The Effectiveness of Decompression Surgery on Low Back Pain in Patients with Central Lumbar Spinal Stenosis

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Abstract

Background: Patients with central lumbar spinal stenosis (CLSS) complain of not only the lower leg symptoms but also low back pain (LBP) simultaneously in many cases. Therefore, patients who undergo decompressive surgery expect recovery from LBP as well as lower leg symptoms, and surgeons who perform decompression surgery are making efforts to improve both symptoms. The objective of this study is to investigate whether decompression surgery can improve low back pain and symptoms of lower limb pain in patients with one level central lumbar spinal stenosis.

Methods: The present study included 39 patients who had findings of central lumbar spinal stenosis and underwent decompression surgery due to its corresponding claudication and lower leg radiating pain complaints from 2013 to 2018. Their pain (lower leg radiating pain and low back pain) and functional outcomes (Oswestry Disability Index (ODI), Roland–Morris Disability Questionnaire (RMDQ), and Short Form-36 (SF-36)) were evaluated before surgery and 6 and 12 months after surgery.

Results: Mean lower leg radiating pain continuously showed statistically significant improvement ($p < 0.05$, $p = 0.003$); however, the clinical significance of differences above minimum clinically important difference (MCID) was up to 6 months. Mean low back pain was 4.72 ± 3.40 before surgery, 2.33 ± 2.27 at 6 months after surgery, and 2.21 ± 2.02 at 12 months after surgery, showing statistically and clinically significant improvement ($p < 0.05$) up to 6 months after surgery, after which there were no findings of improvement.

Conclusion: Decompression surgery for patients with central lumbar spinal stenosis showed clinically significant improvements in lower leg radiating pain and low back pain up to 6 months after surgery and continuous improvements in lower leg radiating pain up to 12 months, but there was no continuous improvement in LBP.

Introduction

Lumbar spinal stenosis (LSS) is a degenerative disease causing neurogenic intermittent claudication (NIC), lower leg radiating pain (LLRP), sensory deficit or motor weakness of the lower leg, and LBP due to encroachment on the cauda equina and its nerve root, and the number of patients is also increasing with the rise in elderly population. These symptoms worsen when lumbar extension causes increased encroachment on the cauda equina, and symptoms are relieved by lumbar flexion. Accordingly, because sudden and severe neurological abnormality in patients with LSS is very rare and functional loss develops gradually, conservative treatment is primarily applied as a general rule [1], but in case of failure of that conservative treatment, decompression surgery is the standard surgical method [2]. When the chief complaints of patients are LLRP or NIC before surgery, they can be the best indication for surgery [3], and various authors have reported good results in improvements of LLRP or NIC [4–8].

There are cases in which some patients with lumbar foraminal stenosis complain of lower leg symptoms only, but most of the patients with CLSS complain of not only the lower leg symptoms but also LBP

symptoms simultaneously in many cases. Therefore, patients who undergo surgery expect recovery from LBP as well as lower limb symptoms, and surgeons who perform decompression surgery are making efforts to improve both symptoms. However, as LBP can be affected by various other factors, there is a great deal of disagreement on improvement of LBP after surgery. Many authors reported that, with more correct diagnosis and surgical indications applied, a single procedure of decompression surgery for CLSS can improve LBP up to approximately 60% as well as lower leg pain in the first year after surgery [3, 9–11]. But some authors insist that serious LBP is a contraindication for surgery because it can cause poor surgical results [12–14], and Spetzger et al. [15] claimed that during the procedure of decompression surgery, persistent retraction of the multifidus muscles can cause muscle damage, as a result of which muscle atrophy occurs and LBP deteriorates accordingly [15–17].

In this regard, this study aimed to investigate whether LBP as well as lower leg symptoms can also be improved in patients with CLSS by one-level lumbar decompression surgery, based upon the study results by other authors [18–21] that encroachment on the cauda equina itself can be a cause of LBP in CLSS.

Materials And Methods

Patient selection

Among patients who underwent one-level lumbar decompression surgery by a spine surgeon in this hospital from January 2013 to December 2018, 39 patients who met the inclusion/exclusion criteria (Table 1) were selected for a retrospective study. All patients had findings of CLSS at one level in magnetic resonance imaging and complained of the corresponding claudication and LLRP. This study also included patients who underwent surgical treatment (decompression surgery using laminotomy) because there was no improvement after conservative treatments such as administration of medicine, selective nerve root block and so on.

Table 1
Inclusion /Exclusion Criteria

Inclusion Criteria	
1	Single level central lumbar spinal stenosis seen on MRI corresponding to lower leg symptom level
2	Primary lower leg symptom (Lower leg radiating pain and neurogenic intermittent claudication)
3	Failure of at least 3 months of medical management, which included physical therapy, epidural injections, selective nerve root block, oral anti-inflammatory drugs and opioid analgesics.
4	No visible instability requiring instrumentation in dynamic X-ray before surgery.
Exclusion Criteria	
1	Prior lumbar surgery
2	Cauda equine syndrome
3	Recurrent lumbar spinal stenosis
4	Presence of multi-level central lumbar spinal stenosis (longer than two levels)
5	Rapid progressive severe motor deficit (less than grade 3 of 5)
6	Patients with secondary compensation (active medical or workmen's compensation lawsuit)
7	Vertebral fractures, spine infection or tumor, inflammatory spondyloarthropathy, pregnancy
MRI: Magnetic Resonance Imaging	

Study interventions

Standard open decompression was applied to all surgeries, wherein paraspinal muscles were retracted after a midline skin incision using the approach to the interlaminar space. We perform a decompression "over the top" to the central canal. In some patients, partial excision was done in the medial border of the superior articular process to make space for decompression. In all cases, a unilateral approach was made to the area of more severe lower leg symptoms and unilateral partial laminotomy was performed. Decompression of the affected cauda equina and nerve root was then confirmed through the space by removal of the lamina, and a probe was used to confirm that the affected nerve root is freely mobile. All surgeries were performed under microscopic guidance and patients started ambulation while wearing a corset 2 days after surgery. Wearing a corset was recommended for 2–4 weeks to reduce trunk motion and weaning a corset was recommended during this period gradually. The back muscle stretching and strengthening exercise was performed 4 weeks after surgery.

Clinical outcomes (LBP and functional outcomes) and quality of life measurements

The intensity of LBP and LLRP was measured using a 10-mm visual analog scale (VAS), where 0 point meant no pain and 10 points meant the worst conceivable pain [14]. The two types of pains were

measured before surgery and 6 and 12 months after surgery. Functional outcomes were assessed by Oswestry Disability Index (ODI) and Rolland-Morris Disability Questionnaire (RMDQ) at 6 and 12 months after surgery. An additional Short Form-36 (SF-36) was used for quality of life assessment at 6 and 12 months after the surgery. The minimum clinically important difference (MCID) is 2.2 points for LBP-VAS, 5.0 points for LBP-LLRP by Parker et al. [22].

Results

Epidemiological results

The average age of 39 patients (13 men and 26 women) was 70 ± 6.85 (range, 56–85). The mean age of males was 66.46 ± 6.10 (range, 56–77) and that of females was 71.77 ± 6.51 (range, 56–85). As for surgical sites, 6 patients were operated at L3–4, 24 at L4–5, and 9 at L5–S1 (Table 2). There were no cases of visible instability on dynamic X-ray until the last follow-up.

Table 2
Epidemiological Results

		Number	Mean ± S.D.
Sex	Male	13	66.46 ± 6.10
	Female	26	71.77 ± 6.51
Level	L3-4	6	
	L4-5	24	
	L5-S1	9	

Time course and improvement of the two types of pain

Mean LLRP was 7.62 ± 2.23 before surgery, 2.85 ± 2.62 at 6 months after surgery, and 1.36 ± 1.37 at 12 months after surgery. LLRP showed a statistically significant decrease at 6 months after surgery compared with that before surgery, and at 12 months after surgery compared with that 6 months after surgery ($p < 0.05$, $p = 0.003$). However, the VAS difference between before surgery and that 6 months after surgery was more than the MCID, but LLRP at 6 and 12 months after surgery was less than MCID, which had no clinical significance.

Mean LBP was 4.72 ± 3.40 before surgery, 2.33 ± 2.27 at 6 months after surgery, and 2.21 ± 2.02 at 12 months after surgery. Although there was a statistically significant decrease in LBP at 6 months after surgery compared with that before surgery ($p < 0.05$), the decrease in LBP from 6 to 12 months after surgery had no statistical significance ($p = 0.795$). It showed a clinically significant decrease in LBP 6 months after surgery compared with that before surgery (Table 3).

Table 3
Time course and improvement of the 2 types of pain

	Period	VAS	P value
LBP	Initial	7.62 ± 2.23	
	6month	2.85 ± 2.62	< 0.05*†
	12month	1.36 ± 1.37	0.003*
LLRP	Initial	4.72 ± 3.40	
	6month	2.33 ± 2.27	< 0.05*†
	12month	2.21 ± 2.02	0.795
* VAS: Visual Analogue Scale, LBP: Low Back Pain, LLRP: Lower leg radiating pain, *: p < 0.05,			
†: > Minimum Clinical Importance Difference			

Table 4
Time course and improvement of the functional outcome and quality of life.

	Period		P value
ODI	Initial	23.33 ± 9.06	
	6month	19.92 ± 9.33	0.11
	12month	16.33 ± 8.96	0.091
	Initial – 12month		0.001*
RMDQ	Initial	10.63 ± 6.44	
	6month	9.85 ± 5.52	0.572
	12month	8.45 ± 4.96	0.252
	Initial-12month		0.106
SF-36 PCS	Initial	23.82 ± 15.40	
	6month	31.09 ± 18.38	0.066
	12month	39.19 ± 20.57	0.074
	Initial-12month		< 0.05*
SF-36 MCS	Initial	34.22 ± 19.25	
	6month	42.01 ± 21.78	0.103
	12month	48.62 ± 23.28	0.206
	Initial-12month		0.004*

* ODI: Oswestry Disability Index, RMDQ: Rolland-Morris Disability Questionnaire, SF-36 PCS: Short Form-36 Physical Component Score, SF-36 MCS: Short Form-36 Mental Component Score, *: $p < 0.05$.

Time course and improvement of the functional outcome and quality of life

The functional outcome, mean ODI, was 23.33 ± 9.06 before surgery, 19.92 ± 9.33 at 6 months after surgery, and 16.33 ± 8.96 at 12 months after surgery, which showed no statistically significant improvement at each time point ($p = 0.11$, $p = 0.091$). Another functional outcome, mean RMDQ, was 10.63 ± 6.44 before surgery, 9.85 ± 5.52 at 6 months after surgery, and 8.45 ± 4.96 at 12 months after surgery, which did not show statistically significant improvement by period ($p = 0.572$, $p = 0.252$). However, ODI showed significant improvements statistically and clinically at 12 months after surgery compared with that before surgery ($p = 0.001$), and RMDQ did not show statistically significant improvements before surgery and 12 months after surgery ($p = 0.106$)

Regarding the assessment of quality of life, mean SF-36 Physical component score (PCS) was 23.82 ± 15.40 before surgery, 31.09 ± 18.38 at 6 months after surgery, 39.19 ± 20.57 at 12 months after surgery, which did not show statistically significant improvement by each period ($p = 0.066$, $p = 0.074$). Mean SF-36 Mental component score (MCS) was 34.22 ± 19.25 before surgery, 42.01 ± 21.78 at 6 months after surgery, 48.62 ± 23.28 at 12 months after surgery, which did not show statistically significant improvement by each period ($p = 0.103$, $p = 0.206$). In contrast, there was statistically significant improvement in both SF-36 PCS and MCS at 12 months after surgery compared with those before surgery ($p < 0.05$, $p = 0.004$).

Discussion

There are numerous causes of LBP, so it is difficult to clearly identify the exact cause [23]. Ko et al. [3] reported that they are related to the cross sectional area (CSA) and the sedimentation sign (SedSign) but have little to do with SedSign and LLRP, LBP, and NIC, and they insisted that the spinal area has nothing to do with lower leg pain and LBP except for severe spinal stenosis. However, according to many authors, the narrower CSA is in CLSS, the more pressure is applied to the nerve root in the cauda equina [24]. It is known that such central compression of the cauda equina is extensively involved in LBP as well as LLRP and NIC [19–21]. This is in line with the fact that there is severe LBP among the “red flag” symptoms of the cauda equina syndrome due to the severe cauda equina compression [25]. According to Ogikubo et al. [23], the extent of lower leg pain is more severe than that of LBP mainly in LSS, and because lower leg pain can deteriorate by actions such as ambulation and changes in posture, the symptoms in the lower legs are emphasized. In addition, they reported that the narrower the CSA is in the spinal canal, the shorter the walking distance is with severe lower leg and LBP, which led to the poor quality of life. Although LBP can be caused by degenerative intervertebral joints apart from the direct cauda equina compression, Ogikubo et al. [23] claimed that improvements in LBP as well as LLRP are considered feasible if the CSA within the spinal canal is extended.

Jones et al. [19] presented that decompression surgery for LSS is effective in improving LBP up to 6 weeks after surgery in patients with central and lateral recess spinal stenosis and in some patients even with spondylolisthesis and scoliosis. But as studies on pain and functional outcomes were conducted 6 weeks and a year after surgery, the changes between the periods were not available and the methods of surgery are almost half-mixed with a laminectomy, in which the spinous process and nearly total laminal are removed, and a laminotomy, which is the partial removal of the lamina. In addition, it is doubtful whether patients with LBP can recover in up to 6 weeks due to pain by muscle damage in surgery; however, the conclusion is that there was statistically significant improvement 6 weeks and a year after surgery, and the outcomes were similar to those of the authors in that they were more than MCID at 6 weeks and less than MCID at a year after surgery.

However, back muscle injuries in surgery can affect LBP after surgery. Decompression through the standard open posterior approach that we conducted is the most commonly used decompression technique; however, there is much manipulation of paraspinal muscles, and iatrogenic muscle damage is

caused due to detachment and retraction of muscles. There is a study in which as for one-level lumbar decompression, there is no relationship between LBP and muscle damage [26], and some authors [10, 15] reported that LBP continues up to 4 years after surgery and shows improvement after that. Several researchers claim that over time after surgery, degeneration and denervation of damaged back muscles, particularly multifidus muscles after surgery progress, can cause atrophy and fatty degeneration of muscles and at last can be a cause of LBP [26–29]. It was observed in this study that one-level lumbar decompression surgery reduces LBP in patients up to 6 months after surgery, whereas it slowly deteriorates for 6 months afterward.

There are several limitations in this study. First, the follow-up period of 1 year is somewhat too short, but as the period increases, LBP can be affected by various causes such as degeneration of paraspinal muscles and facet joints and degeneration of the intervertebral discs besides decompression surgery itself of LSS. In this regard, the period was limited to a year. Second, no evaluation was made on what causes LBP apart from decompression of LSS. Specifically, the analysis was insufficient with respect to the relationship between stenosis of the CSA within spinal canal in the radiographic imaging before surgery and symptoms of patients, and between decompression of the CSA after surgery and symptoms of patients. Therefore, obtaining and analyzing the additional imaging data after surgery will be required. Third, the sample size was too small with 39 cases and as this is a retrospective study without a control group which is the weakness of this research, it is considered that an extensive and prospective research will be required afterward.

Conclusion

Patients with one level CLSS showed a clinically significant improvement in lower leg pain only by decompression surgery, up to 6 months after surgery and showed continuous improvement in pain up to a year after that, but as for LBP, there was a clinically significant improvement up to 6 months after surgery, but no improvement up to a year after that. Clinical improvements in LBP and lower leg pain can be expected by surgery for mono-segment CLSS up to 6 months after surgery, and lower leg pain shows improvement up to a year after that, but an improvement in LBP is not expected. With a continuous improvement of LLRP and an improvement of LBP up to 6 months after surgery, functional outcomes and improvement of quality of life are also expected as time passes a year after surgery.

Abbreviations

CLSS
Central lumbar spinal stenosis
LBP
Low back pain (LBP)
ODI
Oswestry Disability Index
RMDQ

Roland–Morris Disability Questionnaire
SF-36
Short Form-36
MCID
Minimum clinically important difference
LSS
Lumbar spinal stenosis
NIC
Neurogenic intermittent claudication (NIC)
LLRP
Lower leg radiating pain
VAS
Visual analog scale
PCS
Physical component score
MCS
Mental component score
CSA
Cross sectional area
SedSign
Sedimentation sign

Declarations

Ethics approval and consent to participate: This research has been approved by the IRB of the authors' affiliated institutions (IRB No.: CR-20-096).

Consent for Publication: Not applicable

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