deficits</td>
<td>4 yrs</td>
<td>incomplete (T11-12)</td>
<td>L(T10-L1), F(T11-12)</td>
<td>2 mm no</td>
<td>4 yrs</td>
<td>relief of symptoms</td>
</tr>
<tr>
<td>7</td>
<td>38, M</td>
<td>motor &amp; sensory deficits</td>
<td>5 mos</td>
<td>incomplete (T11-12)</td>
<td>L(T9-L1), F(T11-12)</td>
<td>3 mm yes</td>
<td>4 mos</td>
<td>some relief of symptoms</td>
</tr>
</tbody>
</table>

* L = laminectomy, F = medial facetectomy.

Operative Findings

All seven patients were treated with wide posterior decompression, which included laminectomy to obtain sufficient epidural space and a medial one-third or one-half facetectomy of the superior and inferior articular facets at the site of the myelographic block (Fig. 3). The ligamentum flavum, ranging from 2 to 4 mm wide, was removed, and was found to be hardened and discolored in all patients. Even after this extensive decompression, the pulsation of the dural sac was invisible after surgery in four patients. Spinal fusion was not performed.

FIG. 1. Preoperative computerized tomography scans. Left: Scan at T-11 in Case 7 revealing thickening of the laminar arch and facet joints. Center: Scan at T-11 in Case 1 showing not only thickening of the laminar arch and facet joints but also a linear high-density structure (arrow) in the posterior portion of the spinal canal. Right: Scan at T-11 in Case 7 following metrizamide myelography demonstrating anterior displacement of the spinal cord.
Thoracic spinal stenosis

Histological examination of the excised ligamentum flavum demonstrated numerous fragments of bone and/or cartilage.

Postoperative Course

The surgical results are shown in Table 1. All the patients experienced improvement in their motor and sensory disturbances after surgery. However, two patients (Cases 1 and 5) had a recurrence of their symptoms 2 and 3 years, respectively, after initial relief. At follow-up examination 4 months to 6 years (average 4.2 years) postoperatively, none of the patients exhibited instability on plain thoracic x-ray films.

Discussion

A stenotic spinal canal in the cervical or lumbar region is a well-known clinical entity. However, stenosis involving a thoracic vertebra is rarely mentioned in the literature. Complaints of claudication in the presence of low-back pain are common clinical features of this disorder. This symptom closely resembles that of lumbar spinal stenosis, but is usually not accompanied by pain radiating into the legs.

Motor and sensory abnormalities were evident in all the cases presented here, and in three cases were variously combined with asymmetrical, atrophic, hyporeflexic, or areflexic paraparesis and stocking-type sensory loss. These findings must be distinguished from deficits caused by a lesion at the level of a nerve root. Lesions at the level of the lumbar cord enlargement close to the T10–12 vertebrae alter these segments of the spinal cord and produce not only spastic paraparesis as a segmental sign but also a flaccid type of paralysis with typical signs of lower motor neuron involvement.

The mechanism of these clinical manifestations is not clearly understood. In the early stage of spinal cord compression, the gray matter tends to be damaged first, since the anterior horn is located in a watershed area between the perforating blood supply from the ventral and lateral arteries. Therefore, a compressive lesion between T-10 and the top of the T-12 vertebra causes a mixture of upper and lower motor neuron involvement.

On plain lateral x-ray films, the posterior aspects of the thoracic vertebrae are not condensed and distinct as on the cervical and lumbar spine. Kurokawa, et al., regarded the anterior margin of the spinous process as the posterior wall of the thoracic spinal canal, but it is not always possible to accurately identify this structure because of the normal lateral curvature of the thoracic spine. The average anteroposterior diameter of the normal spinal canal at T-10, T-11, and T-12 is 14.4, 14.3, and 15.9 mm, respectively. In our series, the narrowest anteroposterior diameter on the plain lateral roentgenograms was 10 mm at T-11. Retrospectively, the plain films were diagnostic only, since the narrowing of the spinal cord was not appreciated until a myelogram and a CT scan were obtained. Myelography demonstrated only an extradural mass effect, and therefore tended to be of little value, either misleadingly suggesting a herniated thoracic disc or demonstrating a complete block.

Fig. 2. Magnetic resonance image in Case 7 showing posterior indentations of the spinal cord at T9–10, T10–11, and T11–12.

As we reported in our previous paper, CT proved to be the best diagnostic imaging modality; a plain CT scan showed thickened laminar arches and facet joints with shallow lateral recesses resulting in a decrease of

Fig. 3. Computerized tomography scan at T-11 in Case 7 showing the extent of the posterior decompression.
the spinal canal diameter and cord compression. A calcified ligamentum flavum appeared as a linear high-density structure in the posterior portion of the spinal canal, which was an additional component causing narrowing of the spinal canal. Although the findings of thoracic spinal stenosis on MR imaging have not been reported in the literature, this modality alone seemed to be inadequate for delineation of the stenosis. However, MR imaging should be the primary investigational modality for evaluation of the pathology in the anterior spinal elements such as spondylosis, herniated discs, or hypertrophied posterior longitudinal ligament.

The only successful form of treatment is posterior decompression, which must be sufficient longitudinally and laterally to relieve the stenosis. For that purpose, the procedure includes removal, not only of the thickened laminar arches but also of the medial facets, and in addition the ligamentum flavum should be carefully freed from the occasional adherent dura mater. At that time, the lateral half of both the superior and inferior articular facets should be preserved as much as possible to safeguard against possible instability.

All the patients reported here experienced initial improvement in their motor and sensory disturbances after surgery despite the relatively long clinical history. In two of these cases, however, preoperative symptoms recurred a few years after surgery. This deterioration may be related to inadequate surgical decompression. Although the medial facetectomy at one level did not cause spinal instability, one must always be aware of this possibility during the years subsequent to such an extensive dorsal unroofing.

References

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