# Ossification of Ligamentum Flavum – Beckoning Surgeon's Knife!

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## **Abstract**

Ossification of ligamentum flavum (OLF) is well known but rare entity causing slow progressive thoracic myelopathy. It affects especially lower thoracic spine and is relatively common in the East Asian population particularly in Japan. Posterior decompression in the form of extensive laminectomy with or without instrumented fusion is the treatment of choice. Decompression itself can be very challenging since the flavum is fused with the laminae above and below and it becomes very difficult for the surgeon to insert Kerrison roungers in inter-laminar space. Seven cases of recurrence of OLF at same intervertebral level reported till now but no case of adjacent level OLF in thoracic spine reported yet. We report the case of a 37-year-old male with D6-7-8 ossified ligamentum flavum with coexisting asymptomatic L1-2 disc prolapse and previously operated for D8-9 OLF. Preoperative counseling of patients should be done regarding possibility of reoperation due to new adjacent segment or same level OLF.

Keywords: Ossified ligamentum flavum, Thoracic myelopathy, Posterior decompression

#### Introduction

Ossification of ligamentum flavum (OLF) is a known but very rare entity causing slow progressive thoracic myelopathy [1]. It especially affects thoracic spine and is relatively common in the East Asian population [2]. Posterior decompression in the form of extensive laminectomy with or without instrumented fusion is always the treatment of choice [3]. Decompression itself can be very challenging since the flavum is fused with the laminae above and below and it becomes very difficult for the surgeon to insert Kerrison roungers in inter-laminar space that is uncertain due to the low prevalence of disease and inconstant pre-operative status thus is invaluable for further research. Recurrence of OLF leading to thoracic myelopathy has been reported in the literature after laminectomy at the same intervertebral level [4, 5] but no case of adjacent level OLF in thoracic spine reported yet. We report a case of a 37-year-old male who was already operated for thoracic myelopathy secondary to OLF at D8-D9 level 4 years ago and now

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presented with gait abnormality due to OLF at D6 to D8 level with coexisting asymptomatic L1-2 disc prolapse.

## **Case Report**

A 37-year-old diabetic and obese male (body mass index:  $33.21~{\rm kg/m^2}$ ) presented with a 6-month history of progressively worsening paresthesia of the lower limbs and gait disturbance. He was operated for same complaints 4 years back by different surgeon after which he improved and lead normal life for  $3\frac{1}{2}$  years. We went through his pre-operative magnetic resonance imaging (MRI) done before first surgery 4 years back which revealed D8-9 OLF causing spinal canal compromise (Fig. 1).

When he presented to us, he was dependent on walker/stick for ambulation (Nurick grade IV) from the past 1 month. Preoperative clinical examination showed weakness (4/5) of both lower limbs; hyper-reflexia (3+); +Babinski; decreased sensation below D6 (all modalities). Motor strength, deep tendon reflexes, and sensations were normal in the upper limbs. Blood investigations revealed no abnormality except deranged sugar levels. MRI showed a posterior heterogeneous mass hypointense on both T1- and T2-weighted images from D6 to D8 (Fig. 2a, b). CT scan revealed a posterior bony mass at D6-7-8 levels in canal, which occupied about 50–60% of the spinal canal (Fig. 3a, b) and previous laminectomy caudal to it. No dural ossification (DO) was seen in CT. A posterocentral disc

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protrusion was seen at L1-2 level (Fig. 4) but no cord edema was seen at that level. Opinion of three different radiologists and neurologists was taken for the same and decision was taken to operate only dorsal OLF.

Standard posterior midline approach was taken. Laminae were approached. There was no interlaminar space presents between D6-7 and D7-8 and it was a complete bony bar, so introduction of dura retractor/Kerrison rounger into interlaminar space was not possible.

High speed burr was used. Vertical lateral gutter was made bilaterally without burring the facets. Horizontal separation was done through normal interlaminar space at cephalad D5-6 level and from fibrosis between remaining superior half lamina of D8 from the previous surgery caudally. The previous surgeon excised inferior half lamina and superior half lamina was intact of D8. A complete rectangular bony block was lifted in toto and cord was decompressed. It was observed to be a solid bony block, with no interlaminar flexibility at all (Fig. 5). Large-rounded ossified globules of flavum were seen on the



Figure 1: (a) A 4-year-old MRI sagittal T2-image showing compression at D8-9 level and (b) -A 4-year-old MRI axial T2-image showing posterocentral and left posterolateral mass arising from ligamentum flavum.



Figure 2: (a) MRI sagittal T2-images showing posterior thoracic cord compression ad D6-7-8 levels and (b) MRI axial T2-images showing large posterior mass compromising the canal arising from the ligamentum flavum.

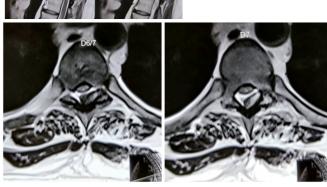




Figure 3: (a) Sagittal CT showing a bony mass occupying significant canal area and previous laminectomy caudal to it. (b) Axial CT showing a bony mass occupying significant canal area.







Figure 4: MRI sagittal T2images showing  $\bar{\text{L1-2}}$  disc prolapse with minimal cord compression and no abnormal cord signal.

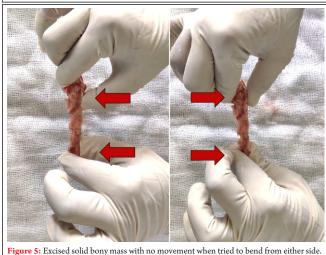




Figure 6: Ossified ligamentum flavum.



Figure 7: Post-operative MRI at the end of 6 months showing complete cord decompression with no residual abnormal cord signal.

ventral aspect of excised bony mass (Fig. 6).

Postoperatively, there was no neurological worsening. Surgical incision healed well. Patient was mobilized on day 1. At 6-month follow-up, even though the lower limb hyper-reflexia persisted, power in both lower limbs was restored to normal; he did not have any gait disturbance whatsoever. Nurick grade improved to Grade 1 from Grade 4. Patient did not have any symptoms related to L1-2 disc and its canal compromise. MRI of dorsal spine showed complete cord decompression (Fig. 7).

## Discussion

OLF is a rare pathology that occurs due to the formation of the lamellar bone secondary to endochondral ossification [6]. In 2017, Geber and Hammer [7] studied Nineteenth-Century Skeletal Population in Ireland and did review of the literature on OLF and concluded that etiology of OLF is largely unclear. They found literature on OLF manifestations in relation to multiple factors including diffuse idiopathic skeletal hyperostosis (DISH), obesity, diabetes, hyperinsulinism, and impaired glucose tolerance. Our patient was obese with BMI 33.21 Kg/m² and diagnosed with diabetes mellitus at the age of

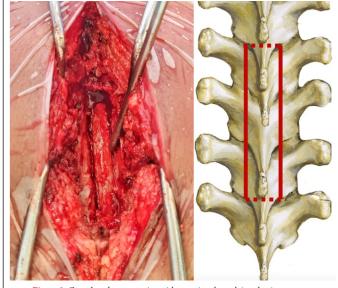


Figure 8: Complete decompression without going through interlaminar space.

14 and had radiographic changes of DISH on CT. In presence of complete ossification, the interlaminar space is always obliterated and entering the Kerrison rounger into the interlaminar space for decompression is impossible. Attempts of forceful entry of the rounger or dissectors may increase the pressure over the cord and lead to post-operative neuro-deficit. Ultrasonic bone scalpel or high-speed burrs can be of great help in this matter. With the help of either of these, the midline decompression can be completely bypassed; lateral gutters can be created; and a rectangular mass can be lifted in toto without touching the cord (Fig. 8).

While carrying out such decompressions in patients with OLF, DO should always be ruled out on pre-operative CT scan as its incidence is 43.4% in thoracic OLF patients [8]. In case of DO, difficulty of surgery increases and chances of dural tear are high. It is difficult to differentiate between the bone/flavum/dura while cutting with a bone scalpel or a burr if dura is ossified too and the complications can be devastating. Fortunately, in our case, there was no DO. Osman et al. [9] did meta-analysis of complications during OLF resection and found that a dural tear happens in 18% of patients. We could avoid it safely in our case. Concurrent lumbar spinal canal stenosis (LCS) can be present in the same patient hence pre-operative MRI should include whole spine screening. Yamada et al. [1] reported 46 cases of concurrent LCS and some of them required additional lumbar decompression surgery. In our case, there was L1-L2 lumbar disc herniation but since it was asymptomatic, it was treated conservatively.

In our patient, the myelopathy was slowly progressive but patients can present with acute onset severe thoracic myelopathy secondary to minor trauma in pre-existing compromised canal with OLF. In this type of cases, prognosis is guarded [2]. Resection of remaining normal flavum should be

complete at that particular level, as the remaining flavum may get ossified with time and may lead to recurrent compression at the same operated level. Till now, seven cases of recurrence of OLF at the same operated level have been documented [5] but no case of adjacent level OLF documented to the best of our knowledge. If recurrence occurs at same level, then decompression with fusion is the treatment of choice considering the possible role of biomechanical stress [4,5]. Hence, to conclude, pre-operative whole spine MRI and CT to establish the diagnosis is mandatory. Former gives information of levels of compression, condition of the cord and later gives information of shape and extent of the ossified mass. Postoperative recovery will be incomplete if irreversible damage of

the spinal cord is there [3] hence early diagnosis and decompression surgery are recommended. Even though detected early, surgery can be a challenge and needs to be carried out delicately and wisely. Ultrasonic bone scalpel or high-speed burr can be a game changer in these types of surgeries. Pre-operative counseling of patients should be done regarding possibility of reoperation due to new adjacent segment or same level OLF.

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**Declaration of patient consent:** The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the Journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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