Nodule of the Ligamentum Flavum as a Cause of Nerve Root Compression

Case Report

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Abnormalities of the ligamentum flavum have been related to the common low back disorder of cauda equina radiculopathy in lumbar spondylolisthesis.2,4,10 Patients with lumbar spondylolisthesis complain of leg fatigue, numbness, and occasionally bladder discomfort and voiding difficulties related to ambulation. Because of these symptoms, attention is often directed to vascular insufficiency of the legs rather than to a spinal abnormality.

The ligamentum flavum lies between the vertebral laminae and is attached to the superior margin of the laminal arch below and at the center of the undersurface of the laminal arch above. Laterally it extends to and enters into the formation of the joint capsule between the articular facets and their intervertebral foramina. Medially it blends with the interspinous ligament and extends the entire length of the vertebral canal.11 The ligamentum flavum is normally composed of elastic tissue which appears yellow. Embryologically, elastic tissue differentiates later than collagen fibers, but in the same general manner. The elastic tissues develop and merge into dense plates or collect into compact elastic bundles about the sixth month of development.2 The thickness of the ligamentum flavum varies, the thickest portion being located medially. Measurements in the lumbar area have been reported, and thickness in this area does not vary significantly between the individual lumbar spaces that have been measured.9,13 Since the anteroposterior diameter of the lumbar spinal canal cannot be well appreciated on routine radiographs, evaluation of the effect of hypertrophy of the ligamentum flavum is difficult.

Case Report

A 48-year-old obese man had complained of left testicular, back, and leg pain for 3 years. The apparent onset was 3 years earlier; while sitting on the toilet, he had sneezed and developed severe pain in the left buttock. Thereafter he had pain in the left calf while walking. Examination at that time disclosed arteriosclerotic occlusive disease of the legs which was confirmed by appropriate contrast studies. A prosthetic arterial bypassing procedure was performed, partially relieving him of his symptoms for 2 1/2 years.

When seen by us the patient complained of pain in the left groin and left testicle, regularly increased by standing, especially on the left leg. Coughing and sneezing did not increase the pain. The patient preferred to sleep on his back for if he slept on his abdomen the outer borders of his feet would become numb. Prolonged walking caused hesitancy and slowness in emptying his bladder. Relief from pain was always gained by sitting down and flexing the low back.

Examination. The patient's gait was normal, and he was able to walk well on heels and toes. He had no spinal tenderness and back motions were free, but inclination to the left always aggravated the testicular pain. There was no sensory loss, and muscular power was normal. All the reflexes including the Achilles reflex were difficult to obtain bilaterally. Vascular studies, including angiograms, showed ample blood supply to the legs.

It was believed that the patient's difficulties stemmed from a spinal abnormality, specifically, spondylotic cauda equina radiculopathy. Roentgenograms of the lumbar spine showed relative narrowing of the lumbosacral interspace but were otherwise normal. Lumbar myelography, using 9 cc of Pantopaque, showed a prominent posterior deformity of the radiopaque column at the L4–5 level (Fig. 1). Contrast medium exam-
Nodule of the Ligamentum Flavum

There was no evidence of abnormality of the laminae or spinous processes of the fourth or fifth vertebrae. The ligamentum flavum appeared much thicker than usual, and with the bone above it and below it removed, it seemed to act as a constricting band producing a slight depression of the dural sac, more on the left. The ligament was then divided in the midline and each half reflected laterally. This revealed a nodule 1 cm in diameter on the undersurface of the left side. The ligament was totally excised for study, and the interspace and nerve roots thoroughly explored. No herniated disc material or other abnormality was present.

The patient had a comfortable postoperative course and when last heard from 14 months after operation was asymptomatic except for mild morning backache disappearing after 10 minutes' activity.

Pathological Examination. The lesion was a yellow-brown, smooth-surfaced, hemispheric protrusion from the inner surface of the ligamentum flavum measuring 1 cm across and 0.5 cm high (Fig. 2). Examination of the cut surface showed the nodule to bulge from a focally thinned area of ligamentum flavum. A small cyst was centrally placed in the nodule and this extended by a narrow neck to the ligament. Microscopically the nodule was composed of disrupted, torn, and degenerated collagen and elastic connective

Fig. 1. Lumbar myelograms showing defect at the L4-5 level. Anteroposterior view (left) and lateral view (right).

Fig. 2. Cross section of gross specimen of ligamentum flavum showing traumatic nodule on inferior surface.
tissue. Granulation tissue with incorporated hemosiderin was present peripherally. The gross and microscopic features indicated that this lesion developed as a result of trauma rather than infection, neoplasia, or other cause.

Discussion

Enlarged yellow ligaments were described in 1913 by Elsberg as accompanying a "peculiar disease" of the roots of the cauda equina. Several other authors described additional instances of hypertrophy of the yellow ligament, bringing the total number of such cases to 13 by 1937. A history of recent trauma has usually been obtained in cases of hypertrophy of the ligamentum flavum and has been regarded as the cause of the syndrome. Flexion and torsion of the trunk has been the most effective trauma. The pain produced is usually unilateral, aggravated by weight bearing, and not aggravated by increased intraspinal pressure such as coughing or sneezing. Patients usually gain relief from rest and lying down. At times the pain may be so strongly related to walking that vascular insufficiency may be diagnosed. This confusion commonly arises when there is associated significant arteriosclerotic occlusive disease of the legs, and the physician is unaware of the existence of spondylotic cauda equina compression. Lumbar myelography is necessary to demonstrate the pathological process adequately. The anteroposterior diameter of the lumbar spinal canal is reduced by bulging of a degenerative disc anteriorly, and laterally by the encroachment of enlarged facets that compromise the spinal canal. Posteriorly, the hypertrophy of the ligamentum flavum or, as in this case, a traumatic nodule of the ligamentum flavum, further reduces the diameter of the lumbar spinal canal, producing compression of the cauda equina. The degree of compression varies with posture of the low back, and the exaggeration of the normal lordotic curve increases the compressive effect upon the roots of the cauda equina. In our case, no degenerative disc material was present in the spinal canal; in fact, the discs appeared normal. After removal of the hypertrophied and herniated ligamentum flavum, adequate decompression of the cauda equina was obtained in this case.

Summary

We have reported a case of spondylotic cauda equina radiculopathy featuring unique compression of the cauda equina by a trauma-produced nodule of the ligamentum flavum.

References