Correlation Between Clinical Improvement and Dural Sac Cross-Sectional Area Expansion in Biportal Endoscopic Lumbar Decompression

Ju-Eun Kim, MD, Daniel K. Park, MD,† and Eugene J. Park, MD‡*

Study Design: Retrospective study.

Objective: To correlate the changes in the dural area on MRI and clinical outcome after unilateral biportal endoscopic (UBE) decompression.

Summary of Background Data: Clinical outcomes after UBE decompression have been published for up to 2 years for patients with isolated spinal stenosis at 1 level. Serial dural expansion after UBE decompression has not been published as well as correlation to clinical outcomes.

Method: We retrospectively reviewed the clinical and radiologic outcomes of 86 patients who underwent UBE decompression for spinal stenosis. Preoperative and postoperative visual analog score (VAS) and Oswestry Disability Index (ODI) were analyzed, and MRI was used for radiologic evaluation before surgery, 3 days after surgery, and 2 years after surgery. The correlation of dural spinal area CSA (preoperative-final) and difference of clinical outcome (preoperative-final) were analyzed.

Result: None of the 86 patients had permanent neurological complications. Back VAS, leg VAS, and ODI showed improvement in symptoms postoperatively and 2 years postoperatively. The postoperative CSA of the dural sac on MRI was statistically significantly increased after surgery at all time points. VAS leg was moderately correlated with change in CSA, while ODI and VAS back were weakly correlated. Correlations were all statistically significant.

Conclusion: UBE decompression showed good clinical outcomes similar to previous studies, and the CSA of the dural sac on MRI significantly increased in the late postoperative phase compared with the early postoperative phase. This technique is viable option to achieve radiographic dural expansion and improvement in clinical outcomes in degenerative lumbar spinal stenosis. However, there is at best only a moderate correlation with change in CSA and clinical outcomes.

Key Words: endoscopic, spinal stenosis, laminectomy

(*Clin Spine Surg* 2025;00:000–000)

Degenerative lumbar disease is increasing in an aging society. Patients may thus experience a deterioration in quality of life and an increase in functional disability.[1] The main symptoms of degenerative spinal stenosis are claudication and radicular pain. The basic treatment for resultant spinal stenosis is conservative treatment; however, if there is no improvement despite conservative measures, surgery may be indicated. The goal of surgery is to relieve symptoms by decompressing the neural elements in the central, foraminal, and/or lateral areas.

A surgical technique that has been introduced as an alternative surgical means to decrease surgical insult is a unilateral laminotomy with bilateral decompression using a microscope. This minimally invasive spinal surgery technique minimizes the violation of the facet joint and takes a unilateral approach, so bilateral decompression can be performed while preserving the soft tissue on the contralateral side.[2,3], However, microscopic decompression is typically performed through a tubular retractor, constraining the working space. The biportal endoscopic (UBE) technique, in which an arthroscope is applied to spine surgery, was introduced in recent years.[4] Studies have demonstrated the efficacy of UBE techniques in degenerative spinal pathology.[5–7] The advantages of this technique include providing a wider view than conventional microscopic techniques with the freedom of movement of the viewing and working channels. In addition, transient muscle ischemia due to the insertion of a tubular retractor is lessened. Studies have shown that UBE is an alternative to microscopic decompression in spinal stenosis and is not inferior to clinical outcomes.[5,6]

However, until now, only the clinical outcome of UBE has been studied. The authors know of only 1 study that analyzed the postoperative expansion of the dura after UBE,[8] but no studies have correlated if a change in the dural cross-sectional area (CSA) is related to clinical outcomes in UBE. While Heo et al[8] found a significant expansion of CSA in 46 patients, a postoperative expansion was only evaluated on postoperative day 2. Previous studies demonstrated postoperative dural expansion

Received for publication December 14, 2024; accepted February 17, 2025.

From the *Baroseomyeon Hospital, Busan, South Korea; †Michigan Orthopedic Surgeons, Professor Orthopedic Surgery, Southfield, MI; and ‡Department of Orthopedic Surgery, Kyungpook National University Hospital, Kyungpook National University, School of Medicine, Daegu, South Korea.

The authors declare no conflict of interest.

Reprints: Daniel K. Park, MD, Michigan Orthopedic Surgeons, Professor Orthopedic Surgery, 26025 Lahser Road, Southfield, MI 48033 (e-mail: danparkmd@gmail.com).

DOI: 10.1097/BSD.0000000000001789

continues past the initial postoperative course.[9] Furthermore, Heo et al[8] demonstrated an expansion of 391.5 ± 93.5 mm^2–723.3 ± 100.8 mm^2 in a tubular group and 398.7 ± 97.8 mm^2–719.5 ± 116.4 mm^2 in the UBE group. While their technique measured the expansion on axial cuts, these values are much higher than other published studies both at baseline and postoperatively.[9–11] Correlations of CSA to clinical scores have been mixed. Many studies have not found a correlation with preoperative CSA to postoperative clinical outcomes.[11–13] Postoperatively, Hermansen et al[14] found a significant correlation between expansion ratio of CSA and self reported effect of surgery. Yamazaki et al[15] also found poor dural expansion was a factor associated with poor clinical outcomes. In this study, we aimed to analyze the change in CSA before and after UBE and observe if there is a correlation with clinical outcome measures.

MATERIAL AND METHOD

This study was conducted in compliance with the principles of the Declaration of Helsinki. The protocol of this study was reviewed and approved by the Institutional Review Board. Eighty-six patients were enrolled who underwent unilateral bilateral decompression using UBE from January 2019 to January 2021. All patients were treated with decompression only by 1 surgeon (J.K.). During this time frame, 151 patients underwent primary interlaminar decompression. Twenty-one patients were lost to follow-up, and 44 patients underwent multilevel decompression. Eighty-six patients remained for the study and followed up 100% at minimum 2 years.

All patients' clinical outcomes were based on electronic medical records, and MRI scans were performed before surgery (within 3 mo of surgery), 3 days after surgery, and 2 years after surgery. The surgery was performed under general or epidural anesthesia, depending on the patient's condition. The technique has been reported previously,[7] but briefly, the patient was placed in a slightly flexed position with the abdomen pressure-free to widen the interlaminar space. The interlaminar space was the working space. Two incisions were made 2–3 cm apart centered around the working space. Arthroscopy and surgical instruments are accessed through the viewing portal and the working portal, respectively. The multifidus muscle above the lamina was slightly dissected with a small Cobb elevator to create an initial space. Using a radiofrequency ablator, the bony landmarks were identified demarcating the interlaminar space. Decortication of the lamina was performed using an arthroscopic burr, Kerrison punch, and osteotome. The ligamentum flavum was removed after the bony work was completed. After checking the dural elements were free of compression, a drain was inserted to prevent postoperative epidural hematoma and removed (Fig. 1).

The inclusion criteria for surgery are as follows:[1] Central stenosis or lateral recess stenosis without foraminal stenosis.[2] Single-level decompression for refractory neurological symptoms to conservative treatment.[3] Up to Meyerding grade 1 degenerative spondylolisthesis with < 3 mm of motion on flexion and extension. Patients with isolated foraminal stenosis, spondylolisthesis Meyerding grade II or higher, Isthmic spondylolisthesis, and dynamic motion (> 5 degrees angulation or 3 mm translation) were excluded. Furthermore, patients with active infection, fracture, or previous spinal surgery at the same level were excluded.

Clinical outcomes were investigated using the Oswestry Disability Index (ODI) and visual analog scale (VAS). VAS and ODI were examined before surgery, 2 weeks, 2 months, and 2 years after surgery. MRI scans were performed using standardized protocols tailored to a 1.5-T Siemens Magnetom Avanto scanner (Siemens, Erlangen, Germany). Sagittal T2 and axial T2 images of the lumbar spine were acquired. Slice thickness was 3.5 mm for the sagittal series and 4 mm for the axial series. The patient's radiologic evaluation included preoperative MRI (within 3 mo of surgery), postoperative day 3, and at the final follow-up (2 y after surgery). For morphometric analysis, the dural CSA of T2 weighted axial MRI was examined. Dimension measurements were made at the facet joint level of each intervertebral space, as this area is the most commonly involved area in degenerative lumbar spinal stenosis.[16] CSA was measured on the axial T2 im-

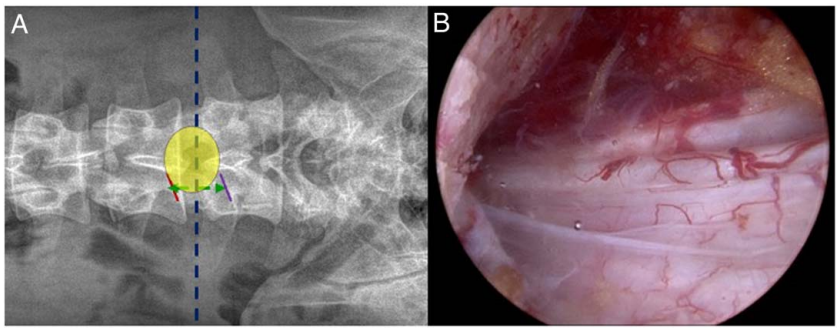

FIGURE 1. A, Location of the viewing portal and working portal, which was performed with bipolar endoscopic interlaminar decompression of L3–4 on the left side. Blue dot line-center of disc space of interest. Red line-proximal portal (initial viewing portal), Purple line-distal portal (initial working portal), and Yellow circle-the working area (interlaminar space). B, Intraoperative view of the decompression. full color online

TABLE 1. Demographic Data of the Cohort

Characteristics	Total, n (%)
Patient number	86
Age (y)	63.5 ± 10.5
Sex (male:female)	41:45
Follow-up time (mo)	26.3 ± 9.2
BMI (Kg/m^2)	25.81 ± 3.25
Smoking history (yes)	26/86 (30.23)
Hypertension	20/86 (23.25)
Diabetes	15/86 (17.44)
Osteoporesis (T score < −2.5)	19/86 (22.09)

ages taken parallel to the operated disc. The CSA measured was the narrowest area in the operated level. Area dimensions were expressed in square millimeters. A spine surgeon and a radiologist independently evaluated all MR images according to a predefined protocol.

Clinical outcome and radiologic outcome were investigated using repeated measured ANOVA tests, and correlations between clinical and MRI parameters were evaluated with Pearson correlation analysis. The paired-sample *T* test was performed to investigate differences in interobserver and intraobservation. All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) version 22.0 (SPSS Inc., Chicago, IL).

RESULT

A total of 86 patients were enrolled in this study. There were 41 males and 45 females. The mean age was 63.5 years (range: 43–87) and the mean follow-up period was 36.3 ± 9.2 months (Table 1). The operation level with the most cases was L4–5, with 34 cases (39.5%), followed by L3–4, with 23 cases (26.7%), and L5–S1 with 21 (24.4%) cases. Operation time per level was 50.5 ± 16.3, time to ambulation was 6.9 ± 2.5 hours, and length of hospital stay was 3.6 ± 1.3 days.

In all 86 patients, preoperative leg VAS, back VAS, and ODI showed sequential improvement compared with before surgery at 2 weeks, 2 months, and the last follow-up. The average VAS leg was 7.24 ± 0.78 preoperatively, 2.56 ± 1.01 at 2 weeks after surgery, 1.87 ± 0.61 at 2 months, and 0.95 ± 0.83 at the last follow-up after 2 years. VAS back significantly improved from 5.38 ± 0.77 preoperatively to 3.34 ± 0.96 at 2 weeks after surgery, 2.38 ± 0.88 at 2 months, and 1.33 ± 1.01 at 2 years. With respect to ODI, the mean ODI significantly improved sequentially from 56.24 ± 7.25 preoperatively to 25.43 ± 6.83 at 2 weeks after surgery 20.79 ± 6.99 and to 13.95 ± 7.15 at 2 years (Fig. 2).

Preoperative CSA changed from 61.0 ± 24.1 mm^2 to 149.8 ± 44.4 mm^2 on the third day after surgery and to 160.1 ± 44.3 mm^2 at the final follow-up. There was a significant change over time ($P < 0.05$), but there was no difference between postoperative day 3 and final follow-up. (Fig. 3). There were no significant differences in interobserver and intraobserver reliability when measuring the CSA.

The correlation between the changes in clinical outcome and CSA from preoperative to postoperative last

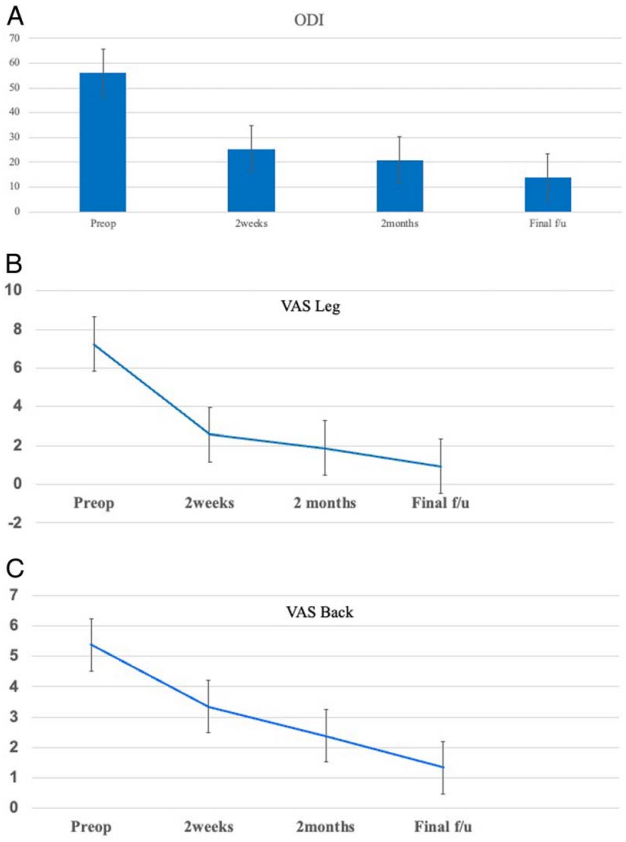

FIGURE 2. A, Graph demonstrating Oswestry Disability Index over time. B, Line graph showing VAS leg over time. C, Line graph depicting VAS back over time. full color online

follow-up CSA was that VAS leg ($P = 0.002$) and ODI ($P = 0.001$) were statistically significant. The VAS back was also correlated with a change of CSA ($P = 0.032$) (Table 2). However, the correlation between improvement in CSA and ODI was weak, with a correlation coefficient of 0.209 and even weaker with a VAS back of 0.131. The correlation between the VAS leg was moderate at 0.365.

Revision surgery occurred in 2 cases: 1 case underwent UBE interbody fusion due to foraminal stenosis at the site of L5–S1 decompression, which was conducted by interlaminar decompression at the same level, and 1 case underwent decompression at the adjacent segment L3–4 due to additional spinal stenosis after decompression at L4–5. Perioperative complications included incidental dural tear in 1 case and transient ankle dorsiflexion weakness in 1 case. The weakness was resolved without the need for additional surgical treatment (Table 3).

DISCUSSION

The goal of surgical treatment in degenerative lumbar spinal stenosis is to relieve neurological compression, usually due to thickened ligamentum flavum and hypertrophic facets. Theoretically, if the narrowed dura CSA increases after decompression, clinical symptoms such as VAS and ODI should improve. Several previous studies

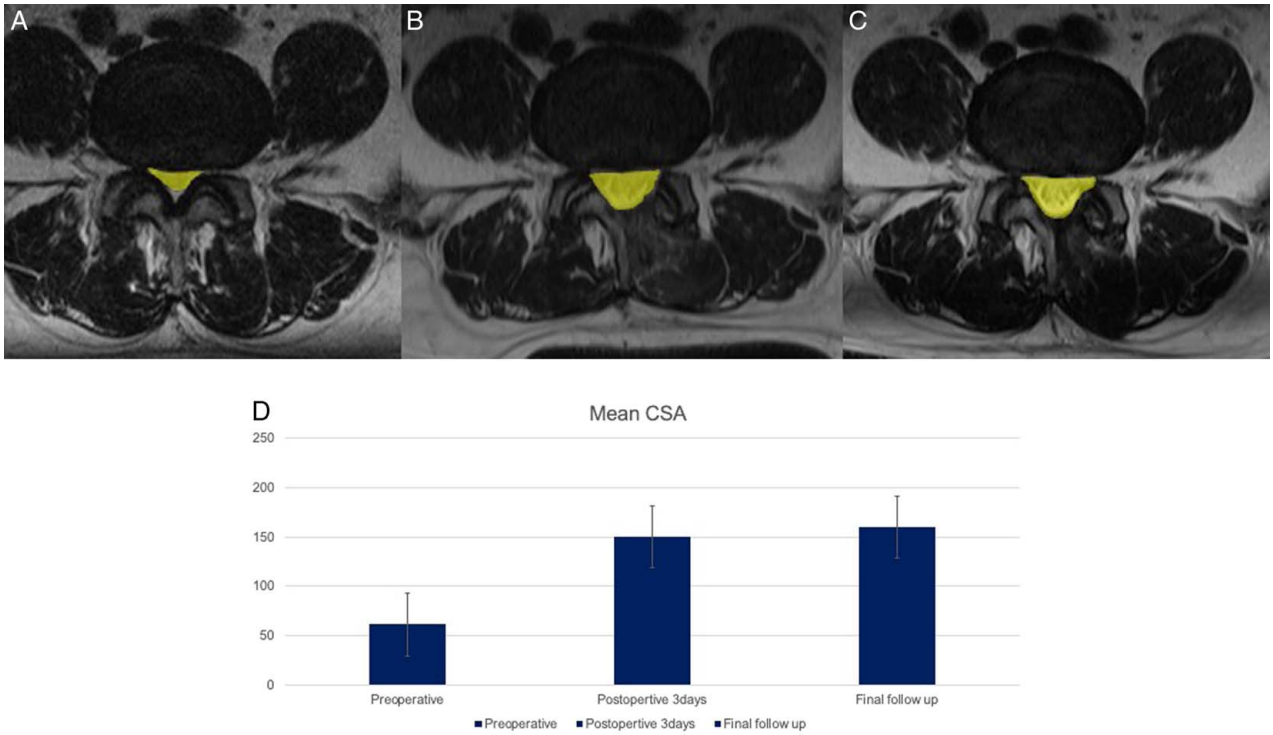

FIGURE 3. A, CSA over time on the same patient. A, Preoperative. B, Postoperative day 3. C, Final follow-up. D, Bag graph showing the CSA value over time. full color online

have analyzed the correlation between spinal stenosis severity and patient symptoms, both qualitatively and quantitatively, but none have analyzed the relationship in UBE.[9,12,17,18] In this current study, we found VAS leg correlated moderately with change in CSA while ODI and VAS back weakly correlated with dural expansion, although all correlations were statistically significant.

Multiple previous studies failed to establish a correlation between stenosis grading preoperatively and patient clinical disability.[13,19–22] Marawar et al[21] also did not find any correlation in patients with multilevel stenosis and even if the CSA was critical. In contrast, Ogikubo et al[23] did find a correlation with smaller CSA and walking distance, leg and back pain, and lower quality of life. Yukawa et al[10] found if CSA was less than the critical level 70 mm^2, there was significant correlation with ODI. Lastly, Caprariu and colleagues found only a weak to moderate correlation between preoperative MRI parameters and ODI. The severity of the stenosis weakly correlated with initial ODI but was not a predictor of surgical success.[24] This inconsistent correlation between CSA and clinical symptoms is not surprising as many people have stenosis but are asymptomatic,[25] variation in canal sizes throughout the population, dynamic compression which cannot be measured in a static MRI, and lack of an accepted system for quantifying stenosis.

When comparing postoperative expansion and clinical improvement, Futatsugi and colleagues, in a multivariate regression analysis, reported that smaller postoperative CSA was significantly associated with early postoperative radicular pain. Each decrease in 10 mm^2 increased the odds 1.26× for postoperative pain. They also found a cutoff of 67.7 mm^2 for radicular pain.[18] Sokolowski et al[26] found a critical ratio was important in determining postoperative outcome. Hermansen et al[14] also reported a correlation between expansion ratio and self reported surgical improvement while Yamazaki et al[15] found poor dural expansion a factor for poor clinical results. In contrast, Herno et al[27] did not find a correlation of postoperative CSA and postoperative outcomes. Rapala et al[28] also stated that there was no correlation between clinical symptom improvement and changes in dural sac area. Chung et al[11] also did not find a correlation between perioperative expansion ratio and clinical outcomes.

All the previous reports discussed above have not inquired if relationships exist in patients who underwent UBE decompression. UBE decompression has shown clinical success up to 2 years postoperatively for lumbar decompression.[7] In this study, we found a statistically significant correlation between postoperative expansion

TABLE 2. Correlation Between Clinical Measures and CSA

	ODI change	VAS leg change	VAS back change
Correlation between change in CSA and clinical outcomes	0.209 ($P=0.002$)	0.365 ($P=0.001$)	0.131 ($P=0.032$)

TABLE 3. Operative Characteristics of the Cohort

Characteristic	Total, n (%)
Operative level	86
L1–2	1 (1.1)
L2–3	7 (8.1)
L3–4	23 (26.7)
L4–5	32 (39.5)
L5–S1	21 (24.4)
Operative time (min)	50.5 ± 16.3
Time to ambulation (h)	6.9 ± 2.5
Postoperative hospital stay (d)	3.6 ± 1.3
Revision surgery	2
Perioperative complication	2

and clinical outcome measures. The correlations were moderately related per VAS leg, while other measures were only weakly associated. The only other study in UBE patients analyzing CSA that the authors are aware of is by Heo et al.[8] Heo and colleagues demonstrated an expansion of 391.5 ± 93.5 mm^2–723.3 ± 100.8 mm^2 in the tubular group and 398.7 ± 97.8 mm^2–719.5 ± 116.4 mm2 in the UBE group. Correlation to patient-reported outcomes was not performed. In Heo and colleagues, the CSA values are much higher than our study and other published paper.[9–11] Similar to our canal expansion, Chung et al[11] in unilateral laminotomy with bilateral decompression through not endoscopic means found an expansion from 60.7 ± 24.7 mm^2 to 161.0 ± 46.9 mm^2.[11] Schonstrom and Hansson found a normal CSA in CT myelogram was 178 ± 50 mm^2. Our study found preoperative CSA changed from 61.0 ± 24.1 mm^2 to 149.8 ± 44.4 mm^2 on the third day after surgery and to 160.1 ± 44.3 mm^2 at final follow-up.

In our study, we also analyzed serial CSA expansion. We believe the serial analysis at least to longer time periods are important because other studies found CSA increased over time. Hermansen et al[29] found at 2 years, the CSA increased slightly more than at 3 months. Oba and colleagues found continued expansion at 1 week to 1 month postoperatively. CSA before surgery was 71.2 ± 4.9 mm^2 then 102.2 ± 5.7 mm^2 and 164.1 ± 6.9 mm^2.[9] Lawton et al[30] report that the bony structure and ligamentum flavum removed after decompression are replaced by mild epidural hematoma, resulting in a less dural expansion in the early phase, which is considered to be the cause of the insufficient dural expansion of the dural sac. Oba et al[9] found difference in speed of expansion differed based on type of decompression that was performed. Interestingly, we found continuous expansion from the early postoperative period to the final follow-up, although not statistically significant in UBE decompression. A possible explanation for the lesser dimension on early postoperative imaging to final follow-up could be mild hematoma in the spinal canal and swelling of adjacent structures due to the fluid medium surgery of UBE. In our study, the difference in CSA between the early and late phases on MRI axial was relatively smaller than in Oba and colleagues. The patients of Oba and colleagues underwent conventional open spinal surgery, whereas all patients in our study underwent UBE decompression. We believe the fluid drainage from the working portal could maintain continuous negative pressure during surgery, thus enhancing dural expansion after removal of the ligamentum flavum. The reason CSA in the late phase is larger than that in the early phase is because the mild hematoma in the early phase is resolved and the dural sac widens as it adapts to the decompressed canal.

A major advantage of this current study is that the patient population was uniform. Only patients who underwent UBE decompression for single-level degenerative lumbar spinal stenosis without instability were included. Because nerve compression typically relates to leg pain, having a correlation between CSA expansion and leg pain appears plausible. Because ODI evaluates functional capacity, the surgical insult and recovery may play a stronger role in ODI. Time may play a more pivotal role than CSA expansion. The same can be said about VAS back. LBP is one of the most common causes of disability. The causes of back pain are more multifactorial than leg pain. Preservation of motion with only a decompression, degeneration of adjacent level issues, and surgical insult all play a role.

There are limitations of this study. This study was retrospective in nature with a small sample of patients. However, since all patients underwent the same technique and only at 1 level, we believe a lot of heterogeneity has been minimized. Second, only 1 axial cut was used to assess the CSA. Some patients had multiple cuts of severe compression, but we could not account for those differences. Last, the early postoperative MRI did not correlate with clinical outcomes.

In conclusion, UBE decompression demonstrated good clinical outcomes similar to previous studies. CSA in UBE continues to increase after the initial 3-day course, likely due to the fluid medium nature of UBE surgery. At best, there is only a moderate correlation between VAS leg and CSA change.

REFERENCE

1. Benoist M. The natural history of lumbar degenerative spinal stenosis. *Joint Bone Spine*. 2002;69:450–457.
2. Liu X, Yuan S, Tian Y. Modified unilateral laminotomy for bilateral decompression for lumbar spinal stenosis: technical note. *Spine (Phila Pa 1976)*. 2013;38:E732–E737.
3. Ikuta K, Arima J, Tanaka T, et al. Short-term results of micro-endoscopic posterior decompression for lumbar spinal stenosis. Technical note. *J Neurosurg Spine*. 2005;2:624–633.
4. Soliman HM. Irrigation endoscopic decompressive laminotomy. A new endoscopic approach for spinal stenosis decompression. *Spine J*. 2015;15:2282–2289.
5. Park SM, Park J, Jang HS, et al. Biportal endoscopic versus microscopic lumbar decompressive laminectomy in patients with spinal stenosis: a randomized controlled trial. *Spine J*. 2020;20:156–165.
6. Min WK, Kim JE, Choi DJ, et al. Clinical and radiological outcomes between biportal endoscopic decompression and microscopic decompression in lumbar spinal stenosis. *J Orthop Sci*. 2019;25:371–378.
7. Kim JE, Choi DJ. Clinical and radiological outcomes of unilateral biportal endoscopic decompression by 30 degrees arthroscopy in lumbar spinal stenosis: minimum 2-year follow-up. *Clin Orthop Surg*. 2018;10:328–336.

8. Heo DH, Quillo-Olvera J, Park CK. Can percutaneous biportal endoscopic surgery achieve enough canal decompression for degenerative lumbar stenosis? Prospective case-control study. *World Neurosurg*. 2018;120:E684–E689.
9. Oba H, Takahashi J, Futatsugi T, et al. Study of dural sac cross-sectional area in early and late lumbar decompression surgery. *Spine J*. 2013;13:1088–1094.
10. Yukawa Y, Lenke LG, Tenhula J, et al. A comprehensive study of patients with surgically treated lumbar spinal stenosis with neurogenic claudication. *J Bone Joint Surg Am*. 2002;84(A):1954–1959.
11. Chung SW, Kang MS, Shin YH, et al. Postoperative expansion of dural sac cross-sectional area after unilateral laminotomy for bilateral decompression: correlation with clinical symptoms. *Korean J Spine*. 2014;11:227–231.
12. Lohman CM, Tallroth K, Kettunen JA, et al. Comparison of radiologic signs and clinical symptoms of spinal stenosis. *Spine*. 2006; 31:1834–1840.
13. Sirvanci M, Bhatia M, Ganiyusufoglu KA. Degenerative lumbar spinal stenosis: correlation with Oswestry Disability Index and MR imaging. *Eur Spine J*. 2008;17:679–685.
14. Hermansen E, Moen G, Barstad J, et al. Laminarthrectomy as a surgical approach for decompressing the spinal canal: assessment of preoperative versus postoperative dural sac cross-sectional areal (DSCSA). *Eur Spine J*. 2013;22:1913–1919.
15. Yamazaki K, Yosida S, Ito T, et al. Postoperative outcome of lumbar spinal canal stenosis after fenestration: correlation with changes in intradural and extradural tube on magnetic resonance imaging. *J Orthop Surg*. 2002;10:136–143.
16. Schonstrom N, Hansson T. Pressure changes following constriction of the cauda equina. An experimental study in situ. *Spine (Phila Pa 1976)*. 1988;13:385–388.
17. Kanno H, Ozawa H, Koizumi Y, et al. Dynamic change of dural sac cross-sectional area in axial loaded magnetic resonance imaging correlates with the severity of clinical symptoms in patients with lumbar spinal canal stenosis. *Spine (Phila Pa 1976)*. 2012;37:207–213.
18. Futatsugi T, Takahashi J, Oba H, et al. Early postoperative magnetic resonance imaging in detecting radicular pain after lumbar decompression surgery: retrospective study of the relationship between dural sac cross-sectional area and postoperative radicular pain. *Clin Spine Surg*. 2017;30:E733–E737.
19. Geisser ME, Haig Aj, Tong HC, et al. Spinal canal size and clinical symptoms among persons diagnosed with lumbar spinal stenoiss. *Clin J Pain*. 2007;23:780–785.
20. Goni VG, Hampannavar A, Gopinathan NR, et al. Comparison of the Oswestry disability index and magnetic resonance imaging findings in lumbar canal stenosis: an observational study. *Asian Spine J*. 2014;8:44–50.
21. Marawar SV, Ordway NR, Madom IA. Comparison of surgeon rating of severity of stenosis using magnetic resonance imaging, dural cross-sectional area, and functional outcome scores. *World Neurosurg*. 2016;96:165–170.
22. Sigmundsson FG, KAng XP, Jonsson B, et al. Correlation between disability and MRI findings in lumbar spinal stenosis; a prospective study of 109 patients operated on by decompression. *Acta Orthop*. 2011;82:204–210.
23. Ogikubo O, Forsberg L, Hansson T. The relationship between the cross-sectional area of the cauda equina and the preoperative symptoms in central lumbar spinal stenosis. *Spine*. 2007;32: 1423–1428.
24. Caprariu R, Oprea MD, Poenaru DV, et al. Correlation between preoperative MRI parameters and Oswestry disability index in patients with lumbar spinal stenosis: a retrospective study. *Medicina*. 2023;59:2000–2010.
25. Boden SD, Davis DO, Dian TS. Abnormal magnetic resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg Am*. 1990;72:403–408.
26. Sokolowski MJ, Garvey TA, Perl J, et al. Postoperative lumbar epidural hematoma: does size really matter? *Spine*. 2008;33:114–119.
27. Herno A, Airaksinen O, Saari T. Computed tomography after laminectomy for lumbar spinal stenosis. Patients' pain patterns, walking capacity, and subjective disability had no correlation with computed tomography findings. *Spine*. 1994;19:1975–1978.
28. Rapala K, Chaberek S, Truszczynska A, et al. Digital computed tomography evaluation of spinal canal and dural sac before and after surgical decompression of lumbar stenosis. *Ortop Traumatol Rehabil*. 2010;12:120–135.
29. Hermansen E, Myklebust TA, Austevoll IM. Dural sac cross-sectional area change from preoperatively and up to 2 years after decompressive surgery for central lumbar spinal stenosis: investigation of operated levels, data from NORDSTEN study. *Eur Spine J*. 2024;33:2234–2241.
30. Lawton MT, Porter RW, Heiserman JE, et al. Surgical management of spinal epidural hematoma: relationship between surgical timing and neurological outcome. *J Neurosurg*. 1995;83:1–7.