Role of thickened ligamentum flavum in lumbar spinal canal stenosis

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Abstract

Background: This is a per operative study of thickened Ligamentum flavum, in lumbar canal stenosis due to degenerative spondylosis, found to be an important compressive force on emerging nerve root, along with hypertrophy of adjacent facet joint, causing radiculopathy [neurological claudication] independent of degenerative disc disease.

Materials and Methods: We operated on, 98 elderly patients, suffering from degenerative lumbar spondylosis with canal stenosis with radiculopathy. The surgeries were performed over the time period from 2011 October to 2014 May. Seventy patients were males and twenty eight were females. The mean age of patient was of 62 years (range 56 to 74 years). Out of 70 male patients, Ligamentum flavum is thickened bilaterally in 62 patients and it is more thickened unilaterally in eight patients, all 28 female patients have bilateral uniform thickening of Ligamentum flavum. All the patients of lumbar canal stenosis due to degenerative spondylosis , lumber sacral spine screened by MRI scanning, and found Ligamentum flavum is thickened in all patients.

Results: Internal decompression of lumbar canal stenosis, by thorough removal of thickened Ligamentum flavum, along with partial excision of hypertrophied superior facet; make the intervertebral foramen wide, and thus creating more room for emerging nerve root. All patients were free from symptoms post operatively.

Conclusion: Thickened Ligamentum flavum must be excised thoroughly, along with partial fascetectomy of adjacent superior facet and thus decompressing the spinal root in lateral canal where it is being compressed by thickened Ligamentum flavum and by hypertrophied facet joint.

Key words: Ligamentum flavum, internal decompression, hypertrophied facet joint.

INTRODUCTION

Narrowing of lumbar spinal canal is an increasing problem affecting the elderly. The advantage of CT and MRI imaging has provided a better understanding of the pathology of lumbar canal stenosis. Lumbar spinal canal stenosis is narrowing of the spinal canal, nerve root canal and the intervertebral foramen. Ligamentum flavum or yellow ligament connects two adjacent laminae and anteriorly it is continuous with capsular ligament of facet joints. It is stretched during flexion of vertebral column and recoils back during extension. In degenerative spine, Ligamentum flavum found to be thickened and fibrosed and it is responsible for nerve root compression[1]. Hence removal of thickened Ligamentum flavum, as a part of internal decompression, gives good results..

MATERIALS AND METHODS

A study of 98 patients, suffering from lumbar canal stenosis, revealed presence of hypertrophied ligamentum flavum, confirmed by MRI scanning, most of them have bilateral thickened Ligamentum flavum, a few patients have unilateral thickening of Ligamentum flavum, associated with hypertrophied facet joint [2]. As the lateral portion of Ligamentum flavum constructs the dorsal lateral wall of the inter vertebral foramen, its thickening causes, narrowing of lateral spinal canal and inter vertebral foramen, thus causing compression over the emerging nerve root, leading to neurological claudication [3].

How to Cite this Article:
RESULTS & DISCUSSION

The degeneration of canal components including Facet arthropathy, Ligamentum flavum hypertrophy, osteophyte formation, dehydrated I.V disc. The sites of stenosis were observed both in the spinal canal and root tunnel in many cases. The Ligamentum flavum loses its elasticity and shortened and projected into the spinal canal due to narrowing of I.V disc space, and in addition to this, Ligamentum flavum shows qualitative changes such as degeneration, hypertrophy and fibrosis.[4] There was more Ligamentum flavum thickening on the side with greater facet degenerative disease. Ligamentum flavum thickening can be secondary to facet joint degenerative (bony proliferation over facet joints) changes, independent of disc space narrowing. Lumbar ligamentum flavum hypertrophy may be due to accumulation of inflammation related scar tissue[5]. In asymmetric facet hypertrophy, the Ligamentum flavum thickening is more on the side where facet hypertrophy is more. Removal of Ligamentum flavum is an essential goal of the surgical treatment to decompress the neural tissue.

Operative technique

In our study we did routine laminectomy, foraminotomy by excising spur and hypertrophied superior facet, and thorough removal of thickened Ligamentum flavum which is very closely merging with dorso medial part of capsular ligament of facet joint, which is also hypertrophied with fibrosis, exerting compressive force on emerging nerve root. Care must be taken not to injure either dura or the root.

Per operative observation

The degenerated part of the spine especially L3- L4, L4-L5, L5-S1, intervertebral spaces are reduced due to compressed dehydrated discs, and inter spinous and inter laminar spaces are also reduced, as the Ligamentum flavum is within the spinal canal which is already thickened, makes the spinal canal further narrow and the space available for the roots to emerge was hardly found[6]. Thickened Ligamentum flavum, is mainly compressing the dorsolateral part of cauda equina, and as we remove it, the lateral canal provides more room for the dura to bulge.

In a normal spine, Ligamentum flavum protects the meninges, and helps in flexion and extension of spine, as it is an elastic ligament. In a degenerative spine, Ligamentum flavum loses its elasticity, and it is thickened. The degree of calcification as well as the structural changes of the elastic fibres in the ligamentum flavum are also responsible for its hypertrophy. This hypertrophied ligamentum flavum found to be an important compressive force on emerging nerve root. Though it is a part of motion segment, its removal will not cause any instability, because, in degenerative spine, due to natural stabilization, stability will be maintained. Post operatively, all the patients, were free of symptoms, no patient had instability.

Competing interests

The authors have declared that no competing interests exist.

References


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