Original research article

MRI Study of Lumbar Spinal Stenosis and its Correlation with Ligamentum Flavum Hypertrophy

Sumity Singh[1], Sanjay Kumar Suman[2], Amit Kumar[3], Waghambre Vaibhav[4], Sanaullah Mudassir[5]

[1]Senior Resident, Dept. of Radiology, IGIMS, Patna.

[2]Professor & Head, Associate Professor, Dept. of Radiology, IGIMS, Patna

[3]Associate Professor, Dept. of Radiology, IGIMS, Patna

[4]DNB Resident, Dept. of Radiology, IGIMS, Patna

[5]Senior Resident, Dept. of Neuromedicine, IGIMS, Patna

Corresponding Author: Sumity Singh

Abstract

Background and Objectives: - This study was conducted to determine the relationship of LF thickness with spinal canal diameter with LF thickening identification at a specific lumbar level. The relationship of lumbar spinal LF thickening with age and sex using magnetic resonance imaging (MRI) was also assessed.

Materials and Methods:- This was a prospective study done on 60 cases at IGIMS, Patna. Patients were referred from different department specially neuromedicine and neurosurgery department. Measurements of spinal canal diameter and LF thickness were taken from T1- and T2-weighted axial MRI scans using optima MR360/ 1.5T MRI.

Results: A total of 300 spinal canal diameters, along with 300 intervertebral disc heights and 600 ligamentum flavum thicknesses with mean value of bilateral LF thickness were assessed from MRI scans. The most common involved level with maximum stenosis was L4-5 (53.33 %) which needed surgery with more non-DSS involvement. Maximum thickness of LF with mean value of 9.4 mm was noted at L4-5 level (53.33 %) which was associated with maximum spinal canal stenosis.

Conclusion: This study shows significant difference in the relationships of spinal canal diameter, LF hypertrophy in DSS and non-DSS group. Thickness of the ligamentum flavum increased with age and this increase is most pronounced at the lower lumbar levels which lead to lumbar spinal stenosis of variable degrees, specifically in Non-DSS group.

Keywords: Lumbar Spinal Stenosis, MRI, Ligamentum Flavum

Introduction

The ligamentum flavum connects the laminae of vertebrae from C2 to S1 and extends medially along the facet joint which consists of 80% elastin fibers and 20% collagen fibers which provides its yellow color .This is a paired structure at each interspinous level which appears thin in cervical ,become broad in thoracic and thickest at lumbar region. Ligament consist of two portion as medial portion which passes to subsequent lower lamina and across the gap between the adjacent vertebrae fusing with the interspinous ligament, the lateral portion which passes before of the facet joint where it attaches to the anterior aspect of the inferior and superior articular processes and forms the anterior capsule and the foremost lateral fibers extend beyond the superior articular process to lower pedicle .

Hypertrophy of this ligament may cause spinal stenosis due to posteriorly located. Increased expression of TIMP-2 in ligamentum flavum fibroblasts is related to fibrosis and hypertrophy of the ligamentum flavum in patients with spinal stenosis[9,10].Degeneration with thickening and calcification of LF can cause foraminal narrowing, vertebral canal stenosis, and nerve root compression at the lumbar level. LF thickening may result radiculopathy.So, lower back pain has become a major healthcare concern in middle-aged and elderly individuals results derranged lifestyle due to its indolent course causes late diagnosis. So, early diagnosis and treatment may improve prognosis. Posterior disc herniation may also cause canal stenosis and compression of the nerve roots in degenerative spine diseases. Few studies showed mechanism of thickening and hypertrophy of the ligament[1,4,] probably results from injury due to excessive spinal flexion as there is little or no regenerative capacity within the connective tissue of LF .So, it is replaced by a dense connectivel tissue and results neural compression. Two theories have been given for LF hypertrophy[8,13]. Firstly, LF hypertrophy due to fibrotic changes in response to degenerative cascade,[9] in which there is increased level of collagen fibers and reduced elastin . Increased number of fibrocartilaginous cells lead to the proliferation of collagen fibers within a hypertrophic LF results compression of neural elements, even without annulus fibrosus bulge or disc herniation.

The second pathomechanism is that the apparent LF hypertrophy as a results of disc height reduction or disc degeneration. A loss of disc height[10] will cause laxity within the ligamentous tissues surrounding the spine, which results in LF buckling.

It is therefore clear that both vertebral canal diameter and LF thickness play crucial roles within the pathogenesis of spinal stenosis. Pre-existing narrowed canal are more prone to stenosis even in milder degree of LF hypertrophy. By comparison, in larger-sized canals, a more significant contribution may exist with flaval hypertrophy. Thus, this study assessed spinal canal stenosis at different lumbar level in both preexisting and degenerative patients with ligamentum flavum thickening assessment as a cause of stenosis alonwith other associated findings of degeneration and disc herniation.

Aim & Objective- This study is done to determine the relationship of LF thickness with spinal canal diameter with LF thickening assessment at a specific lumbar level. Also, to determine the relationship of lumbar spinal LF thickness with age and sex using magnetic resonance imaging (MRI).

Material & Methods

This was a Prospective study done at IGIMS, PATNA with sample size of 60.Patients were referred from neuromedicine and neurosurgery department of IGIMS, Patna.

Inclusion criteria was All age group ,Symptomatic lumbar spinal stenosis, Patients with isthmic and degenerative spondylolisthesis, Scoliosis, Fractures and Previous surgery and epidural injections .Exclusion criteria included Infections and Tumours. **Imaging-** Measurements of spinal canal diameter and LF thickness were taken from T1- and T2-weighted axial MRI scans using optima MR360/ 1.5T MRI. The field of view was 18 x 16.2 mm for axial scans and 32x32 mm for sagittal scans. Slice thickness was 4 mm for both scans, and slice spacing was 1 mm for axial scans and 1 mm for sagittal scans. Imaging matrix was 320 x 224 for axial scans and 320 x 192 for sagittal scans. The repetition time was 800 to 900 ms and 4000 to 6000 ms for T1 and T2, respectively. There were 12 slices per vertebral level and parallel slices were made according to the disc and pedicle levels.

Spinal canal diameter and LF thickness: LF thickness was measured at L1-L2, L2-L3, L3-L4, L4-L5 and L5-S1 intervertebral disc levels. The AP spinal canal diameters were measured using T1-weighted axial MRI, at the cut where the entire bony canal ring[6] could be seen and with the thickest pedicle width. The LF was measured at the midpoint of the facet joint level using T2-weighted axial MRI at the cut with the thickest LF measured. (Fig 1, fig 2) **Disc height**, Disc heights were assessed on T2-weighted MRI sagittal images and were measured from the middle of the intervertebral disc (from the thickest cut) perpendicular to the endplate, from L1-2 to L5-S1 at each level. Any anterior disc bulging was also noted. (fig 3)

Patient Group: atient group classification was pre existing (developmental) or secondary to degeneration (non- developmental) (fig. 4). The phenotypical definitions of DSS[3,5,6,7] by Cheung et al.,patients were classified into 3 groups: DSS with critical stenosis (Group 1), DSS with relative stenosis (Group 2), and non-DSS (Group 3). Measurements were based on the AP bony spinal canal diameter at the vertebral level. Critical stenosis values were defined as: L4 < 14 mm, L5 < 14 mm, and S1 < 12 mm. DSS was defined as: L1 < 20 mm, L2 < 19 mm, L3 < 19 mm, L4 < 17 mm, L5 < 16 mm, and at S1 < 16 mm. We have included two broad group as DSS(group 1) & Non-DSS(group 2) in this study. Normal thecal sac transverse diameter is 16-18mm and stenosis is described < 15 mm.
Lateral recess (subarticular or entrance zone)-;> 5mm in height, 3-5mm relative stenosis and < 3 mm is absolute stenosis.

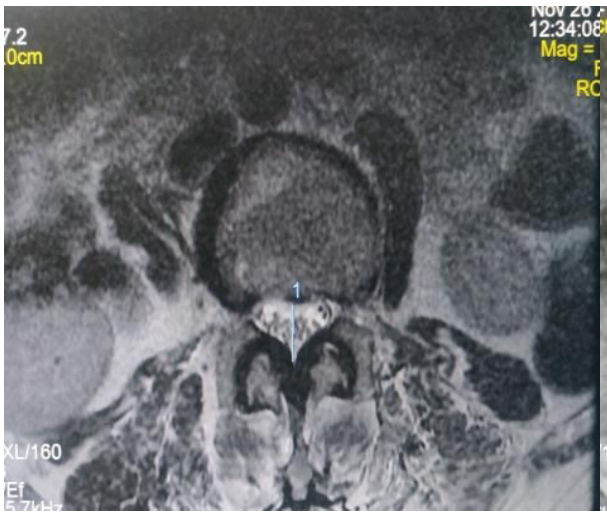

Fig. 1

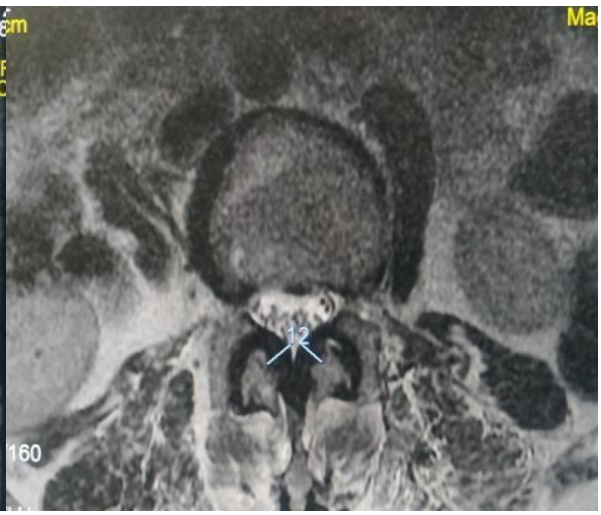

Fig. 2

Fig. 1: T2WI shows measurement of anteroposterior (AP) spinal canal diameter at L4-L5 where the entire bony canal ring and the thickest pedicle width is seen.
Fig. 2: Measurement of ligamentum flavum maximum transeverse thickness.

Fig.3 **Fig. 4**

Fig.3 Measurement of disc height at midpoint of IVD.
Fig.-4 Developmental lumbar scoliotic structural deformity.

Result

Our study taken sample size of 60 patients, out of which 32 were female and 28 were male patients (table 1). Females had a mean age of 48 ± 10 years and males had a mean age of 52 ± 11.0 years. Patients were referred from different department specially neuromedicine and neurosurgery department of IGIMS,patna. Measurements[9] of spinal canal diameter and LF thickness were taken from T1- and T2-weighted axial MRI scans using optima MR360/ 1.5T MRI. Statistical analysis of sample size and population mean values were evaluated using the standard formula for small sized population.

A total of 300 spinal canal diameters, along with 300 intervertebral disc heights and 600 ligamentum flavum thickness with mean value of bilateral LF thickness were assessed from MRI scans. The most common involved level with maximum stenosis was L4-5 (53.33 %) which needed surgery[4] for severe pain and paraplegic symptoms.L3-L4 (30%) was second most common followed by L2-L3 (13.33%) and L1-L2(3.33%).Least common involvement of L5-S1level was seen.(table3) Mean spinal canal diameter(in mm) were 11.2 mm at L1-L2 level,12mm at L2-L3,9 mm at L3-L4,6mm at L4-L5 and 14 mm at L5-S1 lumbar level for all 60 cases. Maximum diameter was seen at L5-S1 and minimum were noted at L4-L5 level.L1-L2 involvement and resultant canal narrowing was mostly seen in DSS cases and few were associated with disc bulge. Patients were classified as DSS (Developmental spinal stenosis) and Non-DSS patients (Non- developmental).10 were DSS and 50 were Non-DSS (table 2). L1-L2 was identified most common lumbar involvement with mean spinal canal diameter 11.2 mm in DSS cases. Mean LF thickness was 3mm at this level.While L4-L5 was the most common level involved in Non-DSS cases with mean spinal canal diameter was 6mm that is significant spinal canal stenosis .Mean LF thickness was 9.4 mm which was more thickened(table 4) .This also contributed to posterolateral thecal sac compression and results spinal canal narrowing. Maximum thickness of LF with mean value of 9.4 mm was noted at L4-5 level (53.33 %) which was associated with maximum spinal canal stenosis .L3-L4(30%) was second most common with mean LF thickness 7.3 mm followed by L2-L3(13.33%) with mean value 3.5 mm and L1-L2(3.33%) with mean LF thickness of 3 mm.Least common involvement of L5-S1level was seen.More common side of increased flaval thickness was right side in all 60 cases(100%).(table 5) LFH was seen more commonly in >45 year of age group patients with direct positive relation with age due to different predisposing condition increased with age. Maximum mean value was 8.5 mm in more than

55 year of age group. Similar findings were also noted for disc height. There was direct negative relationship with age group and disc height. Maximum 11 mm for 10-15 years age group and minimum 2 mm for more than 55 year age group were seen. We found associated spondylosis, spondylolisthesis with these findings in increased age group patients. There was significant differences found in spinal canal diameter ($p < 0.001$) among two study groups that is DSS and Non-DSS;despite this LF thickness, disc height did not demonstrate any differences in DSS group. The difference in canal diameter was 10.2 mm at L4, and 4.8 mm at L5 for Groups 1 and 2. Central disc bulging occurred most commonly within non-dss group (90 % of all non-DSS) as compared to DSS group (10 %).

Discussion

Ligamentum flavum connects the laminae of vertebrae from C2 to S1 and extends medially along the facet joint. LF hypertrophy can lead to compression of the spinal cord, cauda equina, and nerve roots by narrowing of posterolateral aspect of spinal canal.Different factors of disc herniation, degeneration are commonly associated with LFH in non-developmental cases. However, in developmental architectural distortion can also lead to isolated LFH and may result spinal canal stenosis.But exact cause is still in reaseach.

However, two main theories are noted for hypertrophic LF. These are collagen synthesis, fibrotic changes due to Increased expression of biomarkers in response to inflammatory changes and various stress conditions. Another one theory given suggests that LFH can be secondary to in-folding of the LF into the spinal canal due to disc collapse and reduced disc height. Due to decreased elasticity of LF and disc bulge into the canal space results canal narrowing.

Altinkaya[11] et al., studied among 224 patients with lumbocrural pain[12].He studied that all segments of ligamenta flavum thickness had a positive correlation with age, but had no correlation with sex. Abbas, 2010 studied 65 patients with lumbar spinal stenosis and 150 with the thickness of the ligamentum flavum in all segments was independent normal adults .In his study, Ligamentum flavum thickness was not related to sex. It was thicker on the left than on the right at the segments L5 and S1.Ligamentum flavum thickness was not positively correlated with age.In another study of Safaq et.al., Ligament flavum thickness was not related to sex .It was thicker on the left than on the right at the segments L4-5 and L5-S1,.Ligamentum flavum thickness was not positively correlated with age.(table 7)In our present study, 60 patients (25 male, 35 female) of all age group from 10 to more than 55 yrs were included. Our study showed Ligamentum flavum hypertrophy was directly related to spinal canal stenosis This was more commonly noted in non developmental(degenerative) cases.LFH showed positive correlation with increase in lumbar vertebral level mostly at L4-L5 which were directly increased as patient age.This study didn’nt show correlation with sex.However, female cases were more in our study. Cause of narrowing of spinal canal and associated LFH were determined by patient group classification whether pre existing (developmental) or secondaryto degeneration (non- developmental). We have included two broad group as DSS (group 1) & Non-DSS (group 2) in this study. Other associated finding we evaluated was reduced disc height ,degeneration and spondylolisthesis resulting spinal canal stenosis more commonly seen in in older age group non- developmental cases. (table 6).This may result segmental instability and repetitive motion stress can lead to LF hypertrophy.Developmental spinal stenosis commonly results from congenital short pedicle which may have genetic origin. Due to pre-existing narrowing there was lesser degree of LF hypertrophy, as compared to normal-sized canals and early development of symptoms. The

average thickness of ligamentum flavum[1,6] is about 2.5-3.4mm in a healthy adult. A thickness of 3.5mm or above is considered a hypertrophy. This was identified at all levels including L4-L5 which is the most commonly involved level.

Table 1:

(N=60)

MALE	**FEMALE**
25	**35**

Table 2:

N=60

DSS	NON -DSS
10	50

Table 3:

Vertebral Levels N=60	Mean LF thickness (mm (±SD))		Mean Spinal Canal Diameter (mm (±SD))
	Right	Left	
L1-L2	3	1.5	11.2
L2-L3	3.6	3	12
L3-L4	7	6.8	9
L4-L5	9.8	9	6
L5-S1	4	3.5	14

Table 4:

NUMBER OF PATIENTS(TOTAL=60)	SPINAL CANAL NARROWING LUMBAR LEVEL	AVERAGE LIGAMENTUM FLAVAL THICKNESS(t) (mm)		
		Right	Left	mean
8	L2-L3	3.8	3.2	3.5
32	L4-L5	9.8	9	9.4
18	L3-L4	7.6	7	7.3
2	L1-L2	4	2	3

Table 5:

AGE(yrs)	LFH(t)(mm)mean			SPINAL CANAL DIAM.(MM)	DISC HEIGHT(AVG.)MM
	RT.	LT.	MEAN		
10-15	3	2	2.5	11.2	11
16-30	4	3.8	3.9	16	10
31-45	5.6	5.5	5.55	12	6
46-55	8	7	7.5	9	3
>55	9	8	8.5	6	2

Table 6:

	MEAN LFH (MAX. THICKNESS)MM	SPINAL CANAL STENOSIS	LUMBAR LEVEL
DSS	**3**	**11.2**	**L1-L2**
NON-DSS	**9.4**	**6**	**L4-L5**

Conclusion

This study shows significant difference in the relationships of spinal canal diameter, LF hypertrophy in DSS and non-DSS group. Thickness of the ligamentum flavum increased with age and this increase is most pronounced at the lower lumbar levels which lead to lumbar spinal stenosis of variable degrees, specifically in Non-DSS group. Reduced disc height and disc bulge with herniation were also seen exclusively in Non-DSS group symptomatic cases mostly in osteoporotic female patients.DSS group showed no to few association of LF hypertrophy with congenital spinal canal stenosis mostly of severe grade.

References

1. Safak AA Is M, Sevinc O, Barut C, Eryoruk N, Erdogmus B, et al. The thickness of the ligamentum flavum in relation to age and gender. Clin Anat. 2010; 23:79–83.
2. Yi Liu, et.al.Treatment for lumbar spinal stenosis secondary to ligamentum flavum hypertrophy using percutaneous endoscopy through interlaminar approach: a retrospective study PMID: 32811508 PMCID: PMC7437061 DOI: 10.1186/s13018-020-01874-5 2020 Aug 18; 15(1):337. doi: 10.1186/s13018-020-01874-5**.**
3. Lee CK, Hansen HT, Weiss AB. Developmental lumbar spinal stenosis. Pathology and surgical treatment.Spine. 1978;3:246–55. doi: 10.1097/00007632-197809000-00010.
4. Yong-Hing K, Reilly J, Kirkaldy-Willis W. The ligamentum flavum. Spine. 1976; 1:226–34. doi: 10.1097/00007632-197612000-00007.
5. Kitab SA, Alsulaiman AM, Benzel EC. Anatomic radiological variations in developmental lumbar spinal stenosis: a prospective, control-matched comparative analysis. Spine J. 2014; 14:808–15. doi: 10.1016/j.spinee.2013.09.012.
6. Cheung JP, Shigematsu H, Cheung KM. Verification of measurements of lumbar spinal dimensions in T1- and T2-weighted magnetic resonance imaging sequences. Spine J. 2014; 14:1476–83. doi: 10.1016/j.spinee.2013.08.054.
7. Cheung JP, Samartzis D, Shigematsu H, Cheung KM. Defining clinically relevant values for developmental spinal stenosis: a large-scale magnetic resonance imaging study. Spine. 2014; 39:1067–76. doi: 10.1097/BRS.0000000000000335.
8. Hansson T, Suzuki N, Hebelka H, Gaulitz A. The narrowing of the lumbar spinal canal during loaded MRI: the effects of the disc and ligamentum flavum. Eur Spine J. 2009;18:679–86. doi: 10.1007/s00586-009-0919-7.[PMC free article] [PubMed] [CrossRef] [Google Scholar]
9. Sairyo K, Biyani A, Goel V, Leaman D, Booth R, Jr, Thomas J, et al. Pathomechanism of ligamentum flavum hypertrophy: a multidisciplinary investigation based on clinical, biomechanical, histologic, and biologic assessments.Spine. 2005;30:2649–56. doi: 10.1097/01.brs.0000188117.77657.ee. [PubMed] [CrossRef] [Google Scholar]
10. Weiler C, Lopez-Ramos M, Mayer HM, Korge A, Siepe CJ, Wuertz K, et al. Histological analysis of surgical lumbar intervertebral disc tissue provides evidence for an association between disc degeneration and increased body mass index. BMC Res Notes. 2011;4:497. doi: 10.1186/1756-0500-4-497. [PMC free article] [PubMed]

[CrossRef] [Google Scholar
11. Altinkaya N, Yildirim T, Demir S, Alkan O, Sarica FB. Factors associated with the thickness of the ligamentum flavum: is ligamentum flavum thickening due to hypertrophy or buckling? Spine. 2011;36:E1093–7. doi: 10.1097/BRS.0b013e318203e2b5. [PubMed] [CrossRef] [Google Scholar]
12. Munns JJ, Lee JY, Espinoza Orias AA, Takatori R, Andersson GB, An HS, et al. Ligamentum flavum hypertrophy in asymptomatic and chronic low back pain subjects. PLoS One. 2015;10:e0128321. doi: 10.1371/journal.pone.0128321. [PMC free article] [PubMed] [CrossRef] [Google Scholar]
13. Yoshida M, Shima K, Taniguchi Y, Tamaki T, Tanaka T. Hypertrophied ligamentum flavum in lumbar spinal canal stenosis. Pathogenesis and morphologic and immunohistochemical observation. Spine. 1992;17:1353–60. doi: 10.1097/00007632-199211000-00015. [PubMed] [CrossRef] [Google Scholar]