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Clinical Study

Preoperative versus postoperative MRI comparisons after the MILD procedure versus open decompression for lumbar spinal stenosis: multicenter cohort study

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Abstract

BACKGROUND CONTEXT: The minimally invasive lumbar decompression (MILD) procedure, introduced in 2005, is a percutaneous fluoroscopy-guided technique designed as an alternative to open decompression for patients with lumbar spinal stenosis (LSS) causing neurogenic claudication. MILD specifically targets hypertrophied ligamentum flavum, allowing for expansion of the dural sac to relieve central stenosis, but it is not intended to treat lateral recess or foraminal stenosis. While published studies have reported symptomatic improvement, no study has evaluated pre and postoperative MRI to assess its effectiveness in achieving anatomical decompression and improving nerve root sedimentation sign (SedSign).

PURPOSE: To compare pre and postoperative MRI findings in patients undergoing MILD versus open decompression, specifically evaluating changes in SedSign and anatomical decompression for LSS.

STUDY DESIGN/SETTING: Multicenter cohort study conducted at 7 centers between 2019 and 2024.

PATIENT SAMPLE: A total of 182 patients with LSS and a positive SedSign on preoperative MRI underwent either MILD (n=162) by interventional pain management (IPM) physicians or open decompression (n=20) by an orthopedic spine surgeon. All patients received postoperative MRI for assessment.

OUTCOME MEASURES: Measures included pre and postoperative MRI assessments evaluating SedSign improvements, decrease in ligamentum flavum thickness, presence of a lamina defect and multifidus muscle scar signal changes.

METHODS: Patients were assigned to treatment based on the specialty of the consulting physician: IPM physicians performed MILD (Group 1), and an orthopedic spine surgeon performed open decompression (Group 2). Blinded radiologists reviewed pre and postoperative MRI scans to assess SedSign and anatomical changes. Interobserver and intraobserver reliability were analyzed using Cohen kappa statistics.

RESULTS: Group 1 demonstrated no SedSign improvement or anatomical changes. Group 2 achieved 100% SedSign reversal, >90% ligamentum flavum removal, 100% lamina defect and multifidus muscle scar signal changes postoperatively. No complications were reported in either group.

CONCLUSION: The MILD procedure did not produce anatomical decompression or SedSign reversal on MRI, whereas open decompression reliably achieved both. Secondary anatomical changes, such as scar tissue and muscle signal alterations, were observed only in the open surgery group. These findings suggest that while MILD is tissue-sparing, it may be insufficient for achieving anatomical goals in patients with central LSS. Future percutaneous strategies should aim to combine the anatomical effectiveness of open decompression with the reduced tissue disruption characteristic of less exposure approaches.

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Keywords: Interventional pain management; Lumbar spinal stenosis (LSS); Magnetic resonance imaging (MRI); Minimally invasive lumbar decompression (MILD) procedure; Minimally invasive spine surgery; Nerve root sedimentation sign (SedSign); Open decompression surgery

Introduction

Lumbar spinal stenosis (LSS) is a degenerative condition characterized by narrowing of the spinal canal, leading to neural compression and symptoms such as neurogenic claudication, back pain, and impaired mobility, which significantly diminish quality of life [1–3]. LSS affects nearly 50% of individuals over 60 years of age, posing a significant public health burden, with annual U.S. medical costs exceeding $2 billion [4–8]. Key contributors include hypertrophic ligamentum flavum, osteophyte formation, and intervertebral disc degeneration, which collectively cause neural compression and chronic pain [9,10].

The nerve root sedimentation sign (SedSign), introduced by Barz et al., is a validated biomarker of spinal stenosis severity [11,12]. A positive SedSign, which reflects the absence of sedimentation of the lumbar nerve roots within the dorsal dural sac, is strongly associated with severe LSS. Wang et al.'s systematic review and meta-analysis demonstrated the SedSign's high diagnostic sensitivity, specificity, and clinical efficacy, establishing its significant clinical utility in the diagnosis of LSS [13]. The initial treatment of LSS often involves conservative treatments such as physical therapy, nonsteroidal anti-inflammatory drugs, epidural steroid injections, and weight management [14,15]. While providing temporary relief in up to 70% of patients, the long-term efficacy remains limited [16].

Surgical intervention is often required for patients with refractory symptoms or severe stenosis [17]. Open

decompression with direct visualization, pioneered in the mid-20th century, marked a significant advancement in the treatment of LSS [18,19]. Laminectomy, often considered the gold standard, involves removal of the lamina to relieve neural compression, while laminotomy provides a more localized decompression by partially removing the lamina [20,21]. These procedures, with success rates ranging from 62% to 70% at 5 years, are highly effective but carry notable risks, including dural tears (5%−10%), wound infections (2%−6%), and iatrogenic spinal instability, particularly in elderly populations or those with comorbid conditions [22]. These limitations have spurred interest in minimally invasive techniques that aim to reduce surgical risk while maintaining efficacy.

The Minimally Invasive Lumbar Decompression (MILD, Vertos Medical, Aliso Viejo, CA, USA) procedure, developed in 2005 by interventional radiologists Dr. David Solsberg and Dr. Donald Schomer, was initially designed for cancer patients with spinal stenosis who were unable to tolerate traditional surgery because of significant comorbidities [23]. MILD employs a percutaneous approach under fluoroscopic guidance to debulk the hypertrophic ligamentum flavum and minimize tissue disruption. Marketed as a safer and cost-effective alternative, MILD has gained acceptance among interventional pain management (IPM) physicians and interventional radiologists (IR) [23−25]. However, it has not been widely adopted by orthopedic spine surgeons or neurosurgeons, who consider open decompression as the gold standard. Despite its growing use and inclusion under the Medicare CPT code 0275T, MILD's efficacy of MILD in achieving measurable anatomical decompression remains uncertain, particularly when evaluated using MRI. To our knowledge, no prior studies have provided evidence comparing MILD with open decompression surgery based on MRI-confirmed changes.

A key distinction between MILD and traditional open decompression lies in their approaches to achieving anatomical changes. Open decompression allows direct visualization and removal of compressive structures, such as the ligamentum flavum, hypertrophic bone, and osteophytes, resulting in measurable changes, such as dural sac expansion. However, its invasiveness carries risks, including nerve root injuries, extensive scar tissue formation, and failed back surgery [26]. In contrast, MILD uses a percutaneous approach with indirect visualization through fluoroscopy and epidurogram contrast flow to guide decompression, leading to less tissue disruption and scarring [27]. This raises the question of whether MILD can result in meaningful anatomical changes.

The objective of this study was to evaluate and compare SedSign improvements, decrease in ligamentum flavum thickness, presence of a lamina defect and multifidus muscle scar signal changes on MRI following MILD versus open decompression surgery. By using MRI as a standardized and reliable tool, we aim to clarify MILD's role as a decompressive technique in the management of LSS and assess its efficacy relative to open surgical decompression.

Materials and methods

Trial design

This study was a multicenter cohort study with a parallel group design comparing the Sedsign and anatomical changes of the MILD procedure to open decompression surgery in patients with lumbar spinal stenosis (LSS) between 2019 and 2024. No changes in the trial design occurred after the commencement. To ensure the consistency and accuracy of imaging assessments, interobserver and intraobserver reliability were rigorously evaluated. Institutional Review Board (IRB) approval was granted for this study through the Western Institutional Review Board (WIRB), now known as the WIRB-Copernicus Group (WCG IRB) (WIRB#20181251), and informed consent was obtained from all individual participants. and was approved by the institutional review boards (IRBs) of all participating centers. Written informed consent was obtained from all the participants.

Participants

A total of 665 patients were enrolled across 7 participating centers, but only 185 met the inclusion criteria. Eligible patients had LSS with neurogenic claudication persisting for more than 3 months despite conservative treatment, confirmed by a preoperative MRI showing ligamentum flavum hypertrophy (>2.5 mm) and a positive SedSign. Exclusion criteria included a history of prior surgery at the same treatment level, lumbar spondylolisthesis > Grade 1, significant symptomatic disc protrusion, excessive facet hypertrophy or osteophyte formation, symptomatic lateral recess stenosis, inability to walk more than 10 feet unassisted, symptomatic epidural lipomatosis, or bleeding disorders [28].

Group 1 (MILD, n=162) included patients treated by IPM physicians who met the indications for the MILD procedure (Current Procedural Terminology [CPT] code 0275T), ensuring regulatory compliance. Group 2 (open decompression, n=20) consisted of patients treated by an orthopedic spine surgeon. Preoperative and postoperative MRI scans were analyzed by blinded radiologists to assess anatomical changes and SedSign status. A patient flow diagram is shown in Fig. 1.

Interventions

Group 1 underwent the MILD procedure, performed posteriorly through a percutaneous incision under local anesthesia and intravenous sedation. Fluoroscopic guidance was used to insert an access portal and establish the working zone. Specialized instruments were used to remove small portions of the lamina and debulk the hypertrophic ligamentum flavum, creating additional space within the spinal canal. Decompression was indirectly confirmed by observing an improved epidurogram contrast flow.

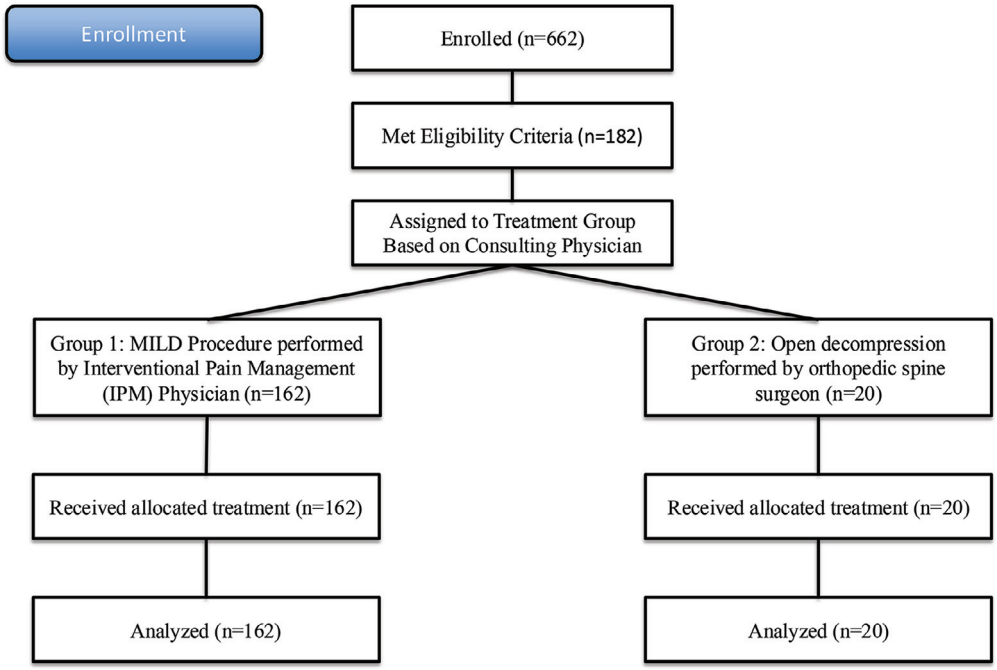


Fig. 1. Patient flow diagram.

Group 2 underwent open midline decompression surgery under general anesthesia. The procedure involved laminotomy or hemilaminectomy, allowing direct visualization and removal of compressive elements such as the hypertrophic ligamentum flavum, bony overgrowth, and other stenotic structures. Adequate decompression of the spinal canal was confirmed intraoperatively by direct visual inspection.

Outcomes

The primary outcome was the improvement in SedSign status (from positive to negative) on MRI assessed preoperatively and postoperatively. Secondary outcomes included comparisons of preoperative and postoperative MRIs scans to evaluate decrease in ligamentum flavum thickness, presence of a lamina defect and multifidus muscle scar signal changes. SedSign status (positive or negative), ligamentum flavum thickness (decreased or unchanged), lamina defect (present or absent), and multifidus muscle scar signal changes (present or absent) were treated as categorical variables.

Sample size

To ensure sufficient power to detect a clinically significant difference in MRI changes, a power analysis was conducted prior to enrollment. Based on prior studies and expected procedural outcomes, we hypothesized that the open decompression group would achieve a SedSign reversal rate >90%, whereas no improvement (0%) was expected in the MILD group. Using Cohen's h effect size (2.50) for this proportion difference, a minimum of 10 patients per group was required to achieve 80% power at α=0.05.

The final study cohort included 182 patients who had both pre and postoperative MRI scans (Group 1: MILD, n=162; Group 2: open decompression, n=20). The distribution of patients across groups was expected based on clinical practice patterns and does not impact the validity of statistical comparisons.

Statistical methods

To evaluate pre and postoperative differences between the MILD and open decompression groups, Chi-square tests were used for categorical variables between groups. Interobserver and intraobserver reliability were evaluated using Cohen's Kappa coefficient. All statistical analyses were performed using Python (version 3.12.0) in Visual Studio Code, employing packages such as pandas, numpy, statsmodels, and scipy. Statistical significance was set at $p<.05$.

Results

Across 7 participating centers, 182 patients were studied, with 162 in Group 1 (MILD) and 20 in Group 2 (open decompression), all having pre and postoperative MRI scans. No complications or adverse events were reported in either group. Baseline patient demographics summarized in Table 1 showed no statistically significant differences between the 2 groups.

Preoperative findings

Preoperative MRI confirmed a positive SedSign in all patients. Baseline ligamentum flavum thickness was

Table 1
Baseline patient demographics

Characteristic	Group 1: MILD (n=162)	Group 2: Open Decompression (n=20)	*p*-value
Age, mean±SD, years	67.4±8.6	66.2±9.1	.58
Sex			.81
Male	84 (51.9%)	11 (55.0%)	
Female	78 (48.1%)	9 (45.0%)	
BMI, mean±SD, kg/m^2	29.3±5.2	28.7±4.9	.60

MILD, minimally invasive lumbar decompression procedure; BMI, Body Mass Index.

comparable between Group 1 and Group 2, with no statistically significant differences (p>.05).

Postoperative findings

Postoperative MRIs for Group 1 showed no measurable anatomical changes (Table 2). There were no changes in the SedSign, ligamentum flavum thickness, presence of a lamina defect and multifidus muscle scar signal changes from preoperative measurements (Figs. 2 and 3), indicating no anatomic resolution of neural compression. In contrast, Group 2 demonstrated significant postoperative improvements with 100% reversal of the SedSign to negative in all patients, over 90% removal of the ligamentum flavum, presence of a lamina defect and multifidus muscle scar signal changes in 100% of patients (Fig. 4). Imaging also showed dural sac expansion and resolution of neural compression. These changes were significant (p<.05).

Comparison of groups

Statistical analyses revealed significant differences between the groups. Pre and postoperative MRI findings in Group 1 (MILD) consistently showed no SedSign reversal or changes in ligamentum flavum thickness, presence of lamina defect or multifidus muscle scar signal, correlating with the absence of anatomical changes. In contrast, 100% of Group 2 (open decompression) patients demonstrated SedSign reversal, decrease in ligamentum flavum thickness, presence of lamina defect and multifidus muscle scar signal changes on postoperative MRI, emphasizing the distinct procedural effects. Categorical differences in postoperative outcomes between the groups were statistically significant (p<.001). Presence of lamina defect and multifidus muscle scar signal changes were observed in 100% of open decompression patients but 0% of MILD patients. Reliability assessments demonstrated excellent agreement among radiologists with a Kappa coefficient of 1.0, looking for the presence or absence of the categorical variables.

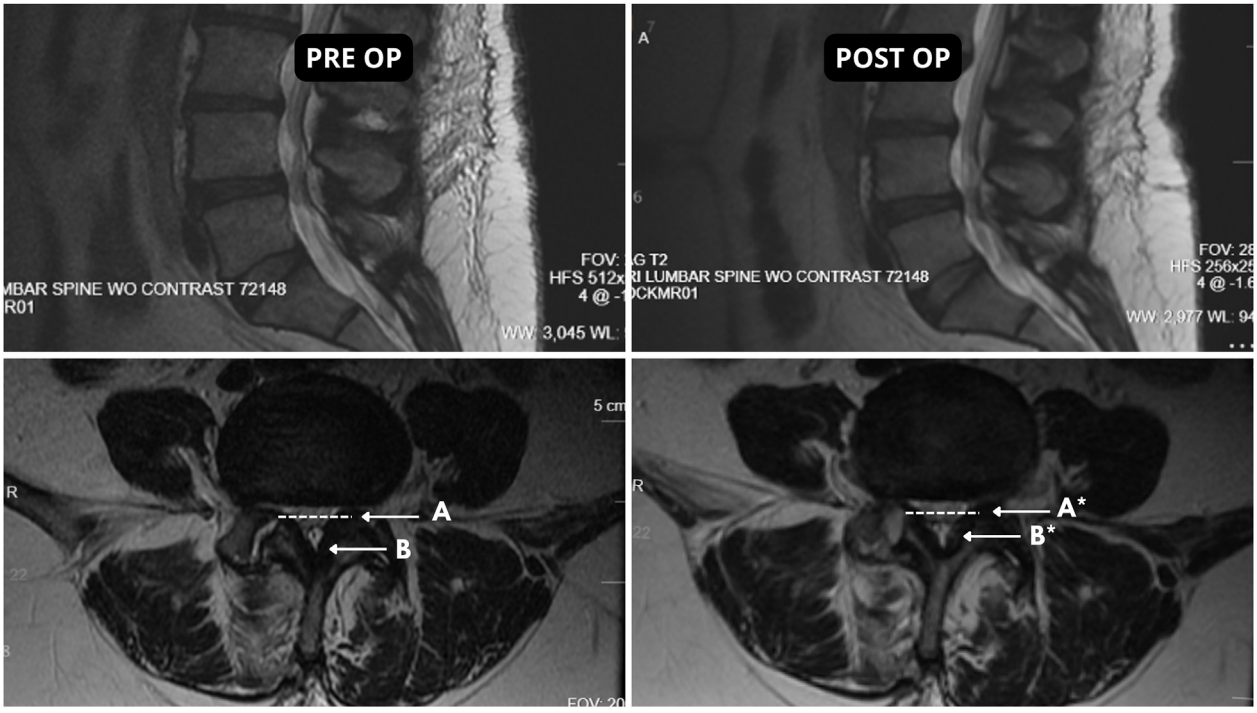


Fig. 2. Sagittal and axial T2-weighted Magnetic Resonance Imaging (MRI) at L4–5 demonstrating anatomical comparison of the ligamentum flavum before and after the MILD procedure. This image was selected to illustrate visual assessment of postprocedural changes, rather than to depict the inclusion criterion of ligamentum flavum hypertrophy. *A*, Positive SedSign, characterized by the absence of sedimentation and clumping of the nerve roots in the dorsal dural sac. *A** showed no changes in the nerve roots position postoperatively, indicating a continued positive SedSign. *B* denotes the preoperative thickened ligamentum flavum, and *B** revealed no decrease in thickness.

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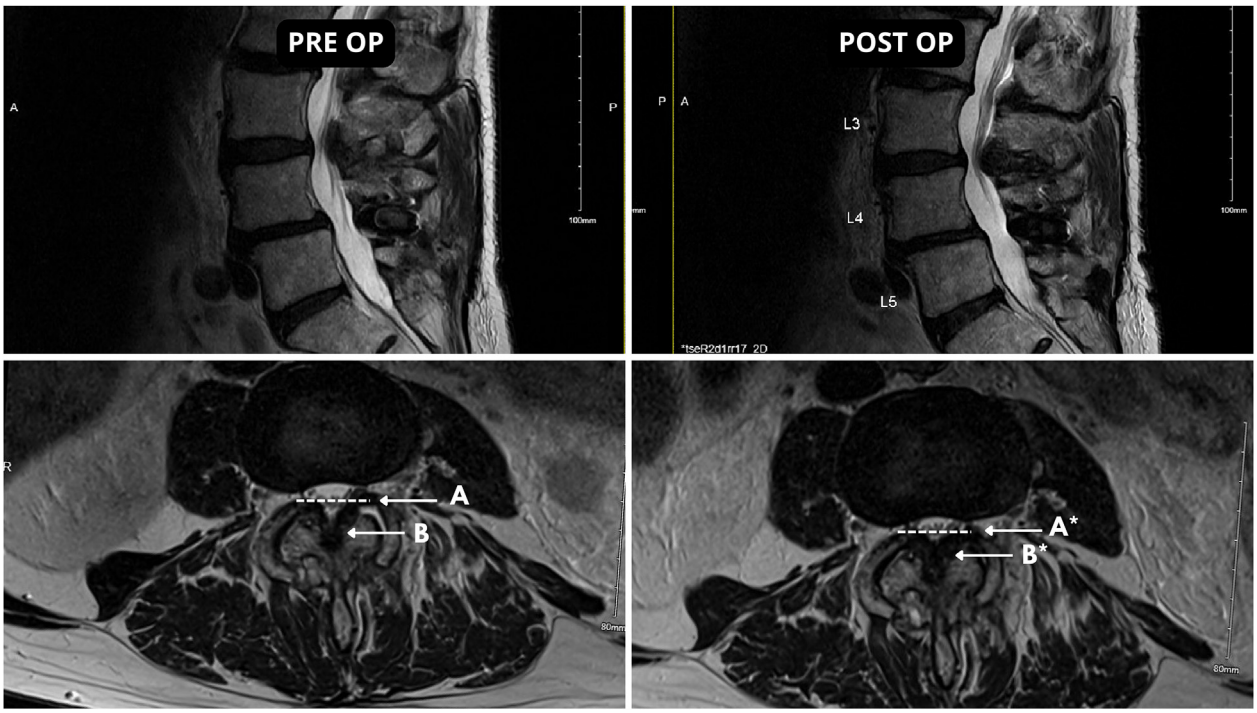


Fig. 3. Magnetic Resonance Imaging (MRI) comparisons between preoperative and postoperative with minimally invasive lumbar decompression (MILD) procedure at L3–4. *A*, Positive SedSign, characterized by the absence of sedimentation and clumping of the nerve roots in the dorsal dural sac. *A** showed no changes in the nerve roots position postoperatively, indicating a continued positive SedSign. *B* denotes the preoperative thickened ligamentum flavum, and *B** revealed no decrease in thickness.

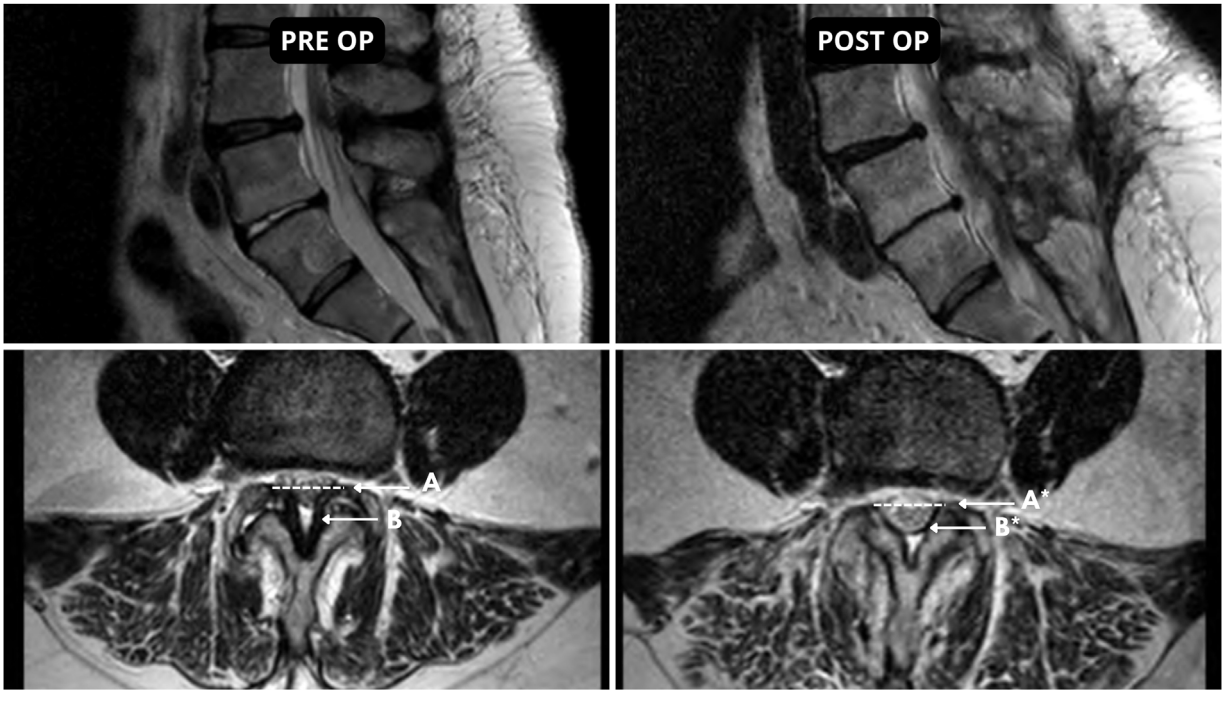


Fig. 4. Magnetic Resonance Imaging (MRI) comparisons between preoperative and postoperative with L4–5 open decompression. *A* demonstrates a positive SedSign with nerve roots clustered anteriorly in the dural sac. *A** shows nerve roots floating freely and posteriorly postoperatively, consistent with a negative SedSign. *B* highlights the preoperative thickened ligamentum flavum, while *B** confirms a postoperative decrease in ligamentum flavum thickness (>90%).

Table 2
Preoperative and postoperative MRI findings: SedSign and anatomical changes in MILD versus Open decompression

MRI Findings	Group 1 and Group 2 (n=182) Preoperative	Group 1: MILD (n=162) Postoperative	Group 2: Open Decompression (n=20) Postoperative
SedSign (nerve root sedimentation sign)	Positive (100%)	Unchanged, Positive (100%)	Changed, Negative (100%)
Ligamentum Flavum Thickness	Positive (100%)	Unchanged (100%)	Decreased >90% (100%)
Lamina Defect	Absent (100%)	Unchanged, Absent (100%)	Changed, Present (100%)
Multifidus Muscle Scar Signal	Absent (100%)	Unchanged, Absent (100%)	Changed, Present (100%)

MILD, minimally invasive lumbar decompression procedure; SedSign, nerve root sedimentation sign.

Discussion

This multicenter cohort study compared the effectiveness of the MILD procedure and open decompression surgery in achieving objective MRI-based improvements in LSS patients with a positive SedSign. Patient-reported outcome measures (PROMs) such as pain or function were not assessed in this study. While open decompression reaffirmed its role as the gold standard for anatomical decompression, the findings highlight the inability of MILD to achieve the measurable anatomical improvements required to alleviate neural compression.

Key findings

Postoperative MRIs demonstrated a stark difference between the 2 groups. Open decompression universally reversed SedSign confirming the increase dural sac volume allowing for the nerve roots to float posteriorly as a positive sign for decreased nerve root compression. There was further anatomical support due to the presence of decreased ligamentum flavum thickness, presence of multifidus muscle scar signal changes and lamina defect. In contrast, MILD resulted in no changes in the SedSign status, and the absence of scar tissue or muscle trauma in MILD cases contrasts with the definitive anatomical changes observed in open decompression. These results suggest that MILD does not address the anatomical pathology underlying LSS. It is noteworthy that this study does not attempt to correlate anatomical changes with PROMs. Pain and functional improvements may be influenced by mechanisms such as reduced neural inflammation, improved local blood flow, or neural plasticity, in addition to decompression or potential for placebo effect. Placebo responses may be influenced by mechanisms such as patient and clinician expectations, therapeutic rituals, and psychosocial context of care [29].

Comparison with prior research

In the literature, studies on the MILD procedure focus on subjective clinical outcomes, such as pain relief and functional improvement, often without corroborating anatomical changes. The MOTION Study demonstrated improved patient-reported outcomes, including the Oswestry Disability Index (ODI), Zurich Claudication Questionnaire (ZCQ) and Numeric Pain Rating Scale (NPRS), when MILD was combined with nonsurgical conventional medical management, but lacked objective imaging data [30]. Two- and 3-year follow-up studies confirmed the durability and safety of the procedure, reporting sustained improvements in walking tolerance and function, with no device- or procedure-related adverse events [31,32]. These findings have reinforced support for MILD as an early interventional option in the treatment of LSS. However, neither the original study nor its follow-up assessments included radiographic confirmation of decompression, limiting insight into the structural basis for the observed clinical improvements.

The MiDAS and MiDAS Encore RCTs showed MILD's superiority over epidural steroid injections (ESIs) for neurogenic claudication, with improvements in ODI and ZCQ scores, but these studies also lacked anatomical confirmation [33,34]. An evidence-based systematic review of 13 studies, including 1 RCT, reported significant improvements in pain (VAS) and function (ODI) following MILD but classified the evidence as low-quality, citing the need for high-quality, independent studies with categorical data and long-term outcomes [35].

While these studies have meaningfully contributed to the adoption of MILD in interventional pain management, most relied heavily on subjective PROMs, which may not accurately reflect structural or physiological changes within the spinal canal. In contrast, our study employed the nerve root sedimentation sign (SedSign), a validated, MRI-based physiologic marker [11,12], to objectively evaluate the extent of decompression before and after intervention. This methodological distinction may account for the discrepancy between the generally favorable clinical literature on MILD and the limited radiographic changes we observed.

In addition, psychosocial and contextual factors may have contributed to the favorable outcomes reported in earlier MILD studies. Many of these trials were conducted by physicians with a strong interest in the procedure's success, and patients may have been particularly motivated to report improvement due to the novelty and minimally invasive nature of the technique. Placebo effects are known to significantly influence subjective outcomes in both conservative and procedural interventions. In a systematic review of randomized controlled trials for chronic nonspecific low back

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pain, placebo effects accounted for 13% to 41% of the overall treatment effect across outcomes such as pain intensity, physical function, and health-related quality of life, with the highest effect observed in quality-of-life measures (41%) [36]. A meta-regression of 72 placebo-controlled interventional trials found that placebo responses accounted for up to 88% of the observed effect in exercise-related outcomes, 61% in clinician-assessed outcomes, and 55% in quality of life scores [37].

Finally, potential confirmation bias and author-related conflicts of interest should be considered when interpreting the existing MILD literature. Many foundational studies have been authored or co-authored by a small group of interventional pain physicians who have actively promoted the MILD procedure, most notably the lead author. While these contributions have helped establish MILD in clinical practice, the recurrence of the same authorship group and known industry relationships highlights the need for independent, objective evaluations.

Our findings demonstrated no significant change in ligamentum flavum thickness, presence of lamina defect, or SedSign status following the MILD procedure. These results suggest that MILD, while potentially effective for symptom relief in select patients, may not achieve the degree of anatomical decompression necessary to alleviate neural compression as visualized on MRI. This aligns with prior calls in the literature for anatomical validation of MILD's clinical effectiveness and highlights the value of radiographically guided outcome assessments.

Limitations

The absence of PROMs in this study limits the ability to correlate SedSign improvements with clinical outcomes. While SedSign serves as an objective physiologic measure, it does not directly reflect pain relief, functional recovery, or quality of life improvements. Future studies should incorporate longitudinal PROMs and functional metrics to better assess symptom resolution, recurrence rates, and the long-term clinical outcomes.

Clinical implications

These findings challenge the role of MILD in the management of symptomatic LSS caused by anatomical pathologies. Clinicians should prioritize procedures such as open decompression, which provides demonstrable anatomical decompression with proven clinical efficacy. While MILD offers reduced tissue disruption and faster recovery, its inability to achieve radiological improvements may limit its role in patients with clear anatomical compression.

Implications for further research

Future innovations should aim to combine the effectiveness of open decompression with reduced tissue trauma associated with procedures, such as MILD. Endoscopic and navigational technologies, including adaptations for intralaminar use, hold promise for achieving anatomical decompression while preserving the minimally invasive benefits.

This study highlights the need for objective anatomical changes when evaluating decompression techniques and highlights the limitations of MILD in its current form. To improve the management of LSS, less invasive techniques must evolve to reliably achieve anatomical decompression, enabling both clinical and radiological success.

Conclusion

This study demonstrated that the MILD procedure does not achieve anatomical decompression or improve spinal stenosis on MRI. Postoperative imaging showed no changes in nerve root sedimentation sign (SedSign) status, ligamentum flavum thickness, presence of lamina defect or multifidus muscle scar signal changes in the MILD group, indicating its inability to anatomically relieve neural compression (Figs. 2 and 3). In contrast, open decompression resulted in 100% SedSign reversal, >90% ligamentum flavum removal, presence of a lamina defect and multifidus muscle scar signal changes in 100% of patients, reaffirming its role as the gold standard for anatomical decompression (Fig. 4). While MILD avoids tissue trauma, it lacks the structural impact necessary to improve spinal stenosis on MRI. Future techniques should aim to match the effectiveness of open surgery while minimizing tissue disruption to achieve meaningful anatomical improvements in spinal stenosis treatment.

Declaration of competing interest

We did not seek or receive any funding from the National of Health (NIH), Wellcome Trust, Howard Hughes Medical Institute (HHMI), or others for this study. KRC is the cofounder and CEO of Kingsley Investment Company (KIC) Ventures and has ownership shares in the company. LF, PP, ST, ES and WMC have shares in KIC Ventures.

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