

## Case Report

# Ligamentum Flavum Hematoma in the Adjacent Segment after a long Level Fusion

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Ligamentum flavum hematoma (LFH) is a very rare condition of dural compression; most are observed in the mobile cervical and lumbar spine regions. A 67-year-old man who had a long level interbody fusion at L3-S1 four years ago presented with symptoms suggestive of dural compression. Magnetic resonance imaging showed a posterior semicircular mass located at the adjacent L2-L3 level. After decompression of the spinal canal and removal of the mass lesion, pathological examination of the surgical specimen revealed a hematoma within the ligamentum. The patient fully recovered to normal status after surgery. Here, we report our experience with a LFH in the adjacent segment after a long level fusion procedure and discuss the possible associated mechanisms.

**Key Words :** Ligamentum flavum · Hematoma · Adjacent segment.

## INTRODUCTION

Ligamentum flavum hematoma (LFH) is a very rare condition which may cause myelopathy, or nerve root compression. Hypertrophy, calcification, or infolding of the ligamentum flavum occurs more often than hemorrhage because of the poor vascularity of this area. Although, LFH may occur from the cervical to sacral vertebrae, most prior cases have been reported in the mobile cervical and lumbar areas<sup>2)</sup>. To date, there has been no report of a LFH in the adjacent segment after a long level fusion procedure in the literature. Here, a patient with LFH that developed at an adjacent upper segment after a long level fusion procedure is reported with a review of the literature.

## CASE REPORT

A 67-year-old male patient with low back pain presented with a seven-day history of abrupt onset of pain in the right leg. Four years ago, back pain and neurogenic claudication developed and he was diagnosed with multiple spinal stenosis. The patient had a long level posterior lumbar interbody fusion procedure at the L3-S1 level. The patient was not able to walk by himself and com-

plained of weakness in both lower extremities, especially in the right leg. Before admission the patient sought medical consultation and began nonsteroid anti-inflammatory medication. However, the pain gradually worsened despite medication, and he was hospitalized seven days later. The neurological examinations showed no restrictions on the straight leg raising test. However, he showed slight weakness in both dorsiflexor and plantar flexor muscles, more on the right side. The patient had no complaints of urinary or rectal incontinence, but did have decreased rectal sphincter tone. At first, we suspected adjacent segment disease, especially cauda equina syndrome caused by acute disc herniation or spinal stenosis. However, magnetic resonance imaging (MRI) at the L2-L3 level showed a round, T2 hyperintense mass at the posterior aspect of the spinal canal displacing the dural sac anteriorly and resulting in severe stenosis of the spinal canal. On MRI with gadolinium-diethylenetriamine penta-acetic (Gd-DTPA), the outer membrane of the mass at the dorsal right side of the spinal canal was enhanced (Fig. 1). Considering the occurrence and course of the disease, it was difficult preoperatively to distinguish a LFH from a spontaneous epidural hematoma, ganglion, or synovial cyst. The patient underwent surgery for decompression of the spinal canal and removal of the mass lesion. After an L2 bilateral partial laminectomy, the thickened ligamentum flavum was exposed. The ligamentum flavum was adhered to the dural sac, and contained a dark brownish fluid. The remaining ligamentum flavum was removed to decompress the spinal canal. Pathological examination of the specimen revealed an old hemorrhage and degenerative changes within

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the ligamentous fiber. There was no disc material, infection, or neoplasm (Fig. 2). The patient's postoperative course was uneventful. Immediately after surgery, the patient became asymptomatic except for mild numbness in the right leg.

## DISCUSSION

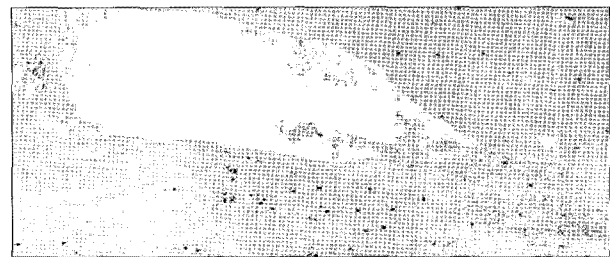
The ligamentum flavum is a discontinuous structure from the axis to sacrum; it helps to maintain an upright posture and resume an upright posture after bending. Due to its poor vascularity, only a few small vessels pass through it and intraligamentous bleeding is uncommon<sup>1)</sup>. To date, the pathogenesis of LFH has been suggested to be due to trivial trauma or stress as a result of increased intra-abdominal pressure transmitting excessive pressure to the very small, thin and irregular blood vessels in the ligamentum flavum<sup>6)</sup>. Increased abdominal pressure transmitted to the epidural space results in spinal epidural hypertension, which can cause a partial tear in the degenerated ligament that can result in an intraligamentous hemorrhage. The cervical and lumbar spine receives transmitted pressure to the epidural space due to their spinal mobility. Therefore, most intraligamentous hemorrhages are observed in the mobile cervical and lumbar spine regions<sup>2)</sup>. However, although most patients might have minor and trivial injuries before the onset of symptoms, they usually cannot remember the exact time of injury due to the interval between injury and symptom onset<sup>2)</sup>. The current patient also had no recent trauma history.

It is possible that another contributing factor in our patient might have been the cause, such as alteration of the biomechanical stress on adjacent upper segments after the solid fusion procedure. Nagata et al.<sup>5)</sup> conducted a biomechanical study on canine cadaveric spines and reported that lumbosacral motion and facet loading on adjacent levels increased significantly after the immobilization of segments in proportion to the number of immobilized segments. Using an *in vivo* dog model, Dekutoski et al.<sup>1)</sup> also demonstrated hypermobility of the facet joints at the upper free segment. As well as biomechanical alterations, degenerative spondylotic changes were also demonstrated. Greater axial biomechanical stress or force appears to be loaded onto the ligamentum flavum and the facet joint at the adjacent level compared to the normal spinal alignment. As a result, the ligamentum flavum of the adjacent level might be influenced by excessive stress and degenerative changes might be enhanced. These continuous stress and degenerative changes appear to have a strong influence on the development of LFH.

The differential diagnosis of LFH includes a synovial cyst, ganglion cyst, and disc herniation. MR imaging typically shows a well-circumscribed extradural cystic lesion as isointense or slightly hypointense with peripheral enhancement after adminis-



**Fig. 1.** Magnetic resonance images reveal severe spinal stenosis and extradural mass lesion at L2-L3 level. A : T2 weighted magnetic resonance image shows an extradural mass at L2-L3 level with severe stenosis. B & C : T1-weighted sagittal and axial magnetic resonance images with gadolinium enhancement demonstrate peripheral enhancement of the mass (arrows).



**Fig. 2.** Photomicroscope of the surgical specimen reveals hemosiderin pigmentation and hemorrhage (arrow) within the degeneration of the collagenous fibers of the ligament (H & E  $\times 100$ ).

tration of Gd-DTPA on T1-weighted images, and hyperintense images on T2-weighted images<sup>3,6,7)</sup>. Although a herniated disc can migrate to a posterolateral position, they are much more common in the anterior part of the spinal canal. Synovial cysts are typically observed in the posterolateral spinal canal or lateral recess adjacent to the facet joint. However, in spite of this information, an accurate diagnosis is difficult even with MR images. In this case, the lesion was confirmed after histological examination. Pathologically, synovial cysts also contain hemorrhage, but they remain clearly outside the ligamentum flavum and are usually attached to the facet joint, whereas LFH is located within the ligament. Evidence of this attachment may help diagnose this lesion. It has been known that prognoses for LFH following surgery are good. We believe that LFH was formed and compressed in relatively long-standing time, so cord had sufficient time to compensate.

## CONCLUSION

This is the first report of LFH that occurred in the upper adjacent segment after a solid lumbar spine fusion procedure. The greater axial biomechanical stress on the adjacent segment may have influenced the pathogenesis of LFH. Although it is unusual we should keep in mind the possibility of this rare complication after solid fusion procedures.

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