Symptomatic lumbar stenosis following fusion using sublaminar hooks

Case report

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A case of postfusion lumbar stenosis caused by the presence of sublaminar hooks is described. The patient was a 52-year-old man who 11 years previously had undergone lumbar fusion with Harrington rod instrumentation for a traumatic L-2 vertebral body fracture. Postoperatively, he developed progressive low-back pain, neurogenic claudication, and significant lower-extremity weakness and atrophy. Upon radiological examination, he was found to have high-grade lumbar stenosis at the level of the caudal sublaminar hooks. The instrumentation was removed and the area of radiological stenosis decompressed. Clinically, both the patient's pain and motor deficits resolved and, on postoperative imaging, the stenosis was relieved. Thus, despite other areas of persisting pathology, it is concluded that the stenosis occurring at the level of the caudal sublaminar hooks contributed to the patient's symptoms. Although not a common cause of postfusion stenosis, the presence of instrumentation in the proximity of neural elements must be considered as an etiology for neurological dysfunction.

KEY WORDS • internal fixation • lumbar fusion • spinal stenosis • instrumentation

The increased popularity and success of internal fixation devices to enhance fusion of the thoracolumbar spine has provided a new set of patients with postinstrumentation postfusion lumbar stenosis. Although a number of other common causes have been described, this case is an exceptional example of stenosis induced by the placement and presence of sublaminar hooks.

Case Report

This 52-year-old man presented in 1992 for evaluation of back and lower-extremity pain. In 1981, he had sought medical treatment for a traumatic L-2 vertebral body fracture and thoracolumbar back pain. He subsequently underwent partial resection of the L-2 vertebral body, anterior fusion with fibular bone graft to replace the partial resection, complete L-1 and L-2 and partial L-3 laminectomies, placement of Harrington rods from T-11 to L-4, and posterior spinal fusion from T-11 to L-4 using the previously removed laminae and spinous processes over decorticated transverse processes. Postoperatively, the patient did well until 1 year later when he was involved in a motor-vehicle accident, at which time he presented with pseudarthrosis and breaking of both Harrington rods. He underwent reoperation; the sublaminar hooks were not changed, but the Harrington rods were replaced, and an autologous iliac bone graft was used to fuse the area of pseudarthrosis. The patient initially improved and his back pain diminished, but within a few months he began experiencing pain in his lower back, with increasing weakness of both legs and increasing difficulty walking long distances.

Examination. The patient came to our Neurosurgery Service in 1992, complaining of back and bilateral lower-extremity pain, accompanied by pain radiating down the posterior aspect of the left leg on ambulation, which had become very limited. On examination, he had a tender lower lumbar spine, not related to flexion or extension. He had weakness in both dorsiflexion and plantar flexion bilaterally (4-/5), mild weakness in knee extension on the left (4+/5), and severe bilateral gastrocnemius atrophy. In addition, he had L-5 and S-1 sensory loss in both legs, with decreased ankle reflexes. The patient had difficulty arising from a chair due to pain and weakness; ambulation was slow, and limited to 40 to 50 feet before he was forced to stop and rest. He denied bowel or bladder dysfunction.

Because of the internal fixation device, adequate magnetic resonance (MR) imaging could not be per-
Lumbar stenosis due to sublaminar hooks

![Image](image_url)

**Fig. 1.** Preoperative lumbar myelograms. *Left:* Lateral view demonstrating severe stenosis at the level of the caudal sublaminar hooks (L4–5), with concomitant angulation at the level of the original vertebral body fracture (L2–3). *Right:* Anteroposterior view demonstrating both levels of dye block in addition to a significant fusion mass at the level of the fracture (arrows).

formed. Plain films and computerized tomography (CT) myelography revealed a complete block distal to the caudal sublaminar hook at the level of the L4–5 disc space (Fig. 1 left). There was concomitant kyphosis at the level of the original fracture (L2–3), with significant dye obstruction; however, this was not as severe as the area of previously mentioned stenosis, nor was it compatible with the patient’s more distal symptoms and absence of upper lumbar radicular symptoms. Radiologically, there appeared to be a large fusion mass at the level of the fracture (Fig. 1 right), and it was believed that this construct provided adequate stability to allow removal of the fixation device.

**Operation.** Intraoperatively, a strong dense fusion mass was encountered, which had grown partially around both Harrington rods and all four sublaminar hooks. With a high-speed drill, enough bone was removed to allow access to the rods so they could be cut and to the hooks so they could be torqued out of the fusion mass and from underneath the laminae. After bone and laminae were removed centrally at the L-4 level, a very thickened ligamentum flavum was encountered, with the remaining imprints of the overlying sublaminar hooks. The subsequent deflection of the ligamentum flavum anteriorly into the thecal sac revealed an obvious site of pathologic stenosis. The ligamentum flavum and a small amount of scar were removed, allowing for substantial decompression of the sac at this level.

**Postoperative Course.** Postoperatively, the patient did well. At his 1-month follow-up examination, he had regained almost complete strength of his lower extremities with the exception of some left-sided weakness in dorsiflexion and plantar flexion (4+/5) as well as knee extension (5-/5). His back and bilateral lower-extremity pain had diminished from excruciating to occasionally noticeable. He was able to arise and walk normally, with minimal lumbar pain. A postoperative MR image confirmed excellent decompression at L4–5, correlating well with the patient’s clinical progress (Fig. 2). Since that time, he has continued to improve slowly without recurrence of his symptoms.

**Discussion**

The use of internal fixation devices in the lumbar spine has become increasingly popular in recent years. Fracture stabilization, fracture site decompression, faster mobilization, and shorter hospitalization have all been noted benefits of Harrington rod instrumentation. Although preinstrumentation postfusion stenosis by the pressure of the fusion mass on the thecal sac was documented as early as 1933, the modern problem of stenosis with progressive neurogenic claudication and weakness as a result of the instrumentation itself has not been well described.

Stenosis occurring after lumbar fusion as a result of a postoperative herniated disc, fusion mass hypertrophy, pseudarthrosis, instrumentation failure, and progressive degenerative spondylosis have all been documented. McAfee and Bohlman and others have described additional neurological complications from Harrington rod placement, including nerve root compression from hook migration into the neural foramen or spinal canal, fracture of the hook-lamina construct, and erosion of the sublaminar hook through the lamina with subsequent neural compression. Similar to the case presented, Eismont and Sime-
one reported a patient with scoliosis who developed symptomatic stenosis from anterior hypertrophy of the laminae involved in the post-Harrington rod fusion. However, symptomatic spinal stenosis due to the presence of sublaminar hooks has not been well described.

Since postinstrumentation radiological studies may be difficult to interpret, particularly in cases of trauma with multiple levels of potential symptomatic pathology, the patient's symptoms and physical examination are important in successfully choosing the causative lesion. In this case, the absence of upper-limb weakness or atrophy with the presence of low-back pain, diffuse lower lumbar radiculopathy, and bilateral calf atrophy made the area of the previous surgery and vertebral body fracture an unlikely source for this man's pain and weakness, despite significant angulation and attenuation of contrast medium on the myelogram. Although nonspecific, the absence of significant compromise of the S-1 roots in the L5–S1 lateral recesses also pointed to compression of these roots at the level of the L4–S stenosis as the cause of these findings.

The diagnosis of neurogenic claudication, that is, back pain with progressive lower-limb pain and fatigue with standing or walking, is believed to be due to the constrictive nature of the circumferentially compromised spinal canal, which is exacerbated in the upright position. In contrast, the angulation in this case at the level of the original compression fracture, although radiologically significant as an area of the sacral impingement, did not correlate with the patient's symptoms or with this notion of lumbar stenosis and neurogenic claudication. Determining the appropriate level to decompress is of importance not only in relieving the patient's symptoms, but in maintaining lumbar stability. Extensive multilevel decompression and destruction of a functional fusion construct, with concomitant removal of the fixation device, may be disastrous in rendering the patient's spine unstable.

The placement of sublaminar hooks is usually uneventful and rarely associated with direct neural injury. However, in a previously narrow canal, the loss of space associated with placement of sublaminar hooks could significantly compromise the thecal sac or exiting nerve roots. It would then be difficult to predict any future hypertrophy of the ligamentum flavum or development of reactive scar tissue and eventual neural compression. In determining the appropriate fixation device in the thoracolumbar spine, assessment of preoperative imaging for pre-existing stenosis might aid in avoiding the type of postfusion stenosis presented here. The need for axial distraction and accommodation for the normal thoracolumbar curve in many of these conditions could perhaps be addressed with other available fixation devices, such as pedicle screw-based instrumentation.

The postfusion patient with sublaminar hooks and progressive neurological deficits should have a complete workup, including plain films and CT myelography, to ensure that spinal stenosis for reasons other than those frequently encountered have been addressed. In conjunction with a focused neurological examination and specific history, the possibility of spinal stenosis caused by the presence of instrumentation near neural elements must be entertained. This may avoid erroneously operating on what appears to be symptomatic stenosis at the fusion site and negating an otherwise well-formed stable fusion for which the placement of such instrumentation was originally intended.

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References


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