L5 Root Compression Caused by Degenerative Spinal Stenosis of the L1-L2 and L2-L3 Spaces

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Abstract

Study Design. A severe bilateral L5 root lesion associated with spinal stenosis at L1-L2 and L2-L3 is described.

Objective. To describe clinical findings and the difficulty in obtaining a correct diagnosis of L5 Root Compression.

Summary of Background Data. The disorder reported in this study has not been reported previously. Only one similar case has been described in the literature: an L5 root compression at L1-L2 caused by disc herniation.

Methods. Diagnosis was obtained by using computed tomography scanning, magnetic resonance imaging, and computed tomography myelography. The findings at L5-S1 were
minimal to justify the patient's clinical symptoms, but a detailed study of the upper levels revealed spinal stenosis at L1-L2 and L2-L3, which could have been causing L5 and S1 root compression. A decompressive laminectomy and partial facetectomy in both levels were performed.

Results. The patient's pain and claudication disappeared, and clinical symptoms associated with the right L5 root improved. The left L5 root deficit remained stable.

Conclusion. An unusual case of L5 root compression caused by degenerative stenosis of L1-L2 and L2-L3 is described.

The symptomatology of spinal stenosis varies widely and may include claudication, low back pain, and sciatica, among other disorders. Radicular symptoms usually are related directly to the level responsible for the lesion, e.g., the L5 root normally is compressed in L4-L5 as a result of disc herniation or root-compressing stenosis. L5 root compression at the level of the foramen is less frequent. An instance of L5 root compression caused by an L1-L2 disc herniation has been described recently.

The current study describes a case of L5 root compression caused by spinal stenosis resulting from joint hypertrophy of the L1-L2 and L2-L3 spaces.

Clinical Case

A 69-year-old male patient came to the authors' hospital with low back pain without sciatica, although he had been experiencing weakness in the left foot for 6 months. The patient also had claudication with sciatic pain on the left side 20 minutes after beginning ambulation. Pain remitted after sitting down, and the patient then was able to continue to walk.

Examination revealed normal, painless spine mobility in all directions; a positive Lasegue sign at 60°; and normal, painless hip mobility. Neurologic examination showed a deficit (0/5) in the musculature that was dependent on the left L5 root (extensor of the great toe, extensor of the toes and anterior tibialis). A minimal hypoesthesia was present in the left S1. A mild deficit (4/5) was observed in the anterior tibialis and great toe extensor of the right leg. Osteotendinous reflexes and peripheral pulses were present and symmetric.

The plain radiography study revealed loss of lumbar lordosis and multiple discopathy from T12 to S1, with anterior osteophyte formations at L3-L4 and L4-L5.

Figure 1. Lateral radiograph showing a multiple discopathy with osteophyte formations.
The last two mobile spaces were studied using computed tomography scanning (Figure 2), which revealed minimal stenosis at L4-L5 and left foraminal disc herniation at L5-S1.

Figure 2. Preoperative computed tomography scan. A, At L4-L5, minimal lateral stenosis is shown. B, At L5-S1, a left foraminal herniation is demonstrated.

An electrophysiologic study was performed, and results confirmed those of the clinical examination, revealing major and minimal involvement of the L5 and left S1 roots, respectively.

In the authors' opinion, the computed tomography findings did not explain the patient's symptoms fully; therefore, it was decided to complete the study using magnetic resonance imaging, which revealed:
discopathy with the anulus bulging in all lumbar spine segments (Figure 3);

Figure 3. Lateral magnetic resonance image showing a discopathy with the anulus bulging in all lumbar spine segments.

obliteration of the right lateral recess at L2-L3 and at the extraforaminal level;

degenerative changes of the facet joints at all levels; and

normal canal dimensions at L4-L5 and L5-S1. Finally, a tomographic myelography study and a computed tomographic myelography study were performed (Figure 4), the results of which confirmed two severely stenotic areas between L1-L2 and L2-L3, which impaired the passage of contrast medium. Stenosis appeared to be caused by facet joint hypertrophy. Compression seemed to be symmetric at L1-L2, while appearing to affect mainly the right side at L2-L3.


Because the deficit persisted and the patient experienced continued claudication, with a reduction of his walking range after less than 5 minutes, decompressive surgery was performed by means of a decompressive laminectomy and partial facetectomy at the L1-L2 and L2-L3 spaces, without discectomy or arthrodesis.

There were no postoperative complications. Four years later, the patient's neurologic status was similar to his preoperative condition on the left side, but the right side was normal neurologic on exploration. At the time of this report, the patient was using a modified Rancho de Los Amigos splint (PRIM, SA, Madrid, Spain) on his left foot for walking, and was not experiencing ambulatory claudication, lumbar pain, or radicular pain. Postoperative
radiologic studies have shown no evidence of postoperative instability.

Discussion

Clinical symptoms of an L5 root compression are classic and consist basically of a positive Lasegue sign and a greater or lesser degree of motor deficit, pain, paresthesia, or sensitivity deficits in the root-dependent area. Based on the results of clinical examination, the physician must diagnose the presumed source of compression, which, in the case of the L5 root, usually is located at the L4-L5 space. In contrast, compression of the L1-L2 and L2-L3 spaces affects the roots of L2 and L3, respectively, producing crural pain or a positive femoral nerve stretch test and an alteration of the patellar reflex. These findings are particularly true of the L5 root, but are less so for the superior roots. A compression at the thoracolumbar level hinge may produce complex symptoms because of the proximity of the medullary epiconus and conus, whose anatomic location varies, although their most frequent level inferior is L1-L2.

On a strictly theoretical basis, there is no reason why a lateral compression of the dural sac containing medullary and radicular tissue should damage the root of L5, because the caudal-most roots tend to take up a more central position and therefore should become compressed as a result of central lesions. Such compression occurred in a case described by Shirado et al., in which a central disc herniation produced an L5 root lesion at L1-L2. Perhaps the only explanation may be found in Porter's hypothesis, in accordance with which a double-space compression of the cauda equina can cause venous pooling between the two levels of compression, which in turn would lead to an inadequate oxygen supply at the level of the roots within the dural sac. By the same hypothesis, the existence of a compressed left L5 root in the L5-S1 foramen would be an additional contributing factor that might aggravate the symptomatology. Moreover, on myeloscopic examination, the diameter of vessels of the cauda equina has shown changes in patients with intermittent claudication and canal stenosis in accordance with postural alteration.

Based on the computed tomography findings in the current case (Figures 1 and 2), the authors believe that it is possible that the root of L5 was compressed at the level of the left L5-S1 foramen. However, such foraminal involvement did not seem significant enough to justify the patient's clinical symptoms; therefore it was decided to complete the study by means of magnetic resonance imaging and computed tomography myelography examinations, the results of which confirmed the presence of stenosis at the L1-L2 and L2-L3 spaces. The authors of the current study believe, in accordance with Shirado et al., that the utility of axial magnetic resonance or computed tomography images should not be overestimated, because to be evaluated correctly, these sections must be completely parallel to the disc, which is especially difficult in cases of degenerative scoliosis. These apparent lesions of the lower levels, which would seem, if overestimated, initially to justify the patient's symptoms, can cause incorrect surgical procedures. Furthermore, conjoined roots or the presence of vascular structures in the area can produce images similar to those of a disc herniation. We must also bear in mind the large amount of pathologic findings in asymptomatic individuals, ranging from 35%, regardless of age, when using computed tomographic examination techniques, to 57% when using magnetic resonance imaging in asymptomatic populations of individuals over 60 years of age.

For the surgical technique, the authors of this study chose a laminectomy associated with partial removal of the facet joints and release of the lateral recesses without discectomy, because disc herniation was not present according to the imaging studies or during surgery. Arthrodesis was not considered, because no disectomies had been performed, and the patient had no preoperative spondylolisthesis. Both of these factors are associated with an
increase in postoperative instability. Furthermore, displacements are rare in cases associated with discopathy and osteophyte formation, and postoperative instability occurs in 2-31% of patients with no preoperative spondylolisthesis. Had there been a central disc herniation compressing the nervous structures at L1-L2, the authors would have favored an anterior discectomy and decompression and arthrodesis.

References


Key words: root compression; spinal stenosis