JMISST Journal of Minimally Invasive Spine Surgery and Technique 6(1):9-13, 2021
Clinical Article

eISSN 2508-2043
https://doi.org/10.21182/jmisst.2020.00206
www.jmisst.org

Implications of the Precise Anatomical Location of Lumbar Stenosis for Minimally Invasive Decompressive Lumbar Surgery

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Received: October 02, 2020
Revised: November 18, 2020
Accepted: December 21, 2020

Objective: The purpose of this study was to characterize an observation that the most severe lumbar stenosis is often displaced from the disc. **Methods:** A retrospective magnetic resonance (MRI) review of displacement and causes of lumbar canal stenosis, was undertaken. Lumbar MRIs (n=3000) were reviewed for stenosis defined as a canal diameter of ≤8 mm. Displacement of maximal stenosis from the disc was measured; measurements inferior to the disc were assigned negative values. Defined causes were; ligamentous hypertrophy, facet hypertrophy, lipomatosis, spondylolisthesis, synovial cyst, or adjacent segment disease. **Results:** Lumbar stenosis levels (n=1,042) identified in 749 patients were; L1-2 (3.8%), L2-3 (20.1%), L3-4 (35.3%), L4-5 (37.7%), and L5-S1 (3.2%). Of these levels 20.8% were attributed to facet hypertrophy, 29.8% ligamentous hypertrophy, 31.1% epidural lipomatosis, 11.2% spondylolisthesis, 5.6% adjacent segment disease, and 1.5% synovial cyst. Mean displacement stenosis (mm) was; synovial cyst (-0.3; range 7 to -5), epidural lipomatosis (-1.1; 5 to -13), ligamentous hypertrophy (-3.5; 5 to -13); facet hypertrophy (-3.9; 7 to -11), adjacent segment disease (-4.7; 7 to -11), and spondylolisthesis (-4.9; 11 to -12). Sub-group analysis revealed a predominantly negative displacement for spondylolisthesis, adjacent segment disease, facet hypertrophy, and ligamentous hypertrophy. **Conclusion:** The site of maximal lumbar stenosis is at or near the center of the disc with lipomatosis or synovial cyst, but significantly inferiorly displaced when ligamentous or facet hypertrophy, spondylolisthesis, or adjacent segment disease is the major cause. Lipomatosis as a cause of stenosis is more common than previously reported.

Key Words: Lumbar stenosis, Minimally invasive spine surgery, Epidural lipomatosis

INTRODUCTION

Lumbar spinal stenosis requiring surgical decompression is a very common spine condition[1,2]. Open surgery has in part been supplanted by minimally invasive procedures, often via tubular retractors or endoscopes[3]. When the operating corridor is thus confined, it is important that an access device be directed at the critical pathology points so that stenotic areas do not remain rostral or caudal to the field of view. The first author has noted (personal observation over 20 years of minimally invasive spine surgery experience) that the most severe site of lumbar stenosis is often displaced from the disc space itself, and, therefore, care must be taken to direct the tubular retractor toward the most severe area of stenosis. The primary purpose of the study was to characterize the observation noted above. Therefore, we undertook a radiographic review, at a single institution, over 2 years, to quantify the location, cranial/caudal displacement, and causes of lumbar canal stenosis as determined retrospectively by lumbar magnetic resonance imaging (MRI).

MATERIAL AND METHODS

1. Study Design

A retrospective review was undertaken of 3,000 lumbar MRIs performed at a single institution in 2017 and 2018. Review was conducted by a neurosurgeon (DAR), a neuroradiologist (JMP), a radiology resident (MC and NPL) and 2 medical students (JE and MS). Only cases of routine spondylosis were included. Cases of trauma, neoplasm, or infectious conditions were excluded. Cases with disc protrusions as the major contributor to spondylotic stenosis were excluded as most surgeons are likely to be aware that disc fragments can migrate and routinely direct the surgery to the site of the imaged fragments. Levels previously operated upon were also excluded.

2. Lumbar Magnetic Resonance Imaging Review

Sagittal and axial T2 weighted images were inspected looking for areas with a midline anterior posterior (AP) canal diameter of ≤8 mm[4]. A central ray was drawn through the most proximate disc space on the sagittal view. Rostral or caudal displacement of the site of maximal stenosis from this central ray was measured on the sagittal image in millimeters, with measurements inferior to the disc space assigned negative values. Note was made of the major cause of the stenosis. We defined the cause of stenosis as that which if corrected would enlarge the canal cross sectional area the most, and classified stenosis as ligamentous hypertrophy, facet hypertrophy, epidural lipomatosis, spondylolisthesis, synovial cyst, or adjacent segment disease associated with a prior fusion at an immediately adjacent level. It was recognized that in many cases, more than one factor contributed to the stenosis (as several of these result simultaneously from primary disc degeneration), but only one factor was assigned as the major cause (Figure 1). All scans were reviewed by the senior author (DAR) and any discrepancies in interpretation were reviewed and adjudicated by the neuroradiologist (JMP). The study was approved by the institutional review board and was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

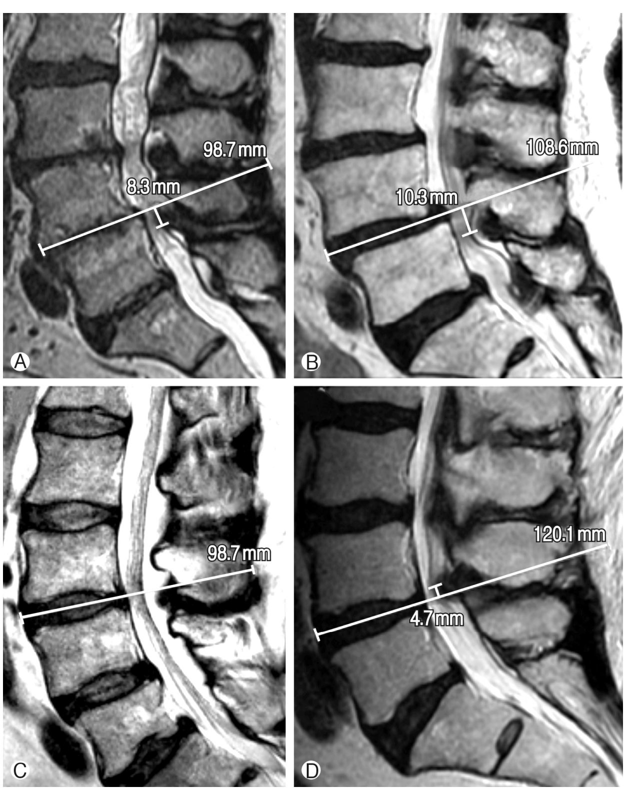

Figure 1. Demonstration examples of the measuring technique and various kinds of stenosis. Ⓐ Maximum stenosis 8 mm distally displaced due to ligamentous hypertrophy. Ⓑ Maximum stenosis 10 mm distally displaced due to spondylolisthesis. Ⓒ Maximum stenosis 0 mm displaced due to epidural lipomatosis. Ⓓ Maximum stenosis 5 mm rostrally displaced due to facet hypertrophy.

3. Statistical Analysis

To evaluate for factors potentially contributing to the amount displacement of the most severe lumbar stenosis from the corresponding disc space, univariate associations were first examined using Pearson's correlation for continuous variables, Spearman correlation for ordinal variables, and Pearson Chi-Square for nominal variables. Factors with statistically significant associations were entered in the multi-variate model as well as their corresponding interaction terms. Iterative grouping of the categorical variables were also performed to maximize associations between the merged variable and the displacement measurements. Factors with significant contributions to displacement measurements in the multi-variate model underwent further ANOVA analysis followed by post hoc Turkey multiple comparisons to determine potential group characteristics. Data is presented as ±standard deviation (SD).

RESULTS

1. Demographics and Stenosis Level

Of the 3,000 original lumbar MRIs, 1,042 levels in 749 separate patients were identified as meeting inclusion criteria. Studies were excluded when there was no significant stenosis at any lumbar level, demonstrated recent trauma, infection, or neoplasm, showed a disc protrusion as the major cause of stenosis, were postoperative, were redundant, or were of poor quality. Age range was 40 to 89 years (mean 65.6±10.7 years). Of the 1,042 levels there were 474 females and 568 males, and 39 cases (3.8%) were at L1-2, 209 cases (20.1%) at L2-3, 368 cases (35.3%) at L3-4, 393 cases (37.7%) at L4-5, and 33 cases (3.2%) at L5-S1.

2. Classification and Cause of Stenosis

Of the 1,042 levels; 217 (20.8%) were classified as being due to facet hypertrophy, 310 (29.8%) to ligamentous hypertrophy, 324 (31.1%) levels to epidural lipomatosis, 117 (11.2%) levels to spondylolisthesis, 58 (5.6%) levels to adjacent segment disease, and 16 (1.5%) levels to a synovial cyst (Table 1). The largest fraction of cases from ligamentous hypertrophy, spondylolisthesis, and synovial cysts was at L4-5, but the largest fraction of lipomatosis, facet hypertrophy, and adjacent segment disease cases was at L3-4.

3. Displacement

Mean displacement from the disc space at levels that were

Table 1. Major cause of cenosis by lumbar level (n=1,042)

		Level (n; %)				
Cause	(n)	L1-2	L2-3	L3-4	L4-5	L5-S1
Lipomatosis	324	20; 6.2	85; 26.2	126; 38.9	86; 26.5	7; 2.2
Ligamentous	310	4; 1.3	42; 13.5	111; 35.8	143; 46.1	10; 3.2
Facet Hypertrophy	217	11; 5.1	54; 24.9	83; 38.2	61; 28.1	7; 3.2
Spondylolisthesis	117	1; 0.9	5; 4.3	18; 15.4	86; 73.5	7; 6.0
Adjacent Segment	58	3; 5.2	20; 34.5	26; 44.8	8; 13.8	1; 1.7
Synovial Cyst	16	1; 6.3	2; 12.5	3; 18.8	9; 56.3	1; 6.3

Table 2. Displacement of maximum stenosis from the disc space by major causative factor

Cause	Percent	Range of displacement (mm)	Mean displacement (mm)	Absolute displacement (mm)
Synovial Cyst	1.5	7 to -5	-0.3	2.8
Lipomatosis	31.1	10 to -11	-1.1	1.6
Ligamentous	29.8	5 to -13	-3.5	3.6
Facet Hypertrophy	20.8	7 to -11	-3.9	4.1
Adjacent Segment	5.6	7 to -11	-4.7	4
Spondylolisthesis	11.2	11 to -12	-4.9	5.2

due to synovial cyst, epidural lipomatosis, ligamentous hypertrophy, facet hypertrophy, adjacent segment, and spondylolisthesis were -0.3 mm (range 7 to -5 mm), -1.1 mm (range 5 to -13 mm), -3.5 mm (range 5 to -13 mm), -3.9 mm (range 7 to -11 mm), -4.7 mm (range 7 to -11 mm), and -4.9 mm (range 11 to -12 mm), respectively (Table 2). Absolute value of the displacement from the disc space at levels that were due to synovial cyst, epidural lipomatosis, ligamentous hypertrophy, facet hypertrophy, adjacent segment, and spondylolisthesis were 2.8 mm, 1.6 mm, 3.6 mm, 4.1 mm, 4 mm, and 5.2 mm respectively (Table 2).

Table 3. A summary of statistical analysis results

Analysis	Displacement	p-value
Univariate	and age	0.009
	and cause	<0.001
	and sex (female)	0.049
	and spine level	<0.001
Multivariate	Cause	<0.001
	Level	0.003

4. Univariate Associations

Significant univariate associations were demonstrated between displacement measurement and patient age ($p=0.009$, more advanced age associated with more negative displacement), lumbar canal stenosis underlying cause ($p<0.001$), sex ($p=0.049$, female sex associated with more negative displacement) and level of disease ($p<0.001$, lower level associated with more negative displacement). Further evaluation with multi-variate linear regression adjusting for interaction terms among patient age, cause of lumbar stenosis, gender, and level of disease confirmed significant contributions to displacement measurements from cause of lumbar canal stenosis and level of disease ($p<0.001$ and $p=0.003$, respectively) but not patient age or sex ($p=0.135$ and $p=0.537$, respectively). Iterative grouping of the categorical variables suggested merging of adjacent segment disease, facet hypertrophy, and ligamentous hypertrophy into one group with a group mean contribution of -3.2 mm as well as merging disease at levels L1-2, L2-3, and L3-4 into one group with a group mean contribution of -2.0 mm (Table 3).

Sub-group analysis showed a common trend of predominantly negative displacement measurements with spondylolisthesis, adjacent segment disease, facet hypertrophy (the synovial joints bilaterally), and ligamentous hypertrophy (the low T2 signal structures internal to the lamina consistent with ligamentum flavum). Epidural lipomatosis (the high T1 and T2 signal internal to the ligamentum flavum and external to the thecal sac) showed a different distribution of displacement values with a significantly higher percentage of stenoses at the level of the disc space (*post hoc* $p<0.01$ compared to spondylolisthesis, adjacent segment disease, facet hypertrophy, and ligamentous hypertrophy; $p=0.038$ compared to synovial cyst). Lumbar stenosis caused by synovial cyst also had a significantly different displacement distribution, evenly distributed among negative, zero, and positive values (*post hoc* $p<0.01$ compared to spondylolisthesis, adjacent segment disease, facet hypertrophy, and ligamentous hypertrophy; $p=0.038$ compared to synovial cyst) (Figure 2).

DISCUSSION

The fastest increasing area of lumbar spine surgery in recent decades is in older patients with lumbar spinal stenosis[2] Minimally

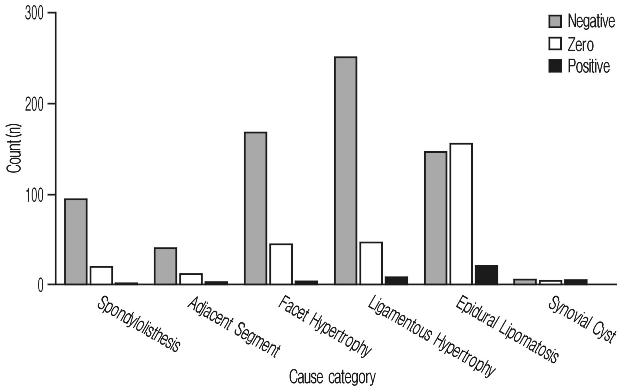

Figure 2. Displacement directionality by cause of spinal canal stenosis.

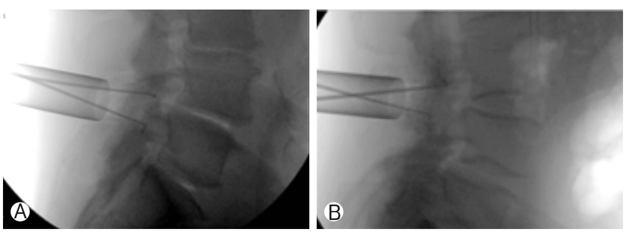

Figure 3. Intraoperative fluoroscopic images showing the position of the operating tubular retractor based upon the location of the pathology. The two instruments show the rostral and caudal extent of the decompression. Ⓐ: Tube directed distal to the disc space for the patient shown in Figure 1 Ⓐ. Ⓑ: Tube directed at the disc space for the patient shown in Figure 1 Ⓒ.

invasive lumbar decompression may have advantages over open surgery, including for example smaller incisions, shorter operative time, shorter hospitalization, lower blood loss, less opiate use, faster recovery, lower systemic stress levels, lower infection rates, less postoperative delirium, and less postoperative instability[3,5-11]. However, working through a small access port has a learning curve[5,12-14]. The small access tube prevents palpation of the anatomy and limits the field of view. A spine surgeon is therefore more reliant upon careful analysis of the preoperative MRI and/or CT imaging and upon fluoroscopic imaging during the procedure to guide the extent of the decompression.

There is little mention in the minimally invasive lumbar decompression literature of the need to carefully assess the location of maximal stenosis in planning the procedure. We have conducted a retrospective review of 3,000 lumbar MRIs over 2 years, and found 1,042 levels showing stenosis defined as an AP diameter of the lumbar canal ≤8 mm[4]. Our data shows that when the stenosis is due to facet and ligamentous hypertrophy, spondylolisthesis, or adjacent segment disease, the area of maximal stenosis is commonly displaced inferiorly from the disc space. If the stenosis is due to lipomatosis, an increasingly common problem in Americans[15] the maximal stenosis is more likely to be at the disc space. For synovial cysts, the most stenotic segment is more widely distributed at, above, and below the disc space. It is critical to correctly orient the retractor so that the area of maximal stenosis is within the accessible field of the operation (Figure 3). The findings would also be important for open interlaminar decompressions and other less invasive variants.

Intra-operative planning is dependent upon obtaining a true lateral fluoroscopic image that is critical to correctly positioning the access tubular retractor. If the view is obliquely oriented, then the surgeon may misinterpret the relationship of the retractor to the area of most severe stenosis (Figure 3). With maximum displacements in our data of 13 mm from the disc space, working through an 18 mm tube without a true lateral view could easily result in failure to decompress the critical area.

Recently, minimally invasive decompression without fusion has been shown to be effective at a non mobile spondylolisthesis[6] Our data indicate that care must be taken to carry the decompression inferior to the disc space to ensure a complete decompression. In studies suggesting that minimally invasive decompression is not as effective as decompression and fusion[16], it will be critical to ascertain if the decompression actually reached the area of most severe stenosis, as failure to do so could account for poorer outcomes in patients who were not fused.

Minimally invasive decompression is also effective in segments adjacent to an instrumented fusion[17]. Adjacent segment decompression can be hampered by protruding rods and arthrodesis bone. It is important to accurately assess the location of maximal stenosis and to plan a tubular trajectory which will avoid collision with the hardware.

It is of interest that almost one third of our cases were judged to be due in large part to epidural lipomatosis. This is becoming an increasing problem, likely in association with the obesity epidemic and steroid exposure[15], but has not been reported to this extent. According to Theyskens, et al.,[18] who conducted a report-based search for the term lipomatosis in the radiologist's reports of 28,902 spinal MRIs, the overall incidence of lipomatosis was 2.5% of which only 0.1% were definitely symptomatic. Lipomatosis was associated with older age, higher modified Charlson comorbidity index, male sex, body mass index >30, Black/African American race, systemic corticosteroid, and epidural corticosteroid injections. In a series of patients operated upon for symptomatic lipomatosis, no correlated factors were found to be significantly different from other spine surgery patients except for a longer duration of symptoms, suggesting that the diagnosis may be delayed[19]. Our study suggests that epidural lipomatosis sufficient to result in stenosis may be more common than previously reported.

1. Limitations

The study is limited by its retrospective nature, by the precise definition of stenosis selected, and by some variability in assigning a major cause of the stenosis, which is often multifactorial. As this study was undertaken with all lumbar MRIs performed in a finite period of time and obtained for a wide variety of reasons, it is not known if any of the findings were symptomatic or if any patients had subsequent surgery of any kind. It was not the intention of this study to correlate the findings with clinical outcomes.

CONCLUSION

In summary, the site of maximal lumbar stenosis is at or near the center of the disc space when the major cause is lipomatosis or synovial cyst, but is often significantly inferiorly displaced from the disc space when ligamentous or facet hypertrophy, spondylolisthesis, or adjacent segment disease is the major cause. Lower lumbar levels and older patients may harbor larger displacements from the disc space. Correctly orienting the access tube for minimally invasive lumbar decompression will help to prevent leaving an undecompressed stenotic segment caudal or rostral to the field of view.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article.

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