

Multicenter Cohort Validation of Endoscopic Dual-Zone Decompression for Lumbar Spinal Stenosis: A Comprehensive Assessment of Perioperative Hidden Trauma and Hidden Blood Loss

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Abstract

Background

Traditional assessments of minimally invasive spine surgery (MISS) often rely on superficial metrics like incision length and overt blood loss (OBL), neglecting systemic stress and hidden blood loss (HBL).

Objective

To evaluate the perioperative hidden trauma, muscle injury, and HBL of endoscopic dual-zone decompression (DZD) compared with transforaminal lumbar interbody fusion (TLIF) in elderly patients with degenerative lumbar spinal stenosis (DLSS).

Methods

A retrospective, multicenter cohort study was conducted on 432 elderly patients with DLSS from January 2024 to December 2025. Patients were divided into the DZD group (n = 220) and the TLIF group (n = 212). Propensity matching verified homogeneous baselines across 12 variables ($P > 0.10$). Total blood loss (TBL) and HBL were derived utilizing the Nadler and Gross equations. Systemic inflammatory dynamics (Interleukin-6 [IL-6], Tumor Necrosis Factor- α [TNF- α]) and multifidus muscle damage (Creatine Kinase [CK]) were tracked longitudinally at preoperative (T0), postoperative 24h (T1), 72h (T2), and 7 days (T3).

Results

OBL was significantly lower in the DZD group than in the TLIF group (45.2 ± 12.4 mL vs. 412.8 ± 85.4 mL, $P < 0.001$). However, HBL accounted for $79.31 \pm 6.42\%$ of TBL in the DZD group, which was significantly higher than the $53.33 \pm 5.18\%$ observed in the TLIF group ($P < 0.001$). Repeated-measures ANOVA revealed significant group $\times$ time interaction effects for IL-6, TNF- α , and CK ($P < 0.001$). Serum CK in the DZD group experienced a mild elevation at T1 (124.2 ± 32.5 U/L) and completely reverted to baseline at T3 (86.2 ± 15.4 U/L), whereas the TLIF group exhibited severe, prolonged muscle destruction ($P < 0.001$).

Conclusion

Although endoscopic DZD demonstrates a paradoxically higher proportion of hidden blood loss due to hydrostatic containment intraoperatively, it significantly mitigates systemic inflammatory responses and preserves the structural integrity of the posterior tension band compared to open surgery.

1. Introduction

Degenerative lumbar spinal stenosis (DLSS) stands as the primary source of chronic low back pain, neurogenic intermittent claudication, and radicular lower extremity symptoms among the aging population.¹¹ As life expectancy increases globally, the prevalence of DLSS has escalated, imposing a massive socioeconomic burden on modern healthcare systems.²¹ The pathological hallmark of DLSS involves a progressive, multi-factorial cascade characterized by intervertebral disc degeneration, ligamentum flavum hypertrophy, facet joint arthropathy, and osteophyte formation.²⁰ Together, these structural alterations culminate in the mechanical entrapment and ischemic compression of the cauda equina nerve roots and surrounding vascular structures within the central canal, lateral recesses, and neural foramina.²⁶

For decades, open laminectomy combined with transforaminal lumbar interbody fusion (TLIF) and instrumented pedicle screw fixation has served as the conventional gold standard for patients refractory to conservative therapy.⁵ Although open TLIF provides comprehensive mechanical decompression and definitive immediate segmental stability, it requires extensive subperiosteal detachment and prolonged mechanical retraction of the paraspinal musculature—specifically the multifidus and erector spinae muscles.²⁴ This violent retraction often disrupts the delicate medial branches of the lumbar posterior rami and compromises the segmental pedicle arterial supply, precipitating localized ischemic necrosis, denervation, and persistent fatty infiltration of the posterior tension band.¹³ Clinically, this post-traumatic muscular atrophy manifests as chronic post-laminectomy syndrome, intractable low back pain, and adjacent segment degeneration (ASD), which significantly impairs long-term patient satisfaction.²

With the paradigm shift toward minimally invasive spine surgery (MISS), full-endoscopic dual-zone decompression (DZD) has emerged as a mainstream surgical alternative. DZD utilizes a unilateral interlaminar or biportal endoscopic approach to achieve bilateral sublaminar under-cutting decompression.¹ By working through narrow mechanical corridors under high-definition visualization, it successfully relieves central canal, lateral recess, and foraminal stenosis while meticulously preserving the vital midline osteoligamentous complexes (spinous processes, supraspinous and interspinous ligaments) and contralateral muscular attachments.⁴

However, a conventional cognitive bias persists within contemporary spinal surgery, blindly equating minimal skin incisions and negligible intraoperative overt blood loss (OBL) directly with absolute systemic and localized tissue preservation. Clinical observations frequently counter this naive assumption: elderly patients undergoing full-endoscopic decompression occasionally experience unexplained postoperative fatigue, delayed physical rehabilitation, or an insidious drop in hemoglobin (Hb) levels despite negligible intraoperative blood loss and the absence of postoperative drainage.²⁵ This glaring discrepancy strongly points to the existence of a "black box" of hidden blood loss (HBL)—wherein erythrocytes infiltrate into exposed cancellous bone beds, microvascular beds, and interstitial tissue spaces without leaking out of the wound.

Furthermore, the continuous high hydrostatic pressure fluid irrigation required to maintain visual clarity during endoscopy, combined with the mechanical pressure exerted by the working sleeve on the multifidus muscle, may trigger systemic immune-inflammatory cascades and local skeletal muscle structural destruction.²² To date, the exact temporal kinetics of these systemic and microstructural traumas remain poorly quantified.¹⁸

Therefore, this retrospective multicenter study established a rigorously matched cohort of 432 patients to isolate and quantify HBL using fluid dynamic conservation laws (the Nadler and Gross equations) and to map the longitudinal profiles of systemic inflammatory cytokines (IL-6, TNF- α) and a skeletal muscle-specific biomarker (Creatine Kinase, CK) between endoscopic DZD and traditional open TLIF surgery.

2. Materials and Methods

2.1 Study Design and Patient Selection

This study was designed as a retrospective, multicenter cohort investigation and was formally approved by the Institutional Review Boards of all participating spine centers (including the Department of Spine Surgery, Honghui Hospital, Xi'an Jiaotong University, Department of Surgery of Spine and Spinal Cord, Henan Provincial People's Hospital and Department of spine surgery, Shanghai East Hospital, School of Medicine, Tongji University). Comprehensive medical records and surgical registries from patients aged ≥ 60 years who underwent surgical decompression for symptomatic DLSS between January 2024 and December 2025 were systematically reviewed.

Inclusion Criteria:

1. Age ≥ 60 years with typical symptoms of neurogenic intermittent claudication (maximum walking distance < 300 meters) and radicular lower extremity pain.
2. Advanced neuroimaging (high-resolution CT and 3.0T MRI) confirming severe central canal stenosis (sagittal diameter < 10 mm), lateral recess stenosis (≤ 2 mm), or severe foraminal stenosis (Schizas Grade C or D) correlating perfectly with clinical dermatomal findings.
3. Complete failure of structured conservative treatment (including physical therapy, non-steroidal anti-inflammatory drugs [NSAIDs], and epidural steroid injections) for at least 3 consecutive months.

Exclusion Criteria:

Prior lumbar spine surgery at the index or adjacent levels.

2. Lumbar instability defined as > 3 mm translational motion or $> 10^\circ$ angular change on dynamic flexion-extension radiographs, degenerative spondylolisthesis exceeding Meyerding Grade II, or severe degenerative lumbar scoliosis (Cobb angle $> 20^\circ$) requiring deformity correction.

3. Pathological spine fractures, primary or metastatic spinal tumors, tuberculosis, or pyogenic spondylodiscitis.
4. Primary hematopoietic diseases, coagulation disorders, or failure to stop antiplatelet/anticoagulant therapy for 7–10 days perioperatively.
5. Severe organ insufficiency (NYHA Grade III-IV heart failure, Child-Pugh B-C cirrhosis, or end-stage renal disease).

A total of 432 patients met the criteria and were categorized based on the surgical approach: the DZD group (n = 220, full-endoscopic dual-zone decompression without instrumentation or drainage) and the TLIF group (n = 212, conventional total laminectomy, transforaminal lumbar interbody fusion, pedicle screw fixation, and deep negative pressure drainage).

2.2 Surgical Techniques

DZD Group: Under general anesthesia, the patient was positioned prone on a radiolucent Wilson frame to optimize lumbar flexion. Under fluoroscopic guidance, a 1.2-cm longitudinal incision was made over the medial border of the target inferior articular process. Serial dilators were inserted bluntly along the natural direction of the multifidus fibers to minimize mechanical tearing. A 10.5-mm working sleeve and endoscope were introduced. Constant saline irrigation was delivered via an automated pump, maintaining hydrostatic pressure at 40–60 mmHg and a flow rate of 200–300 mL/min to clear bone debris and maintain clear visualization. The first zone (central canal and ipsilateral recess) was decompressed using high-speed diamond burrs and endoscopic Kerrison rongeurs, followed by contralateral under-cutting via an ipsilateral approach. The second zone (foramen and extraforaminal exit zone) was cleared. Radiofrequency bipolar electrocoagulation was applied for precise hemostasis, and the incision was closed primarily without a drain.

TLIF Group: A standard 10–15 cm midline incision was performed. The bilateral multifidus and erector spinae muscles were subperiosteally detached from the spinous processes and laminae using Cobb elevators and retracted laterally under constant tension. Total laminectomy, bilateral facetectomy, pedicle screw instrumentation, and cage insertion were executed. A 4.5-mm negative pressure drain was placed before layered closure to prevent hematoma formation.

2.3 Mathematical Derivation of Hidden Blood Loss (HBL)

To isolate HBL from confounding fluid factors, patient-specific estimated patient blood volume (PBV) was first calculated using Nadler's equations: $PBV = K_1 \times H^3 + K_2 \times W + K_3$

Where H is height (m), W is weight (kg), and the sex-specific coefficients are defined as:

For Males: $k_1 = 0.3669$, $k_2 = 0.03219$, $k_3 = 0.6041$

For Females: $k_1 = 0.3561$, $k_2 = 0.03308$, $k_3 = 0.1833$

According to the Gross equation, assuming vascular volume equilibration by postoperative day 3, the total blood loss

$$TBL = (Hct_{pre} - Hct_{post_3d}) \div Hct_{mean} \times PBV$$

Where $Hct_{mean} = (Hct_{pre} + Hct_{post_3d}) / 2$. HBL was subsequently isolated by subtracting overt blood loss (OBL, comprising intraoperative suction volume and postoperative drainage) from TBL: $HBL = TBL - OBL = TBL - (V_{intraoperative} + V_{drainage})$

2.4 Biomarker Tracking and Statistical Analysis

Venous blood samples were collected at four distinct time points: preoperative baseline (T_0), postoperative 24h (T_1), 72h (T_2), and 7 days (T_3). Serum Interleukin-6 (IL-6) and Tumor Necrosis Factor- α (TNF- α) were quantified via enzyme-linked immunosorbent assay (ELISA). Serum Creatine Kinase (CK) activity was measured using standard rate assays.

Statistical analyses were performed using R (v4.3.1). Longitudes of biomarkers were analyzed via repeated-measures ANOVA (RM-ANOVA). Mauchly's test was used to assess sphericity; if violated ($P < 0.05$), Greenhouse-Geisser corrections (ϵ_{GG}) were enforced. Post-hoc multiple comparisons were adjusted using the Bonferroni method. Alpha was set at 0.05.

3. Results

3.1 Demographic and Baseline Characteristics

Propensity matching and baseline data cleaning verified homogeneous baseline characteristics across both groups ($P > 0.10$, Table 1), guaranteeing that subsequent perioperative stress variations were independent of host confounding factors.

Table 1
Preoperative demographic and baseline clinical characteristics of the cohort.

Characteristics	DZD Group (n = 220)	TLIF Group (n = 212)	Statistics (t / χ^2)	P-value
Age (years, $\bar{x} \pm s$)	69.4 $\pm$ 5.8	70.1 $\pm$ 6.2	t = -1.214	0.225
Sex (Male/Female, n)	98 / 122	91 / 121	χ^2 = 0.113	0.737
BMI (kg/m ²)	23.8 $\pm$ 2.9	24.1 $\pm$ 3.1	t = -1.038	0.300
Disease Duration (months)	24.5 $\pm$ 8.2	25.8 $\pm$ 9.1	t = -1.558	0.120
Lumbar BMD T-score	-2.42 $\pm$ 0.45	-2.48 $\pm$ 0.51	t = 1.296	0.196
Hypertension (n, %)	88 (40.0%)	82 (38.7%)	χ^2 = 0.076	0.783
Type II Diabetes (n, %)	42 (19.1%)	39 (18.4%)	χ^2 = 0.038	0.846
Preoperative Hb (g/L)	134.2 $\pm$ 12.5	133.8 $\pm$ 11.9	t = 0.340	0.734
Preoperative Hct (%)	41.2 $\pm$ 3.5	40.9 $\pm$ 3.2	t = 0.929	0.353

3.2 Distribution of Blood Loss Matrix

The mathematical breakdown of the perioperative blood loss profiles revealed a highly significant divergence between the two groups ($P < 0.001$, Table 2).

Table 2
Quantification of perioperative blood loss distribution based on fluid dynamics.

Variables (mL, $\bar{x} \pm s$)	DZD Group (n = 220)	TLIF Group (n = 212)	t-value	P-value
PBV (Estimated Total Blood Volume)	4125.8 $\pm$ 342.5	4158.4 $\pm$ 361.2	-0.962	0.337
TBL (Total Blood Loss)	218.6 $\pm$ 5.8	884.5 $\pm$ 142.6	-65.120	< 0.001
OBL (Overt Blood Loss)	45.2 $\pm$ 12.4	412.8 $\pm$ 85.4	-62.410	< 0.001
HBL (Hidden Blood Loss)	173.4 $\pm$ 38.5	471.7 $\pm$ 92.3	-43.450	< 0.001
HBL Ratio (HBL/TBL, %)	79.31 $\pm$ 6.42%	53.33 $\pm$ 5.18%	46.250	< 0.001

Notably, while the TBL of the DZD group was substantially lower than that of the open group, the HBL ratio (HBL/TBL) reached 79.31 $\pm$ 6.42% in the endoscopic group—nearly 1.5 times higher than that in the TLIF group (53.33%).

3.3 Systemic Inflammatory Cascades (IL-6 and TNF- α)

For serum IL-6, Mauchly's test violated the sphericity assumption ($W = 0.412$, $P < 0.001$), necessitating a Greenhouse-Geisser adjustment ($\epsilon_{GG} = 0.712$). The corrected RM-ANOVA revealed:

Time Effect: $F(2.14, 918.5) = 1412.42$, $P < 0.001$

Group Effect: $F(1, 430) = 812.55$, $P < 0.001$

Interaction Effect (Group $\times$ Time): $F(2.14, 918.5) = 345.88$, $P < 0.001$

Post-hoc Bonferroni tests showed that at the acute postoperative peak (T_1), serum IL-6 surged over 13-fold in the open TLIF group (58.4 ± 12.4 pg/mL) but was heavily suppressed in the DZD group (18.5 ± 4.2 pg/mL). By T_3 , the DZD group had completely normalized (5.1 ± 1.1 pg/mL), whereas the TLIF group sustained elevated levels (24.8 ± 5.3 pg/mL). TNF- α exhibited identical trends, with the DZD group remaining below the rigid safety threshold of 12.0 pg/mL across all time points.

3.4 Posterior Tension Band Damage (Creatine Kinase Dynamics)

Serum CK activity acted as a precise surrogate for structural damage to the multifidus muscle. The longitudinal trajectories across the four target time points are compiled in Table 3.

Table 3
Longitudinal changes in serum Creatine Kinase (CK) activity (U/L).

Group	Pre-op (T_0)	Post-op 24h (T_1)	Post-op 72h (T_2)	Post-op 7d (T_3)
DZD Group (n = 220)	84.5 ± 18.2	124.2 ± 32.5	98.4 ± 22.4	86.2 ± 15.4
TLIF Group (n = 212)	86.1 ± 19.4	584.6 ± 112.4	412.5 ± 88.5	188.4 ± 34.2
Group t-value	-0.874	-53.420	-48.550	-39.140
P-value	0.624	< 0.001	< 0.001	< 0.001

The TLIF group experienced a massive spike in CK activity at T_1 (nearly 7-fold baseline), indicating widespread muscle fiber necrosis and cell membrane breakdown. In contrast, the DZD group demonstrated only a minor, transient fluctuation at T_1 (124.2 ± 32.5 U/L) and achieved 100% baseline restoration by T_3 (86.2 ± 15.4 U/L, $P > 0.05$).

4. Discussion

4.1 The Hydrostatic Tamponade and Fluid Dynamics of Hidden Blood Loss

The core mathematical discovery of this multicenter matched cohort investigation lies in the quantitative unmasking of the "high hidden blood loss ratio paradox" within full-endoscopic spine surgery.

Conventional surgical dogmas often default to the assumption that a minuscule skin incision accompanied by negligible intraoperative drainage or suction volume directly implies minimal circulatory stress.¹⁵ However, our rigid mathematical isolation utilizing the Nadler and Gross fluid-dynamic equations challenges this oversimplification.³ Although the absolute TBL in the DZD group was controlled at a safe, low baseline compared to open TLIF (218.6 ± 45.8 mL vs. 884.5 ± 142.6 mL), the internal proportion of HBL within the endoscopic cohort reached an astonishing $79.31 \pm 6.42\%$, vastly exceeding the $53.33 \pm 5.18\%$ calculated for open surgery ($P < 0.001$).

To unravel this clinical paradox, one must analyze the localized fluid dynamics and microvascular anatomy unique to full-endoscopic procedures. During DZD, constant saline irrigation driven by automated perfusion pumps is mandatory to clear bone debris and maintain excellent visualization. This continuous fluid influx establishes a persistent intraoperative hydrostatic pressure grid, typically calibrated between 40 and 60 mmHg.⁷

From a microcirculatory standpoint, this localized pressure deliberately surpasses the baseline physiological pressure of the epidural venous plexus and the microvascular capillary beds within the lumbar marrow sinusoids.¹⁷ Consequently, minor venous oozing and bone surface bleeding are immediately tamponaded and hidden from the surgeon's sight. This hydrostatic containment explains the deceptively low OBL (45.2 ± 12.4 mL) observed intraoperatively.

However, the biological boundary conditions shift immediately upon the termination of the procedure. The moment the endoscope and working sleeves are extracted and the 1.2-cm skin portal is closed primarily without deep negative-pressure drainage, the localized intraoperative hydrostatic pressure drops instantly to zero. At this juncture, the extensive cancellous bone beds exposed during bilateral under-cutting laminectomy and facet decompression remain vulnerable.²⁸

These cancellous bone channels house disrupted marrow sinusoids that are devoid of traditional vascular smooth muscle walls and are thus structurally incapable of spontaneous vasoconstriction or mechanical closure.¹⁰ As the patient emerges from general anesthesia and systemic arterial blood pressure returns to preoperative baseline levels, persistent, insidious microvascular oozing inevitably takes place within the closed interstitial tissue boundaries and dead spaces.¹²

Because the DZD approach relies on primary skin closure without deep fascial or subcutaneous drainage, the extravasated blood volume (averaging 173.4 mL) becomes permanently sequestered within the enclosed intermuscular fascial planes and epidural spaces. Trapped within this confined anatomical chamber, these displaced erythrocytes are exposed to osmotic shifts driven by residual, interstitial irrigation saline.²³

This triggers accelerated erythrocyte membrane fragility, osmotic lysis, and subsequent hemoglobin breakdown.²⁹ The localized hemolysis and subsequent systemic macrophage phagocytic clearance manifest clinical lags, appearing as a delayed but highly statistically significant drop in circulating

hemoglobin (Hb) and hematocrit (Hct) levels by the third postoperative day. This explains why elderly patients occasionally experience unexpected fatigue and delayed mobilization despite minimal visible blood loss.

4.2 Paraspinal Muscle Destruction and Posterior Tension Band Preservation

Beyond systemic volume depletion, the structural preservation of the posterior lumbar tension band constitutes a critical determinant of long-term functional recovery, sagittal alignment balance, and the prevention of adjacent segment degeneration (ASD).⁶ The multifidus muscle is recognized as the primary active stabilizer of the lumbar spine, responsible for providing localized mechanical stiffness and counteracting anterior bending moments.¹⁶

Our longitudinal tracking of serum Creatine Kinase (CK) activity provides a precise, biochemical surrogate of paraspinal muscle structural trauma. Serum CK is an enzyme confined strictly within the sarcolemma of skeletal muscle fibers; its elevation in peripheral circulation serves as a highly specific indicator of muscle fiber mechanical disruption, ischemic necrosis, or cellular membrane lysis.³¹

In the conventional open TLIF group, serum CK levels experienced an enormous, violent spike at 24 hours postoperatively (584.6 $\pm$ 112.4 U/L, approaching a 7-fold increase from baseline) and remained severely elevated through day 3. This biochemical profile reflects the macro-surgical trauma induced by standard posterior exposures. Open TLIF necessitates extensive subperiosteal elevation of the erector spinae and multifidus muscles off the spinous processes and laminae.⁹

Furthermore, the prolonged use of self-retaining steel retractors exerts continuous mechanical compression on the muscle bellies that frequently exceeds the critical capillary perfusion pressure of skeletal muscle (30 mmHg).³² This induces severe localized tissue ischemia, microvascular thrombosis, and widespread coagulative necrosis of the muscle fibers.³⁰ Simultaneously, the aggressive lateral retraction routinely damages the medial branches of the lumbar posterior rami, leading to irreversible neurogenic atrophy and subsequent fatty infiltration of the paraspinal musculature.¹⁴

In stark contrast, the full-endoscopic DZD group demonstrated an exceptionally mild, transient elevation of serum CK at 24 hours (124.2 ± 32.5 U/L) and achieved complete restoration to baseline values within 7 days postoperatively (86.2 ± 15.4 U/L, $P > 0.05$). This superior biochemical profile provides definitive evidence of the tissue-sparing nature of the endoscopic corridor. The DZD technique completely avoids broad subperiosteal muscle stripping.¹⁹

Instead, it utilizes sequential, blunt dilators that separate the paraspinal muscle fibers along their natural anatomical fascicular orientations, avoiding mechanical tearing. The working sleeve protects the surrounding muscle tissue from direct mechanical friction caused by high-speed burrs and instruments. Crucially, by operating strictly within the interlaminar window or via precise biportal coordinates, DZD

preserves the integrity of the medial branches of the lumbar posterior rami and maintains the continuity of the segmental pedicle artery branches.⁸

By preserving the paraspinal muscle architecture and maintaining the attachment points of the contralateral tension band, DZD secures the long-term dynamic stability of the lumbar spine. This shields the patient from the mechanical instability and chronic low back pain that frequently plague patients following open laminectomy.²⁷

4.3 Systemic Immunological Cascade and Inflammatory Stress Signatures

Surgical trauma inevitably provokes a systemic complex immune-inflammatory cascade. While localized inflammation is fundamental to tissue healing, an excessive, uncontrolled systemic inflammatory response syndrome (SIRS) can cause secondary tissue damage, exacerbate postoperative pain, delay functional recovery, and increase the risk of cardiopulmonary complications in elderly patients with pre-existing comorbidities. In this investigation, the temporal dynamics of two cardinal pro-inflammatory cytokines, Interleukin-6 (IL-6) and Tumor Necrosis Factor-alpha (TNF- α), were tracked to map the systemic stress profiles of both surgical cohorts.

Our results demonstrate a severe systemic inflammatory storm in the open TLIF cohort. Serum IL-6 surged more than 13-fold at 24 hours postoperatively (58.4 ± 12.4 pg/mL) and failed to return to baseline even at 7 days. This prolonged cytokine release is triggered by extensive surgical wounds, widespread soft tissue disruption, bone transection during total laminectomy, and the implantation of titanium pedicle screws and polyetheretherketone (PEEK) interbody fusion cages.

Tissue injury activates local resident macrophages and mast cells, unleashing a wave of alarmins and damage-associated molecular patterns (DAMPs) into the circulation. This stimulates the hepatic synthesis of acute-phase reactants and drives the systemic inflammatory cascade. High circulating levels of IL-6 and TNF- α act directly on the central nervous system to induce hyperalgesia, fever, and postoperative anorexia, while simultaneously impairing endothelial boundaries and increasing capillary permeability.

Conversely, the full-endoscopic DZD group demonstrated an attenuated systemic immune response. Serum IL-6 levels at 24 hours were suppressed to less than one-third of the levels observed in the open group (18.5 ± 4.2 pg/mL) and achieved absolute normalization by day 7. Similarly, serum TNF- α levels in the DZD cohort remained safely below the clinical inflammatory threshold across all post-operative time points.

This confirms that the localized micro-environment of endoscopic decompression restricts the liberation of pro-inflammatory cytokines. The minimized soft tissue disruption and meticulous fluid-mediated clearance of local inflammatory debris limit the systemic expression of these signaling molecules.

Consequently, elderly patients are insulated from severe systemic metabolic stress, protecting organ function and facilitating accelerated clinical recovery.

4.4 Clinical Realities, ERAS Implementation, and Methodological Limitations

The insights gained from this large-scale matched validation hold significant value for clinical practice, particularly within the framework of Enhanced Recovery After Surgery (ERAS) for geriatric spine care. By breaking through the traditional surface-level assessment of "minimally invasive" interventions, this study provides objective data to help clinicians optimize perioperative pathways.

First, surgeons must acknowledge that full-endoscopic spine surgery is not entirely free from hematological stress. The "high HBL ratio paradox" identified in this study demands proactive clinical oversight. For elderly patients with marginal cardiovascular reserve, pre-existing anemia, or severe osteoporosis, relying solely on the absence of visible blood in the suction canister can be dangerously misleading.

We recommend mandatory monitoring of hemoglobin and hematocrit levels through postoperative day 3, irrespective of minimal intraoperative blood loss. Furthermore, for patients identified as high risk for HBL, the intraoperative or early postoperative administration of intravenous tranexamic acid (TXA) should be considered. TXA can safely inhibit fibrinolysis within exposed cancellous bone beds and microvascular structures without elevating the risk of deep vein thrombosis (DVT).

Second, the rapid normalization of serum CK and systemic cytokines in the DZD group provides a biological rationale for aggressive early mobilization. Patients undergoing DZD can safely initiate out-of-bed ambulation and structured physical rehabilitation within 24 hours postoperatively, as the structural integrity of the posterior muscular tension band remains intact. This early mobility reduces the risk of prolonged bed rest complications, such as hypostatic pneumonia, muscular atrophy, and thromboembolic events.

Nonetheless, this study is not without limitations. First, despite utilizing a robust, multicenter cohort of 432 patients and applying propensity score matching to achieve homogeneous baselines across 12 confounding variables, its retrospective design introduces inherent selection biases. A prospective, randomized controlled trial (RCT) remains necessary to confirm these findings.

Second, the quantification of HBL utilizing the Nadler and Gross equations assumes a stable vascular volume and steady-state fluid distribution by postoperative day 3; variations in postoperative intravenous fluid resuscitation or third-space fluid shifts may introduce minor analytical noise.

Finally, this investigation focused primarily on perioperative biochemical markers and fluid dynamics; long-term clinical correlation specifically the long-term incidence of adjacent segment degeneration (ASD), paraspinal muscle fatty infiltration via advanced MRI spectroscopy, and patient-reported outcome measures (PROMs) over a 5-year follow-up period—will be reported in a forthcoming companion study.

5. Conclusion

In conclusion, this multicenter cohort study successfully isolated and quantified the multi-dimensional perioperative hidden trauma profile of full-endoscopic dual-zone decompression (DZD) compared to traditional open TLIF surgery in elderly patients with severe DLSS. Our fluid dynamic analysis confirms the existence of the "high hidden blood loss ratio paradox" in endoscopic surgery: although DZD reduces total blood loss, its hidden blood loss accounts for a high proportion (79.31%) of total blood volume shifts due to intraoperative hydrostatic containment.

However, at the micro-biochemical and structural level, DZD achieves superior tissue preservation, effectively preventing the severe systemic inflammatory storm and extensive paraspinal muscle destruction characteristic of open TLIF. These objective findings substantiate the true tissue-sparing nature of full-endoscopic dual-zone decompression, establishing it as a highly compatible intervention for the geriatric population within modern ERAS frameworks.

Declarations

Ethics Approval: This study was conducted in accordance with the 1964 Declaration of Helsinki and its later amendments. Ethical approval was granted by the Ethics Committee of Xi'an Honghui Hospital (Approval No.: [2025021]). Due to the retrospective nature of this study, the requirement for written informed consent was waived by the Ethics Committee of Xi'an Honghui Hospital.

Consent to Participate: Not applicable.

Consent for publication: Not applicable.

Availability of data and materials: The data that support the findings of this study are available from Xi'an Honghui Hospital, but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are, however, available from the authors upon reasonable request and with permission of Xi'an Honghui Hospital.

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