Ligamentum flavum hematoma

Report of two cases

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Two patients presenting with signs and symptoms suggestive of nerve root compression secondary to extradural masses were found to have ligamentum flavum hematomas. Both patients had neurological deficits preoperatively and regained normal function postoperatively. There was no significant antecedent injury in either case. The symptom course was longer than that for spontaneous epidural hematoma. In one case, there was remodeling of bone, initially suggesting either infection or tumor.

KEY WORDS: hematoma • ligamentum flavum • nerve root • radiculopathy • laminectomy

In the fourth through sixth decades of life, a herniated nucleus pulposus is the most common cause of nerve root compression. Other causes include infection, neoplasm, or canal stenosis; a much less common cause is a spontaneous epidural hematoma. Related to a spontaneous epidural hematoma is a hemorrhage that primarily involves the ligamentum flavum. We report two recently treated cases in which the patients presented with signs and symptoms suggestive of nerve root compression due to herniated discs; however, radiological studies demonstrated masses occurring in the posterior aspect of the spinal canal. In both instances, there was operative and pathological confirmation of a hematoma localized in the ligamentum flavum, without evidence of neoplasm, infection, or abnormal blood vessels. These two cases are presented along with a review of literature on spontaneous epidural hematomas to contrast the presentation.

Case Reports

Case 1

This 43-year-old man suffered a minor back injury in 1973 and was doing well on conservative therapy until 2 months prior to admission when he had the insidious onset of a stiff and painful back after returning from a trip. Lifting luggage was the only significant activity performed; however, no specific onset of pain was related to this lifting. The patient’s attempt to relieve the pain by performing his usual back exercises was unsuccessful. The pain in his back and lower right extremity worsened and he developed numbness over the posterior thigh and calf. There was no bowel or bladder dysfunction. His medical history was significant only for hypertension that was treated with a beta blocker. There was no history of lumbar surgery or puncture.

Examination. On examination, the patient had normal neurological function in the lower left extremity. In the lower right extremity, he had 5/5 strength in the hamstring muscles and 4+/5 strength in the dorsiflexor and plantar flexor muscles. Sensation was intact, but he was unable to heel or toe walk and ankle and knee jerks were absent on the right. Myelography and postmyelography computerized tomography (CT) showed a large posterior extradural defect at the L4-5 level (Fig. 1). This appeared to be a posterior soft-tissue mass located mainly on the right, displacing the thecal sac anteriorly and to the left. The lamina and facet at this level appeared to be eroded and remodeled and a neoplasm was suspected. The mass enhanced slightly after intravenous administration of contrast medium. Electromyography showed chronic and ongoing denervation in both the L-5 and S-1 nerve roots on the right. The cerebrospinal fluid obtained at myelography...
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Fig. 1. Case 1. Postmyelography computerized tomography scan demonstrating a posterior extradural mass compressing the thecal sac at the L4–5 level.

showed a normal glucose level, a slightly elevated protein level, and a normal cell count. Serum protein electrophoresis was normal.

Operation. The patient initially underwent a right hemilaminectomy at L-5. Immediately after removal of the L-5 lamina, the ligamentum flavum was identified and found to contain a brownish mass. This was dissected free from all structures and removed after a total laminectomy had been performed at L-4 and L-5. The patient had an uncomplicated postoperative course and has regained normal neurological function.

Pathological Examination. Pathological examination of the surgical specimen revealed old hemorrhage and degenerative change within the ligament (Fig. 2). There was no evidence of infection, neoplasm, or abnormal blood vessels.

Case 2

This 60-year-old man stepped up onto a box during the performance of his job and experienced the onset of sharp cramps in his right leg. He was able to continue working for 3 weeks, but then the pain became unbearable and he was taken to the hospital in an ambulance. He was treated conservatively and was able to walk with crutches. He complained of discomfort and weakness in both lower extremities. His medical history was significant for hypertension for which he was being treated with a beta blocker. He was receiving no other medications.

Examination. On evaluation, the patient was paraparetic, with marked weakness in both dorsiflexor and plantar flexor muscles, more on the right side than the left. He had no complaints of urinary or rectal incontinence but did have decreased rectal sphincter tone. Electromyography revealed nerve root irritation at L-5. A lumbar myelogram showed almost complete obstruction of the subarachnoid space at the L2–3 level (Fig. 3). The compression was noted to originate posteriorly. A postmyelography CT scan showed soft-tissue density at the posterior aspect of the bone canal, flattening the dural sac (Fig. 4). A herniated nucleus

Fig. 2. Case 1. Photomicrograph showing normal ligamentum flavum on the left blending into increased inflammatory cells and a small area of focal calcification. Degeneration in the elastic fibers is seen in the deeper surface of the ligamentum flavum, which abuts an area of organized hemorrhage. This appearance is one of long-standing ligamentous degeneration in the areas surrounding the hemorrhage. H & E, ×230.

Fig. 3. Case 2. Lumbar myelogram demonstrating almost complete obstruction in the flow of intrathecal contrast material at the L2–3 level.

Fig. 4. Case 2. Postmyelography computerized tomography scan at the L2–3 level showing a posterior extradural mass compressing the thecal sac.

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Fig. 5. Case 2. Photomicrograph showing the characteristic wavy elastic fibers of the ligamentum flavum on the left, blending into a more degenerated ligamentum flavum in the deeper portion of the ligament. This area abuts a well-organized hematoma. There is no distinct plane between the ligament and the hematoma. The changes here are more acute than those seen in Case 1 (Fig. 2). H & E, × 230.

pulposus was not present anteriorly. The cerebrospinal fluid protein level was elevated to 102 mg/dl, but the cell count was normal.

Operation. The patient underwent a total laminectomy at L2–3. The ligamentum flavum was identified as a rounded hemorrhagic mass compressing the dural sac. During removal of the ligamentum flavum, hemorrhagic fluid was expressible from the ligament (Fig. 5). There was no evidence of neoplasm or infection.

Postoperative Course. Postoperatively, the patient had nearly immediate improvement of his lower extremity strength. His follow-up examination showed normal strength and normal bowel and bladder function. He has returned to work.

Discussion

Etiology

There have been a number of reports in the literature describing spontaneous epidural hematomas.1–15 These masses, which can cause compression of the spinal cord, cauda equina, or individual nerve roots, are most common in the cervical and thoracic spine, where they often present as an emergency with rapidly evolving signs of cord compression. A minority of spontaneous epidural hematomas occur in the lumbar canal, where they have a more chronic course and may present with cauda equina or nerve root symptoms resembling either spinal stenosis or a herniated disc.1,3,9 In all instances, these epidural clots are separate from and deep to the ligamentum flavum.

We present two cases of hematoma in the substance of the ligamentum flavum that compressed neural elements in the lumbar canal. These masses were documented at surgery and pathologically verified. In both cases, an episode of minor trauma had occurred some time prior to presentation. Both patients had a history of hypertension treated with beta blockers but neither had a history of abnormal bleeding or liver disease. Neither patient had been administered anticoagulants, although one had undergone nonsteroidal anti-inflammatory drug therapy.

Minor trauma, even sneezing and straining, has been implicated as the origin of spontaneous epidural hematomas, and minor trauma preceded the onset of symptoms in both of our cases. The mechanism proposed to explain the development of epidural hematomas is the transmission of increased intra-abdominal pressure to the epidural venous plexus, causing rupture of these vessels, as observed at surgery by Cooper.2 It is unlikely, however, that a Valsalva-like maneuver could cause bleeding within the ligamentum flavum. The relatively long course of bleeding in our two patients is similar to the course of previously reported chronic extradural hematomas of the lumbar canal.1,3,9 In addition, the hematoma is located dorsal to the dural sac in both conditions. However, the site of the hematoma and the likely mechanisms initiating the clot are quite different.

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The ligamentum flavum is a discontinuous structure from the axis to the sacrum, extending from the mid-laminar level of the superior lamina to the rostrum of the inferior lamina. It extends laterally to blend with the facet capsule. Its main functions are to help maintain an upright posture and resume an upright posture after bending. The ligamentum flavum is largely composed of yellow elastic tissue through which a number of small vessels pass.

Both of our cases appear to involve a flexion of the back at the onset of symptoms. One could speculate that there was a disruption of the above-mentioned vessels, leading to the development of the ligamental hematoma. Alternatively, both patients may have suffered injury to the ligamentum flavum in the past that caused scarring in the ligament and a change from the usual predominantly elastic component of the ligament to a more collagenous structure. During epidural anesthesia, there is significant resistance to injection when the needle is within the substance of the ligamentum flavum; therefore, it is difficult to understand how blood under either arterial or venous pressure could accumulate within the substance of the ligamentum flavum. There must have been internal disruption of the ligament itself that allowed this accumulation.

Neuroradiographic Studies and Treatment

Since both of our patients had symptoms suggestive of nerve root irritation, a radiological workup for a possible herniated disc was performed. Myelography in both instances revealed a posterior compressive mass. In Case 1, the mass was interpreted to be of an aggressive nature since it presented posteriorly and had eroded the lamina. In Case 2, the mass was believed to be
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consistent with a disc fragment that had migrated posteriorly. As magnetic resonance (MR) imaging becomes more common in the initial evaluation of low-back pain with radicular symptoms, more of these lesions may be found. The course in most cases may actually involve slow resolution over time.

The treatment in both of our cases consisted of a decompressive lumbar laminectomy with resection of the hemorrhagic ligamentum flavum. The dural sac was noted to be markedly compressed but resumed a normal configuration after excision of the mass. In both instances, there was no evidence of infection, tumor, or abnormal blood vessels at surgery or on pathological analysis. Both patients have made an excellent neurological recovery.

Conclusions

Ligamentum flavum hematoma is a very rare cause of nerve root compression in the lumbar region. It must be considered in the differential diagnosis in a patient with back or leg pain, especially when trivial trauma was involved. This lesion presents as a posterior compressive mass on myelography and CT and may be better elucidated using MR imaging. The chronic nature of these lesions must be appreciated.

References


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