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Case Presentation

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Thoracic Spinal Stenosis from Calcified Ligamentum Flavum Co-Existing with L5-S1 Foraminal Stenosis - A Case Report and Literature Review

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Abstract

Calcification of the ligamentum flavum (LF) is a rare cause of spinal stenosis. The process can occur at any level in the spine, often presents with local pain or radicular symptoms, and is a distinct process from ossification of the spinal ligaments in pathogenesis and treatment approach. Few case reports have described multiple level involvements in the thoracic spine that results in sensorimotor deficits and myelopathy. We reported a 50-year-old male who presented with progressive sensorimotor deficits from T10 distally that resulted in complete sensory deficits and decreased lower extremity strength on the left. Computed tomography (CT) and magnetic resonance imaging (MRI) demonstrated calcification of the ligamentum flavum from T10-11 and T11-12 with foraminal stenosis of L5-S1 on the left. He underwent endoscopic assisted excision of calcified ligamentum flavum. Postoperatively, he had complete motor strength recovery and was discharged home for outpatient physiotherapy. His residual sensory deficits continued to improve with time after decompression and excision of the calcified ligamentum flavum and decompression of the L5-S1 foramen. This case is unique in that the calcific processes involved multi levels of the thoracic spine. The patient had dramatic improvement in his symptoms following resection of the involved responsible levels. Keywords: Calcification–physiologic, ligamentum flavum, spinal stenosis, spine, thoracic vertebrae, foraminal stenosis. endoscopic-assisted spine surgery.

Introduction

Spinal stenosis is the narrowing of the spinal canal and compression of the nerve roots or spinal cord resulting in myelopathy or/and radiculopathy. Stenosis can involve the cervical, thoracic, or lumbar spine. Spinal stenosis can involve 3 regions: the central area, lateral recess, or neural foraminal region. The etiology of stenosis can be either congenital or acquired. Acquired stenosis can be degenerative, traumatic, iatrogenic, or neoplastic in nature **[1,2]**. Uncommon cases of stenosis derive from ossification of the posterior longitudinal ligaments in the cervical spine, a condition that is commonly reported in Japan **[3]**. However, calcification of the ligamentum flavum is a separate entity from ossification of the posterior longitudinal ligaments **[4]** and most commonly occurs in the cervical and lumbar spine **[5-9]**. Not many cases of calcification of the ligamentum flavum in the thoracic region have been reported **[10-12]**. We report a case of spinal stenosis resulting from calcification of the ligamentum flavum of T7-8, T10-11 and T11-12 co-existed with L5-S1 left side foraminal stenosis and resulting in lower extremity weakness and sensory deficits.

Case Presentation

A 50-year-old male presented to clinic for consultation for weakness, pain and tingling left leg for last 6 months and worsened in last 2 months. The patient reported that sensory changes from frontal and lateral thigh down to lateral and plantar foot on the left with associated strength deficits, had been progressing for recent 2 months. He had associated mid-back and left chest wall pain but no cervical spine pain. The CT scan of the thoracic spine showed calcification of the ligamentum flavum from T10-12. He got ipsilateral L5-S1 foraminal stenosis **(Figure 6)** that was responsible for his lower leg and lateral, plantar foot neurological symptoms. His associated thoracic spinal stenosis was severe. However, the hard disc prolapse at T7-8 which was not responsible for his clinical symptoms **(Figure 1)**. Sagittal C7 to L3 (left) and axial (right) CT scans (T10-12) of the thoracic spine showing segmental calcific lesions causing severe spinal stenosis at T10-12 **(Figure 2 and 3)**. MRI showed disc prolapse (arrows) at level of T7-8 on the right which was irresponsible his left side clinical symptom **(Figure 4)**. T2 weighted MRI images (left) showing multiple segmental calcifications of the ligamentum flavum in thoracic spine, more severe spinal stenosis at the T10-12 level (right) **(Figure 5)**. Given that spinal stenosis was severe at T10-12, moderate at T7-8 caused by hard disc herniation that was not responsible his clinical symptoms, the decision was made to perform a posterior laminectomy and resection of the calcified ligamentum flavum from T10-T12 without fusion. The patient's spinal cord was found to be significantly compressed from T10-T12 intraoperatively from calcified ligamentum flavum that was adherent to the duramatter. Intraoperative samples of the ligamentum flavum were sent to pathology, and the report was consistent with calcification and reactive bony processes. L5-S1 foraminal stenosis **(Figure 7)** was decompressed with endoscopic assisted enlargement. He was discharged home with outpatient physiotherapy. He was seen at 2 weeks, 6 weeks, 3 months and 6 months postoperatively. At her 6-month follow-up, the patient was ambulatory independently without limping and had improvement in his sensation below T10 and intermittent claudication was improved significantly with walking distance more than 5 kilometers. Postoperative ODI score, NRS score, JOA score, ASIA neurological

function score, and SF-36 score were significantly better than preoperative scores.

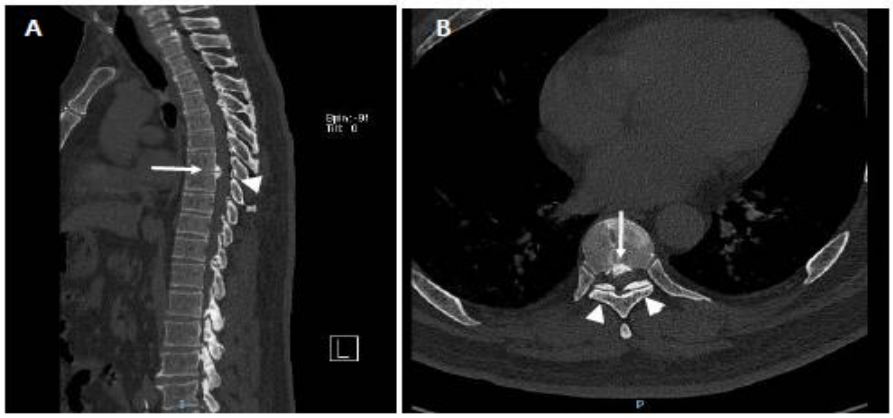

Figure 1: CT scan showed T7-T8 hard disc prolapse for calcified disc osteophytes (arrows) and calcified ligament flavum bilaterally (arrow heads).

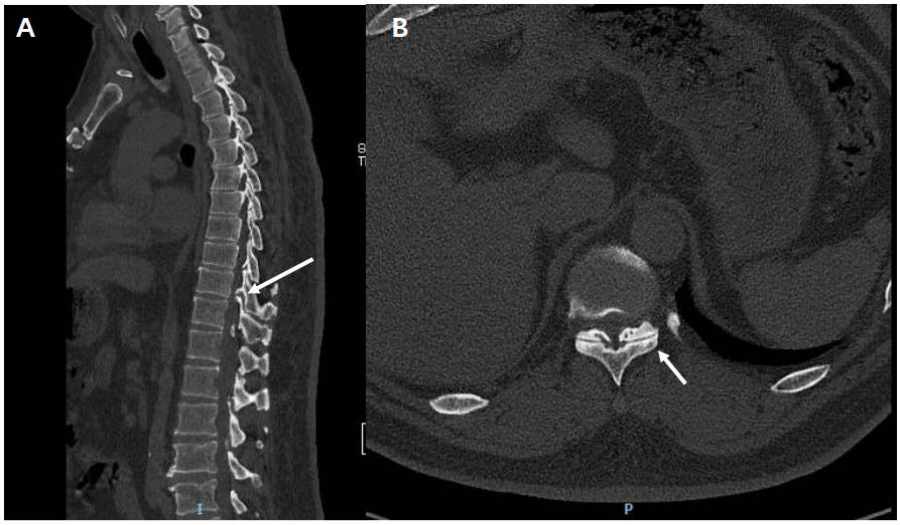

Figure 2: Preoperative computed tomography imaging demonstrates clearly visible ossification of the ligament flavum and compression of the spinal cord. Sagittal (left) and axial (right) CT images of the spine showing the ligamentum flavum ossification causing severe spinal stenosis at T10-T12 levels.

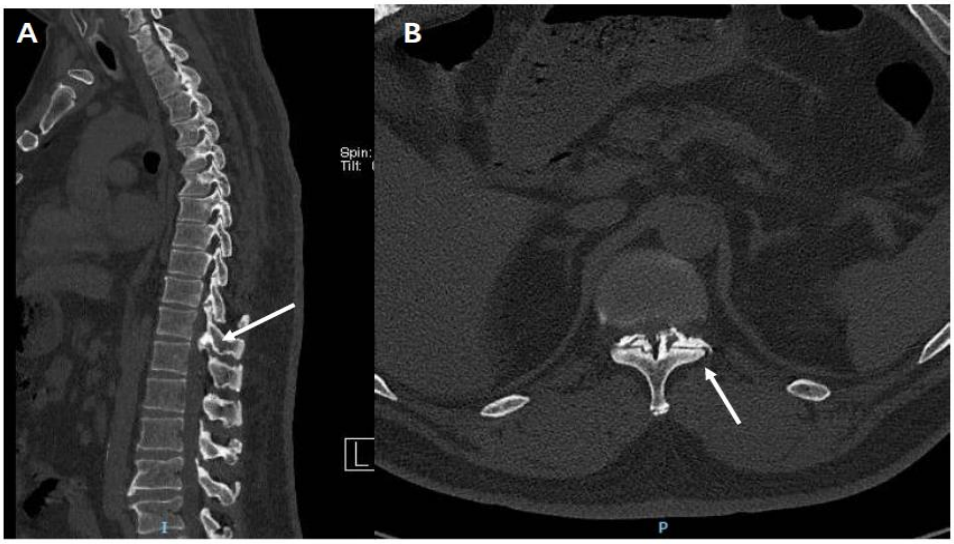

Figure 3: Preoperative computed tomography imaging demonstrates clearly visible ossification of the ligament flavum and compression of the spinal cord. Sagittal (left) and axial (right) CT images of the spine showing more severe ligamentum flavum ossification causing significantly spinal stenosis at T11-T12 levels.

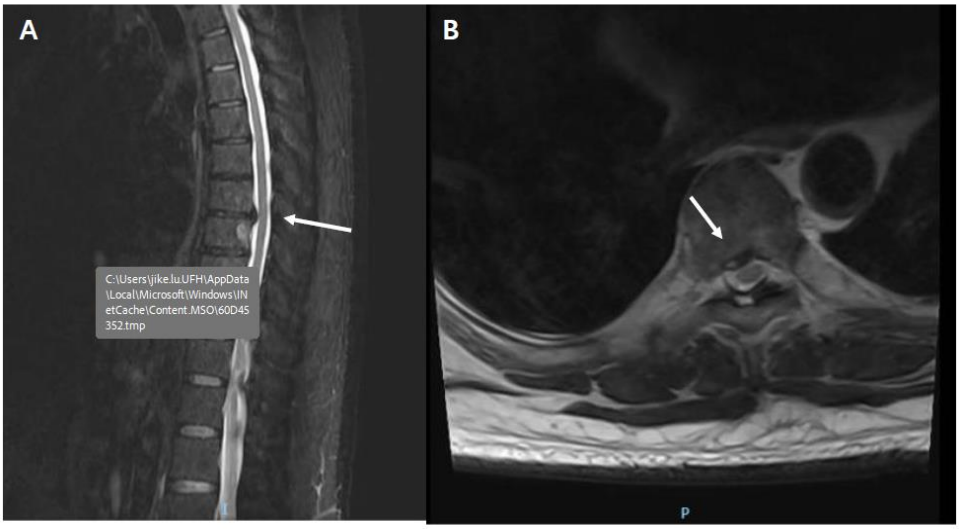

Figure 4: MRI showed disc prolapse (arrows) at level of T6-7 on the right which was irresponsible his left side clinical symptoms.

Figure 5: Thoracic-level MRI findings were consistent with the CT scan obtained earlier. T2-weighted sequence of MRI sagittal images showing hypointense signal in the posterior portion of the vertebral canal of T10-12. Axial views demonstrating spinal cord compression on the T10-T11 segment suggesting compressive myelopathy.

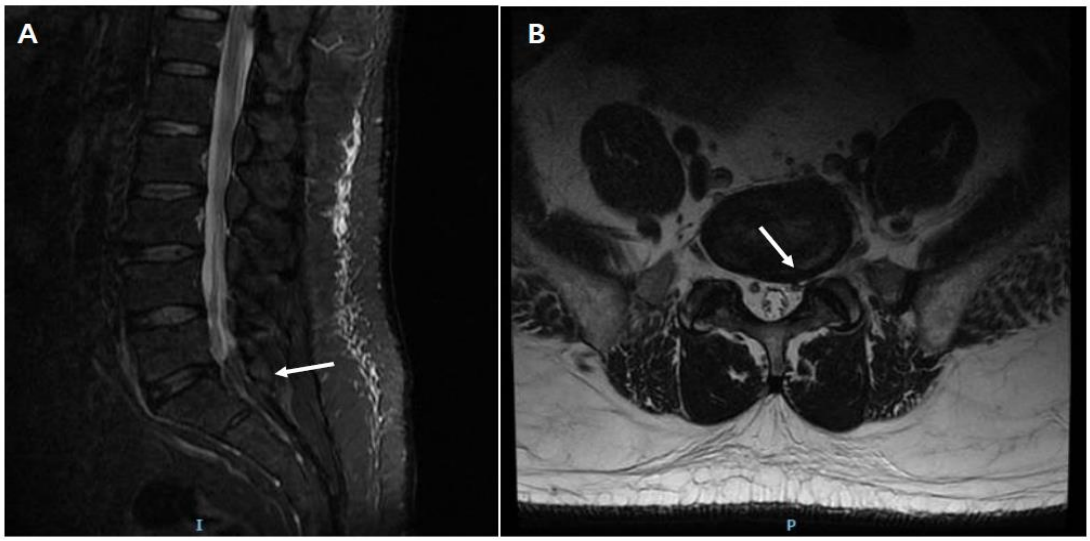

Figure 6: MRI showed L5 S1 foraminal stenosis caused by disc bulging and thickening of ligament flavum (arrows).

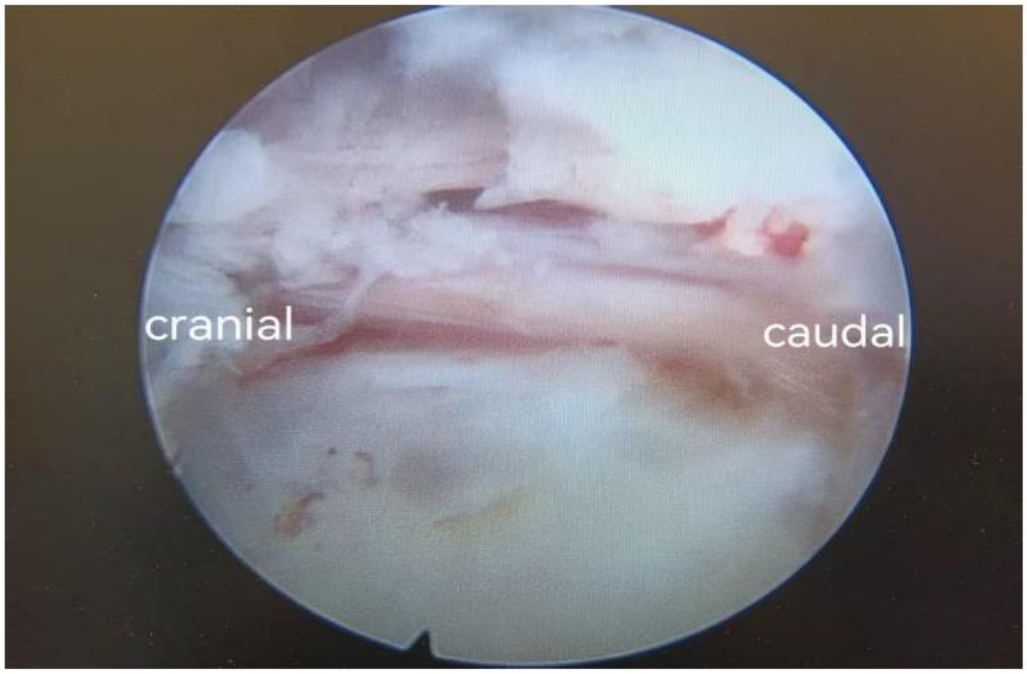

Figure 7: Remove calcified ligamentum flavum and fully expose nerve root.

The Endoscopic Techniques

The operation was performed in the prone position on a radiolucent table under general anesthesia. An anteroposterior radiograph as obtained with a syringe needle to confirm the incision level. After verification of the incision level, the superficial surgical site was marked just lateral to the midline spinous processes. A 1-cm skin incision was made, and the superficial fascia and deep fascia were cut. The obturator was then introduced and used to feel the inferior border of the upper laminae, the superior edge of the inferior laminae, and the medial point of the facet joint. Stepwise expansion tubes were used to extend the soft tissue. The beveled working sleeve was inserted, the expansion tubes were removed, and then the endoscope system was introduced through the working cannula.

Further procedures were done under continuous visual control and irrigation through the intra-endoscopic working channel. The overlying soft tissue was coagulated with the Radio Frequency (RF) probe and removed with endoscopic forceps to expose the anatomical structure of the lamina, and articular process. Once the osseous structures were exposed, the lower border of the superior lamina was drilled with a circular saw on the tip of the cannula until the attachment of the ligamentum flavum was exposed, and the circular saw on the trocar was directed laterally toward the facet joint. The upper boundary of the inferior lamina and articular processes on the same side were excised, and the basal part of the spinous process was excised by a Kerrison rongeur until the calcified ligament flavum boundary of the ligamentum flavum was clearly revealed.

Starting from the midline inter-laminar space and layer-by-layer thinning by an endoscopic drilling-saw system. Thoracic full-endoscopic unilateral laminotomy for ossification of the ligamentum flavum. The lower half of the upper lamina was

removed, followed by the upper half of the lower lamina and the base of the spinous process. Over-the-top technique to decompress the contralateral margin of the spinal cord and then extend it to the cranial and the caudal side. Remanent laminae of the ossified ligament flavum were removed by endoscopic Kerrison punches. Finally, decompression was then extended to the cranial and the caudal side to remove all calcified ligament flavum by forceps and punches till nerve roots visible **(Figure 7)**. The patient was able to get out of bed 6 hours after surgery and discharged the following day.

Discussion

Calcification of the ligamentum flavum is not a commonly seen and separate entity from ossification of the posterior longitudinal ligament. Ossification of the posterior longitudinal ligament involves ossification or bony formation of the posterior longitudinal ligament through endochondral bone formation. Ossification involves a process of laying down bone through osteoblasts **[9]**. Calcification of the ligamentum flavum is a process of unknown pathogenesis **[4]**. A proposed theory is that repetitive microtrauma to the ligamentum flavum initiates the calcific deposition. This microtrauma leads to neovascularization, permeability, and hypertrophy of the ligamentum flavum **[13,14]**. The majority of cases report segmental involvement at just a few levels **[5-12]**. Our case is unique in that our patient presented with multiple levels of involvement from T7-8, T10-T12 and progressive myelopathy that eventually led to ambulatory deficits and severe sensorimotor deficits. To our knowledge, this case is a rarecase report showing multiple levels of involvement of the thoracic spine with a myelopathic presentation and co-existing L5-S1 foraminal stenosis. Histology study showed that there is a variety of pathological conditions affecting the LF, including hypertrophy, calcification, and ossification. In cases of hypertrophy, the normal fibrous structure of the LF is replaced by hypertrophic fibrous tissue containing numerous fibrocartilaginous cells with abundant matrix **[21]**. However, the calcification consists of thickening of the deep layers of the ligament flavum whereas the corresponding ossification affects only the most superficial layers **[22]**.

Ossification or calcification of the ligament flavum is an idiopathic endochondral calcification that affects approximately 20% of individuals of Eastern origin and over 60 years old **[23]**, being rare in those who are not of Eastern origin and in those who are younger than 50 years of age. In fact, the etiology of this calcification/ossification remains unclear. Although the role of initial trauma has yet to be demonstrated, it is known that such trauma can worsen the neurological symptoms **[12]**. Ossification or calcification of the ligament flavum is most common at the lower thoracic level, being uncommon in the lumbar region (as shown in our case) and rare in the cervical region **[23]**. The region most often affected in women is the cervical region, whereas it is the thoracic and lumbar regions in men **[23]**. The typical clinical presentation is chronic myelopathy with cervicothoracic cord compression, which can be accompanied by radiculopathy and sphincter dysfunction. Ossification or calcification of the ligament flavum can be associated with Paget's disease, rickets, chondrocalcinosis, ankylosing spondylitis, hypoparathyroidism, skeletal hyperostosis, obesity, diabetes, hyperinsulinemia, and disorders of calcium metabolism **[23]**. None of the above-mentioned conditions were confirmed in our

case reported here. Although X-ray findings can suggest the diagnosis, it generally has limited value. A CT scan can reveal a calcified/ossified extradural mass located under the vertebral lamina and protruding into the intervertebral foramen, with a loss of extradural fat [23]. In determining the degree of spinal cord compression and the characteristics of ossification, MRI is extremely useful, the ligament flavum appearing hypointense on T1- and T2-weighted images. The typical indentation is best seen on T2-weighted images [11]. The use of CT and MRI in combination provides information that is more accurate and useful for treatment planning than does the use of either alone [20,21].

Miyasaka et al stress the importance of assessment and monitoring through x-ray, CT, and MRI to differentiate ossification from calcification for clinical management [15]. Ossification is often continuous with the lamina and often becomes adherent to the dura, making laminectomy and decompression difficult [16]. Calcification of the ligamentum flavum is often missed or the diagnosis is delayed. Our case highlights the utility of surgical decompression for myelopathic symptoms when conservative management has failed. Unlike open surgeries or the micro-endoscopic technique, full endoscopic surgery is performed under continuous irrigation fluid for clearer vision and reduced bleeding. Liu et al. performed percutaneous endoscopic surgery using the bilateral translaminar osseous channel approach to treat thoracic ossified ligament flavum combined with disk herniation, and obtained sufficient decompression of the spinal cord with minimum trauma [17]. Wu et al. adopted endoscopic visual trepan decompression to treat single-segment ossified ligament flavum and found that it had advantages of reduced trauma, faster recovery, and lower cost than the technique of posterior spinal canal decompression [18]. An et al. adopted percutaneous full endoscopic posterior decompression for the treatment of thoracic ossified ligament flavum at a single level and obtained satisfactory clinical results. They found that endoscopic surgery is a safe, effective, and reliable method for ossified ligament flavum [19].

The treatment of ossification or calcification of the ligament flavum generally consists of posterior surgical decompression by laminoplasty with joint preservation, which increases the stability of the spinal column [23]. The prognosis is variable but typically favorable. However, the course of the disease is sometimes severe because of the size and extent of the stenosis. Nevertheless, a physical therapy rehabilitation program should be started as early as in the immediate postoperative period [23]. Our patient had moderate to severe calcification T10-11 and T11-12 levels that led to sensorimotor deficits and myelopathic symptoms during a 6-month period prior to surgery. He had a near full recovery following decompression and resection of the calcified ligamentum flavum from T10-12.

In conclusion, ossification or calcification of the ligament flavum can lead to spinal cord compression due to spinal canal stenosis. The diagnosis is preferably made by MRI and CT in combination. The endoscopic surgery is a safe, effective, and reliable method for ossified ligament flavum.

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