Functional and Patient-Reported Outcomes in Symptomatic Lumbar Spinal Stenosis Following Percutaneous Decompression

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Abstract

Background: Neurogenic claudication due to symptomatic lumbar spinal stenosis (LSS) is a painful condition causing significant functional disability. While the cause of LSS is multifactorial, thickened ligamentum flavum (LF) accounts for up to 85% of spinal canal narrowing. Mild percutaneous lumbar decompression allows debulking of the hypertrophic LF while avoiding the morbidity frequently associated with more invasive surgical procedures.

Methods: In this prospective case series study, consecutive LSS patients presenting with neurogenic claudication were treated with percutaneous lumbar decompression. Efficacy was evaluated using the Pain Disability Index (PDI) and Roland-Morris Disability Questionnaire. Pre- and postprocedure Standing Time, Walking Distance, and Visual Analog Score (VAS) were also monitored. Significant device- or procedure-related adverse events were reported.

Results: The mild procedure was successfully performed on forty patients. At twelve months, both PDI and Roland-Morris showed significant improvement of 22.6 points (ANOVA, P < 0.0001) and 7.7 points (ANOVA, P < 0.0001), respectively. Walking Distance, Standing Time, and VAS improvements were also statistically significant, increasing from 246 to 3,956 feet (ANOVA, P < 0.0001), 8 to 56 minutes (ANOVA, P < 0.0001), and 7.1 to 3.6 points (ANOVA, P < 0.0001), respectively. Tukey HSD test found improvement in all 5-outcome measures to be significant from baseline at each follow-up interval. No significant device- or procedure-related adverse events were reported.

Conclusion: This study demonstrated significant functional improvement as well as decreased disability secondary to neurogenic claudication after mild procedure. Safety, cost-effectiveness, and quality-of-life outcomes are best compared with comprehensive medical management in a randomized controlled fashion and, where ethical, to open lumbar decompression surgery.

Key Words: lumbar spinal stenosis, neurogenic claudication, decompression, ligamentum flavum, mild®, percutaneous

INTRODUCTION

Spinal stenosis has significant impact on the patient’s quality of life, and on the healthcare system, as the average age of the population increases. Depending on diagnosis and predominant causal factors, treatment...
can range from conservative nonsurgical therapy to open spine surgery with or without fusion. With these treatment options, the cost burden, and the risk of associated morbidity to the increasingly debilitated patients, can be significant. While the exact incidence and prevalence of lumbar spinal stenosis (LSS) are unknown, a recent community-based study reported, and LSS prevalence of 8.4% and 1.2 million U.S. patients were symptomatic at any given point of time.1

Spinal stenosis, in general, is the narrowing of the central spinal canal and or the neural foramina. Although narrowing does not always result in nerve compression, it can create pressure on the nerves, often resulting in pain or numbness in the region impacted by the compressed nerve or nerves. There are many causes of spinal stenosis, including tumors, congenital defects, physical injury, bone disease, etc., the most prevalent being the aging effects of intervertebral disk degeneration, bone overgrowth, and ligament thickening. Lumbar and cervical areas are most commonly affected. When LSS is suspected, diagnosis is confirmed through symptom differentiation, that is, radicular vs. neurogenic claudication vs. vascular claudication pain, in combination with imaging studies, such as MRI.

Neurogenic claudication due to LSS is a painful condition that causes significant functional limitations, especially in the elderly population.2 Nearly all LSS patients present with symptoms of neurogenic claudication3-8 that is described as low back pain that extends to the buttocks and legs. Neurogenic claudication is a painful, progressive condition that is caused by narrowing of the lumbar spinal canal. In an experimental study in vitro, the dynamic changes in the cross-sectional area of the lumbar spinal canal between L3 and L4 was reduced by 40 mm², corresponding to 16% reduction of the initial spinal canal surface area when the lumbar spines moved from flexion to extension.7 Such narrowing results in compression/ischemia of the nerve roots.8 This explains why neurogenic claudication symptoms worsen with standing or walking (lumbar extension) and improve with sitting or bending forward7-9 (lumbar flexion).

Most patients with neurogenic claudication present with multiple level stenosis, which is generally confirmed through magnetic resonance imaging (MRI) studies.10 Further, review of these patient MRIs usually reveals that the cause of symptomatic LSS is multifactorial in nature. Disk bulge, facet hypertrophy, and invariably, ligamentum flavum (LF) hypertrophy, which compromises the central canal, are commonly found. Clearly, it is not necessary to correct every casual factor in a large population of LSS patients. It has been reported that the majority of LSS patients have hypertrophy of the LF and that its thickness may account for 85% of lumbar spinal canal narrowing.11 Thus, recent development of the percutaneous decompression technique (mild12) to remove and debulk LF makes absolute sense in treating symptomatic LSS, and even more so with vulnerable patients needing minimally invasive surgery where general anesthesia is not required. With this in mind, review of neurogenic claudication patient MRIs should include careful observation of ligament thickness and prominence as a stenotic factor at each affected level.

Historically, prior to resorting to open surgical decompression, conservative management of neurogenic claudication usually culminated in the use of epidural steroid injections (ESIs). ESIs often provide some relief in early or moderate cases, but lose effectiveness over time.12 Also, conservative management and ESIs are not likely to help patients who have already progressed to more severe neurogenic claudication. While ESIs may alleviate some of the inflammation related to radicular pain, it is important to differentiate between pain because of irritation/inflammation of the radicular nerves and neurogenic claudication pain that results from compression or ischemia of neural structures (cauda equina).13 The limited short-term success provided by ESIs in the treatment of neurogenic claudication generally leads to the use of more aggressive invasive surgical decompression that ranges from implantation of interspinous process spacers, minimally invasive laminotomy, or traditional-wide laminectomy with or without fusion or instrumentation.

It is frequently difficult for elderly LSS patients to undergo major surgery because of coexisting morbidities. Prior to the introduction of percutaneous lumbar decompression, these patients had no choice but to seek open surgical decompression for pain relief, or to continue receiving repeated, less-than-effective ESIs.13 The ultra-minimally invasive nature of percutaneous lumbar decompression allows debulking of the hypertrophic LF, by removing very small amounts of bone and soft tissue. Advantages of percutaneous lumbar decompression vs. more invasive surgical techniques include the use of less sedation (general anesthesia not required), faster recovery times, less potential for subsequent spinal instability, and no hardware or implants left behind. Further, percutaneous lumbar decompression patient series have reported significant neurogenic
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Claudication pain relief at long-term follow-up, and no major device- or procedure-related adverse events.\textsuperscript{14-19}

A treatment algorithm now exists ranging from conservative treatment, such as physical therapy and NSAIDs, to epidural steroid injection (ESI) and fluoroscopically guided percutaneous decompression, mild\textsuperscript{20} (Vertos Medical, Aliso Viejo, CA, U.S.A.), with the more invasive, open decompression surgeries with or without fusion as a last resort.

The goal of this study was to report changes in the functional abilities and pain relief for the first 40 consecutive LSS study patients treated with mild percutaneous lumbar decompression at the Pain Management Department of the Cleveland Clinic. Safety and effectiveness measures were recorded at the time of the procedure and throughout the twelve-month follow-up period.

METHODS

In this prospective, IRB approved case series study, all patients presenting to Cleveland Clinic Pain Management Department with symptoms of neurogenic claudication were screened using inclusion/exclusion criteria for percutaneous lumbar decompression (mild procedure). All patients provided informed consent and were subsequently treated with the mild procedure. The fluoroscopically guided percutaneous lumbar decompression procedure has been previously described in detail.\textsuperscript{14,15,17-22} This procedure can be performed unilaterally or bilaterally and at multiple levels, as medically indicated. A preoperative visit and 4 follow-up visits with intervals ranging from approximately 3 months (Visit 1) to 1 year (Visit 4) posttreatment were conducted. This report presents results of follow-up visits conducted through February 2012.

Participants

Requirements for study inclusion were patients with neurogenic claudication and radiographic T2-weighted MRI-confirmed LF hypertrophy $\geq$ 4.0 mm.\textsuperscript{23-25} Failure of conservative treatment was also required with lumbar decompression medically indicated in all cases. The minimum distance any patient could walk unaided before being limited by pain prior to the percutaneous decompression procedure was 10 feet. Excluded from the study were patients having prior surgery at the intended treatment level and/or reporting significant radicular leg pain not related to LSS. Further, current users of anticoagulants, NSAIDs within 7 days, or ESIs within 4 weeks prior to the study procedure were excluded. Presence of mobile or greater than Grade 1 spondylolisthesis was also exclusionary in this study.

Outcome Measures

The primary endpoints in this study were Pain Disability Index (PDI) and Roland-Morris Disability Questionnaire (RMQ). Secondary endpoints included patient Standing Time and Walking Distance prior to experiencing symptoms of neurogenic claudication, as well Visual Analog Score (VAS) for pain. These 5 endpoints were collected at baseline and again at each of the 4 postoperative visits.

Pain Disability Index measures the degree to which neurogenic claudication pain interferes with the patient’s ability to perform daily activities. It is a validated self-reported questionnaire that evaluates functioning in broad areas of life activity. For each of these categories, patients rate their level of pain disability from 0 to 10, with lower scores signifying less disability. Overall PDI scores range from 0 to 70.\textsuperscript{26-28} RMQ is a validated self-assessment tool that measures functional disability. This tool was developed specifically for patients with low back pain,\textsuperscript{29} and it has been shown to be reliable and sensitive to functional status change over time.\textsuperscript{30} RMQ uses a 24-point scale, with lower scores indicating less severe symptoms.

Standing Time is a measure of the number of minutes a patient can stand unassisted before being limited by neurogenic claudication symptoms. Walking Distance is a measure of the number of feet a patient can walk unassisted before being limited by neurogenic claudication symptoms. Both of these measures were self-reported with random objective verification conducted by third-party individuals not directly involved with the patient’s care. VAS used in this study was a 10-point scale in which patients rated pain intensity from zero (no pain) to 10 (worst pain imaginable).

Statistical Methodology

The cohort population in this study is comprised of those patients having procedural, as well as primary and secondary endpoint data available for all 4 posttreatment visits including 3-, 6-, 9-, and 12-month follow-ups. The Intent-to-Treat (ITT) population includes all 40 treated study patients. For the 1-year ITT analysis, the last reported visit for each patient was utilized.
Follow-up evaluation time points were adhered to ± 2 weeks.

The incidence of procedure- or device-related serious adverse events was the primary safety endpoint. All patients were thoroughly evaluated for the safety of percutaneous decompression at the time of treatment, as well as at all follow-up visits.

Primary clinical endpoint mean change from baseline was assessed for PDI and RMQ at each follow-up interval using analysis of variance (ANOVA) with repeated measures and post hoc Tukey HSD test. Changes in Standing Time, Walking Distance, and VAS were similarly analyzed. Patient success was defined as any of the following outcomes: an improvement in PDI, an improvement in RMQ, or no further decompression required at levels treated under the study protocol within the first year following percutaneous decompression. Average fluoroscopy time and length of stay are reported as indications of procedural safety.

RESULTS

Forty consecutive patients met the inclusion/exclusion criteria and were treated with percutaneous lumbar decompression at the Cleveland Clinic Pain Management Clinic from September 2010 through August 2011. Patient age ranged from 53 to 86, with a mean of 72.2 years. This patient series included 25 women (62.5%) and 15 men (37.5%). On average, patients had endured painful neurogenic claudication for 5 years prior to study enrollment, and 8 patients had symptoms for over 10 years. Additional medical comorbidities included radicular pain (13 patients), followed by osteoarthritis of the knee, sacroiliac, and hip joints (5, 4, and 3 patients, respectively). Cardiac disease was reported in 3 patients.

Mean pretreatment LF thickness was 7.1 mm. Percutaneous decompression was performed on a total of 53 levels previously identified via baseline MRI. Twenty-seven patients (67%) had decompression at 1 level, and the remaining 13 patients (33%) underwent 2-level decompression. Of the 53 treated levels, 37 (70%) were treated bilaterally and 16 (30%) were treated unilaterally. Total number of percutaneous decompression procedures was 90. Figure 1 presents treatment levels for these procedures. The number of levels and bilaterality was determined intra-operatively based on obtaining adequate epidurogram (Figure 2).

All mild procedures were completed as planned. Mean fluoro time per patient ranged from 170 to 235 seconds depending on the number of levels and sides decompressed. All patients remained in recovery for 1 to 2 hours and were then discharged on the same day as the procedure.

There were no reports of device- or procedure-related serious adverse events at the time of the procedure, or at any point during follow-up evaluation, and there was no patient mortality during this study. All study patients have been carefully monitored for safety. Six patients have not yet been seen for the 1-year visit, but are included in posttreatment analyses prior to 1 year. Two of these 6 patients underwent subsequent spine surgery during the 1-year follow-up period and were discontinued from the study. Both patients, although improved after the mild procedure,
chose to undergo additional back surgery (1 with fusion and the other with discectomy) to address ongoing radicular pain. The remaining 34 patients were available for all follow-up periods and comprise the cohort population.

The cohort patients experienced a statistically significant improvement in PDI from a mean of 41.4 (95% CI ± 4.6) at baseline to an average of 18.8 (95% CI ± 4.9) at 1-year post-mild procedure, an improvement of 55% (ANOVA, P < 0.0001). Improvement from baseline for this cohort at each interim follow-up period was found to be statistically significant (Tukey HSD test, P < 0.01). Interestingly, this test (Tukey) demonstrated that dramatic improvement occurred by the first postoperative visit and showed a lack of difference between each subsequent postoperative visit, indicating a high degree of consistency of the initial result over time. As with the cohort patients, the mean improvement from baseline for all available patients at each interim follow-up period was found to be statistically significant (see Figure 3). The 1-year ITT analysis of PDI was also found to be statistically significant with improvement of 53% (ANOVA, P < 0.0001).

The RMQ baseline average score of 14.3 (95% CI ± 2.1) showed statistically significant improvement at 1 year in the study cohort, with a mean of 6.6 (95% CI ± 2.0), which represents an improvement of 54% (ANOVA, P < 0.0001). As with PDI, improvement from baseline at each interim follow-up period was found to be statistically significant (Tukey HSD test, P < 0.01). Average improvement in RMQ from baseline for all available patients at each interim follow-up period proved to be statistically significant as well (see Figure 4). The RMQ 1-year ITT analysis was also found to be statistically significant with improvement of 49% (ANOVA, P < 0.0001).

Functional improvement as measured by Standing Time and Walking Distance was statistically significant (ANOVA, P < 0.0001). Standing Time improved from a baseline of 8 to 56 minutes at twelve-month follow-up, while Walking Distance improved from a baseline mean of 246 feet to 3,956 feet at Month 12 (see Figures 5 and 6). Level of pain as measured by VAS improved significantly from 7.1 (95% CI ± 0.8) at baseline to 3.6 (95% CI ± 0.9) at twelve-month follow-up (ANOVA, P < 0.0001) (see Figure 7). The 1-year ITT analyses for Standing Time, Walking Distance, and VAS improvement were also found to be statistically significant (ANOVA, P < 0.0001).

Figure 3. Pain Disability index mean value at each follow-up (N = 34).

Figure 4. Roland-Morris Disability Questionnaire mean value at each follow-up (N = 34).

Figure 5. Mean standing time at each follow-up (N = 34).
DISCUSSION

Proper patient screening and differential diagnosis play a key role in treating the large LSS population. It is well-known that LSS is multifactorial. However, as was previously reported in clinical studies, positive outcomes are achieved with very small amounts of bone and hypertrophic ligament removal using the mild procedure in patients having a combination of facet hypertrophy, osteophytes, disk bulge, spondylosis, and/or foraminal stenosis.\textsuperscript{14,16–19} Clearly, it is not necessary to correct every causal factor in a large population of the LSS patients. It has been reported that the majority of LSS patients have hypertrophy of the LF and that this thickening may account for up to 85% of spinal canal narrowing.\textsuperscript{11} The advancement of minimally invasive procedures to debulk LF without large incisions and without major bone removal to affect stability or mobility of the lumbar spine makes the mild procedure an excellent option for patients with significant neurogenic claudication secondary to LSS.

Ligamentum flavum hypertrophy leading to spinal canal narrowing and painful symptoms of LSS was first described by Elsberg.\textsuperscript{31} A normal LF is 1 to 2 mm thick,\textsuperscript{32} and even slight hypertrophic changes can encroach on the neural elements leading to neurogenic claudication.\textsuperscript{33} Park and colleagues found a significant difference in LF thickness in LSS patients vs. a control group without LSS. They reported a mean LF thickness of 4.44 mm for the LSS patients compared with 2.44 mm in the control group.\textsuperscript{23} While inclusion criteria for this study required MRI confirmation of LF thickness $\geq$ 4.0 mm, mean baseline LF thickness for these patients was as high as 7.1 mm. While minimal increases in LF thickness can cause painful compression of the cauda equina, conversely, very small amounts of LF tissue removal can significantly decrease levels of pain for these patients.

Although nearly all LSS patients suffer from neurogenic claudication,\textsuperscript{3–6} several differentiating tests can identify various findings such as radicular pain, disk herniation, vascular claudication, and neurogenic claudication. For example, bending forward or sitting classically increases pain caused by a herniated disk, while the same movements immediately decrease the pain of LSS. Walking can sometimes relieve disk herniation pain, but pain increases with walking or prolonged standing in the LSS patient.

Given the large and growing elderly population who are either unwilling or unable to tolerate general anesthesia and the potential adverse effects associated with open decompression procedures, it is necessary to consider less invasive options, especially if the cost differential and complication profile greatly favor the minimally invasive option. In addition, as the longevity of this aging population increases, the demand for sustenance of active quality of life and reduced cost of health care is rising.

In this study, the mean 1-year PDI improvement of 22.6 points (55%) from baseline was statistically significant, as was the improvement from baseline at each interim postoperative period. Ninety-three percent of all patients experienced an improvement in RMQ by the first postoperative visit, and 1 year after treatment, cohort patients achieved average improvement of 7.7 points. This change represents a significant improvement in patient condition based on reports of
minimum clinically important difference (MCID) for RMQ. Roland\textsuperscript{14} recommends that a change of 2 to 3 points on the RMQ be considered the MCID. Other reports suggest that a change of 4 to 5 points in the RMQ be interpreted as a significant improvement or decline in patient outcome.\textsuperscript{30,35}

Standing Time was subjectively reported by patients with verification of these measures conducted at random by third-party observation. The statistically significant 47-minute improvement in mean Standing Time for this cohort, from 8 minutes at baseline to 56 minutes at 1-year follow-up, is a sevenfold increase. This compares favorably to a recently published report on 116 neurogenic claudication patients treated with X-STOP. This patient subgroup experienced a 39-minute improvement in Standing Time from 20 minutes preoperatively to 59 minutes at 1-year follow-up, demonstrating only a threefold increase in a relatively less severe spinal stenosis patient population.\textsuperscript{36}

Likewise, Walking Distance was subjectively patient reported and verified at random through third-party observation. All forty patients in this series reported a statistically significant improvement in mobility after \textit{mild} treatment, with mean Walking Distance increased by over two-thirds of a mile (3,575 feet) at 1 year. This increased mobility can be seen as an exceptional enhancement in quality of life when translated to activities of daily living. Standing Time and Walking Distance improvement were significant in the first postoperative follow-up period and interestingly continued to improve over time. It is possible that participation in the posttreatment physical therapy program prescribed for these previously inactive patients contributed to this continued improvement. The initial functionality gain likely enabled increased exercise, resulting in the development of greater endurance over time. The mean VAS improvement from baseline to 1-year follow-up of 3.5 points (49\%) was statistically significant, as was the improvement from baseline at each interim postoperative period. These VAS scores recorded over time represent an important sustained reduction in pain for this patient cohort.

These results are comparable to and compare favorably with 1-year results attained in the \textit{mild} long-term multicenter study\textsuperscript{17} in which pain was reduced in 79\% of all patients, with mean pain reduction of 53\% using the Visual Analog Scale and mobility increase of 34\% using the modified Oswestry Disability Index.

More invasive surgical decompression options available to the LSS patient are varied, ranging from interspinous spacers to open surgery with fusion. In LSS cases where thickened LF is the predominant stenotic factor, some physicians attempt to increase the segmental space available for the dural sac by stretching the LF (eg, interspinous devices) rather than debulking the LF or employing a more extensive surgery. In LSS cases where thickened LF is not the predominant stenotic factor, percutaneous decompression (\textit{mild}) or use of interspinous spacers may be insufficient to decompres the spinal canal. In these cases, when more invasive surgical decompression is necessary and can be tolerated by the patient, the conventional treatment continuum leads to open decompression laminectomy. However, this treatment is associated with high complication rates,\textsuperscript{37} and unfortunately, the laminectomy might compromise the stability of the lumbar spine requiring fusion at a future date. A less invasive unilateral laminotomy for bilateral decompression has been developed for the treatment of LSS that uses either a microscope or endoscope for visualization during the procedure. Recent publications reporting treatment with unilateral laminotomy for bilateral decompression showed similar high complication rates when compared with open surgical decompression.\textsuperscript{38,39} Such invasive surgical decompressive procedures are generally associated with good relief and improved function; however, the abovementioned associated morbidities, cost, and possible complications of general anesthesia\textsuperscript{37–39} make the minimally invasive lumbar decompression a first choice to treat LSS. The authors acknowledge the limitations of this study because of the lack of use of a control and lack of randomization. Study strengths include its prospective design, the use of multiple validated outcome measures for pain and disability, and the use of random objective verification of patient-reported outcomes.

**CONCLUSION**

This study demonstrated significant functional improvement as well as decreased disability secondary to neurogenic claudication at 1 year following percutaneous lumbar decompression. There were no significant device- or procedure-related adverse events reported in this study. Treatment of a prevalent LSS factor, ligament hypertrophy, in the presence of multiple other factors proved to be life-altering. Clearly, by decreasing the pain of neurogenic claudication, it is possible to markedly reduce disability without invasive, biomechanically disruptive procedures. Safety, cost-effectiveness, and quality-of-life outcomes are best
compared with comprehensive medical management in a randomized controlled fashion and, where ethical, to open lumbar decompression surgery.

REFERENCES