# Incidence of ligamentum flavum hypertrophy in patients with spinal stenosis Munir Saadeddin

Department of Orthopedic Surgery, King Saud University, Riyadh, Saudi Arabia

Correspondence to Munir Saadeddin, FRCSEd, PO Box 2905, Department of Orthopedic Surgery, King Saud University, Riyadh 11461, Saudi Arabia; Tel: 00966504265797; fax: 00966114679436; e-mail: msaadeddin@ksa.edu.sa

Received 12 January 2014 Accepted 18 April 2014

The Egyptian Orthopaedic Journal 2017, 52:150–152

#### Background

Spinal stenosis is most common in the cervical and lumbar regions. It is often a consequence of multifactorial acquired degenerative changes. Ligamentum flavum hypertrophy (LFH) is an important known cause, although its actual incidence is less well established. This study was therefore undertaken to evaluate its incidence at a tertiary referral center.

#### Patients and methods

A retrospective review of 98 patients with a radiological diagnosis of spinal stenosis was performed. Demographic data were also collected.

#### Results

Totally, 24 (24.5%) cases of LFH were identified. The majority of these (17 cases; 70.8%) were at the level of L4/L5 and six (25%) cases involved multiple levels. The incidence of LFH was greater in those patients with spinal stenosis who were aged 60 or older. LFH was also found in patients with other spinal pathologies, such as disc herniation, degenerative changes, and spondylolisthesis.

#### Conclusion

LFH was diagnosed in 24.5% of patients with spinal stenosis in this series. It predominantly occurs at the L4/L5 level and is more common in those aged 60 and above. These findings should be considered when selecting the type of management of spinal stenosis.

#### Keywords:

ligamentum flavum hypertrophy, spinal stenosis, spine

Egypt Orthop J 52:150–152 © 2017 The Egyptian Orthopaedic Journal 1110-1148

# Introduction

Spinal stenosis refers to the narrowing of the spinal canal anywhere along its axis. It is most common in the cervical and lumbar areas [1-3]. This disorder often results from acquired degenerative changes. The canal components that contribute to acquired stenosis include the facets (hypertrophy, arthropathy), the ligamentum flavum (hypertrophy) (LFH), the posterior longitudinal ligament (PLL), the vertebral body (bone spurs), the intervertebral disk (herniation), and epidural fat (herniation) [4]. Previous authors have suggested various hypotheses to explain the mechanism of LFH, although the incidence of LFH in spinal stenosis remains unknown [5–7]. It has been suggested that ligamentum flavum thickness is usually less than 4 mm in normal individuals [8,9]. This retrospective review was undertaken to better understand this phenomenon of LFH in spinal stenosis, and increase its awareness among orthopedic and spinal surgeons, so that it is incorporated into their decision process and management plans.

# Patients and methods

This is a retrospective study of 98 patients diagnosed with spinal stenosis on the basis of their computed tomography scan or MRI of the lumbosacral spine. IRB/Ethics committee approval was not required for this study. LFH was considered present when its thickness was of 4 mm or more. Initial imaging studies and their original reports were reviewed by independent radiologists (F.B., H.H., and I.O.).

# Results

There were 70 female and 28 male patients with spinal stenosis. Their ages ranged from 14 to 87 years (mean: 50.5). LFH was identified in 24 (24.5%) cases; 17 cases were at the level of L4/L5 (70.8%) and six (25%) cases involved multiple levels. LFH was more common in those aged 60 years and older. In the 56 patients aged over 60 years, the incidence of LFH was 36% (20 cases), compared with the 42 patients younger than 60 years, in which only four cases of LFH were diagnosed (10%). This was significant (P=0.004). Additional findings among the 98 patients included 71 cases of disc herniation, of which 22 displayed LFH (31%). Twenty cases were at the same level (90.9%). There

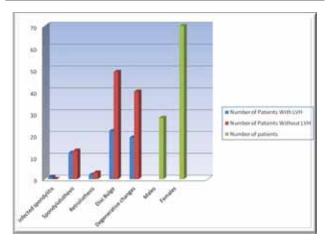
This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work noncommercially, as long as the author is credited and the new creations are licensed under the identical terms.

were also 59 cases of degenerative disc changes, of which 19 demonstrated LFH (32%); 16 cases were at the same level (84.2%). Spondylolisthesis was noted in 23 patients with spinal stenosis. Twenty patients had grade I spondylolisthesis (11 or 55% with LFH), two patients had grade II, and one patient had grade III spondylolisthesis. One patient had infected spondylitis (Table 1 and Fig. 1).

# Discussion

The three major ligaments of the spine are the ligamentum flavum, the anterior longitudinal ligament, and the PLL. The anterior longitudinal ligament and PLL are continuous bands that run from the top to the bottom of the spinal column along the vertebral bodies. They prevent excessive movement of the vertebral bones. The ligamentum flavum is a strong ligament that connects the laminae of the posterior arch of the vertebrae. The term 'flavum' is used to describe the yellow appearance of this ligament in its natural state. The ligamentum flavum serves to protect the neural elements and the

Figure 1



Comparison of male and female numbers of spinal stenosis and number of cases with left ventricular hypertrophy and without. LVH, left ventricular hypertrophy.

Table 1 The number of cases with different types of spinal stenosis

|                      | With LFH | Without LFH | Total |
|----------------------|----------|-------------|-------|
| Degenerative changes | 19       | 40          | 59    |
| Disc bulge           | 22       | 49          | 71    |
| Spondylolisthesis    |          |             |       |
| Grade 1              | 11       | 9           | 20    |
| Grade 2              | 0        | 2           | 2     |
| Grade 3              | 1        | 0           | 1     |
| Retrolisthesis       | 2        | 3           | 5     |
| Infected spondylitis | 1        | 0           | 1     |

LFH, ligamentum flavum hypertrophy.

spinal cord and stabilize the spine so that excessive motion between the vertebral bodies does not occur. It is the strongest of the spinal ligaments and often has a thinner middle section. Together with the laminae, it forms the posterior wall of the spinal canal, stabilizes the spine, and protects the discs. The ligamentum flavum is made of a superficial and a deep component. It is continuous in the midline. The superficial ligamentum flavum inserts onto the superior edge and posterosuperior surface of the caudal lamina. The deep ligamentum flavum inserts for a variable distance onto the anterosuperior surface of the caudal lamina [8–10]. Ligamentum flavum is not innervated, thus explaining lack of pain during spinal puncture. Pain during puncture may be attributed to Luschka's nerve, which innervates the PLL and dural ventral portion. Pain during spinal puncture is a warning that the needle tip has touched a nervous structure [11].

An international classification of lumbar spinalcanal stenosis has been developed, classifying these entities as (a) degenerative, (b) congenital/developmental, (c) combined, (d) spondylolysis/spondylolisthesis, (e) iatrogenic, and (f) post-traumatic stenosis. Degenerative stenosis is the most common type and results from disc degeneration, osteoarthritis of the facet joint, and hypertrophy of the ligamentum flavum. It may also be the result of intervertebral disc degeneration, protruded intervertebral disc, and/or bony spur that compresses the cauda equina and the spinal nerve root anteriorly, whereas joint degenerated facet and hypertrophied ligamentum flavum compress the cauda equina and spinal nerve root posteriorly [12]. The hypertrophy mechanism remains unclear. Accumulation of fibrosis (scarring) causes hypertrophy of the ligamentum flavum [5]. Fibrosis is caused by the accumulation of mechanical stress with the aging process, especially along the dorsal aspect of the ligamentum flavum. Transforming growth factor- $\beta$  released by the endothelial cells may stimulate fibrosis, especially during the early phase of hypertrophy [13]. Some studies suggested that the increased expression of active matrix metalloproteinases by the ligamentum flavum fibroblasts might be related to the elastin degradation and fibrosis of the ligamentum flavum in the patients who suffer from lumbar spinal stenosis [7].

MRI in the sagittal plane are important in demonstrating hypertrophy of the ligamentum flavum and can provide accurate measurements of its thickness [14]. In this study, age over 60 years correlated with a higher incidence of LFH. This finding is in accordance with previous studies that suggested that ligamentum flavum thickness is an age-dependent phenomenon [15,16].

In this study, 17 cases among the 24 patients with LFH were at the level of L4/L5 (70.8%). Most often, LFH was associated with other degenerative changes at the same intervertebral level supporting the hypothesis that LFH is a secondary phenomenon. In some cases of lumbar spine stenosis, thickened ligamentum flavum is found as the main source of the stenosis. Therefore, reducing the ligamentum flavum thickness may increase the segmental space available for the dural sac. This could be achieved in some cases by stretching the ligamentum flavum through interlaminar distraction (e.g. interspinous devices) rather than resorting to a more extensive procedure [17]; interlaminar decompression alone, including excision of the thickened ligamentum flavum with its insertion at the upper part of the lower lamina, could be sufficient to decompress the spinal canal in these instances. Revision surgery following this procedure could be easier and safer.

# Conclusion

In this series, LFH was diagnosed in one-quarter of patients with spinal stenosis. LFH was more common in those aged over 60 years old. The recognition of LFH as a cause for spinal stenosis is important, as this may alter the management options available.

### Acknowledgements

Special thanks to Dr Robert E. Turcotte for his help and advice into the production of this manuscript.

Financial support and sponsorship Nil.

#### Conflicts of interest

There are no conflicts of interest.

## References

- 1 Greenberg MS. Spinal stenosis. In: Greenberg MS, editor, Handbook of neurosurgery. Vol. 1. Lakeland, FL: Greenburg Graphics Inc.; 1997. pp. 207–217.
- 2 White AA III, Panjabi MM. Clinical biomechanics of the spine. 2nd ed. Philadelphia, PA: JB Lippincott; 1990. pp. 342–378.
- 3 Kalichman L, Cole R, Kim DH, Li L, Suri P, Guermazi A, et al. Spinal stenosis prevalence and association with symptoms: the Framingham study. Spine J 2009; 9:545–550.
- 4 Hsiang JN, Furman MB, Keenan MA, Nadalo R, Penar PL, Phillips CD, et al. Spinal stenosis. 2011. Available at: http://emedicine.medscape.com/ article/1913265-overview. [Accessed 19 April 2014].
- 5 Sairyo K, Biyani A, Goel VK, Leaman DQ, Booth R, Thomas J, et al. Lumbar ligamentum flavum hypertrophy is due to accumulation of inflammation-related scar tissue. Spine 2007; 32:E340–E347.
- 6 Sairyo K, Biyani A, Goel V, Leaman D, Booth R, Thomas J, et al. Pathomechanism of ligamentum flavum hypertrophy: a multidisciplinary investigation based on clinical, biomechanical, histologic, and biologic assessments. Spine 2005; 30:2649–2656.
- 7 Park J-B, Kong C-G, Suhl K-H, Chang E-D, Riew KD. The increased expression of matrix metalloproteinases associated with elastin degradation and fibrosis of the ligamentum flavum in patients with lumbar spinal stenosis. Clin Orthop Surg 2009; 1:81–89.
- 8 Olszewski A, Yaszemski MJ, White AA. The anatomy of the human lumbar ligamentum flavum: new observations and their surgical importance. Spine 1996; 21:2307–2312.
- 9 Postacchini F, Gumina S, Cinotti G, Perugia D, DeMartino C. Ligamenta flava in lumbar disc herniation and spinal stenosis. Light and electron microscopic morphology. Spine (Phila Pa 1976) 1994; 19:917–922.
- 10 Nachemson A, Evans JH. Some mechanical properties of the third interlaminar ligament (ligamentum flavum). J Biomech 1968; 1:211–220.
- 11 Zarzur E. Pain during spinal canal puncture and its relationship with ligamentum flavum, dura mater and posterior longitudinal ligament innervation. Rev Bras Anestesiol. 2004; 54:872–876.
- 12 Miyamoto M, Genbum Y, Ito H. Diagnosis and treatment of lumbar spinal canal stenosis. J Nippon Med Sch 2002; 69:583–587.
- 13 Park JB, Chang H, Lee JK. Quantitative analysis of transforming growth factor-beta 1 in ligamentum flavum of lumbar spinal stenosis and disc herniation. Spine 2001; 26:E492–E495.
- 14 Grenier N, Kressel HY, Schiebler ML, Grossman RI, Dalinka MK. Normal and degenerative posterior spinal structures: MR imaging. Radiology 1987; 165:517–525
- 15 Abbas J, Hamoud K, Masharawi YM, May H, Hay O, Medlej B, et al. Ligamentum flavum thickness in normal and stenotic lumbar spines. Spine 2010; 35:1225–1230.
- 16 Fukuyama S, Nakamura T, Ikeda T, Takagi K. The effect of mechanical stress on hypertrophy of he lumbar ligamentum flavum. J Spinal Disord 1995; 8:126–130.
- 17 Davis RJ, Errico TJ, Bae H, Auerbach JD. Decompression and Coflex interlaminar stabilization compared with decompression and instrumented spinal fusion for spinal stenosis and low-grade degenerative spondylolisthesis: two years results from the prospective, randomized, multicenter Food and Drug Administration Investigational Device Exemption Trial. Spine (Phila Pa 1976) 2013; 38:1529–1539.