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Original Article

Comparison of low-concentration ropivacaine epidural anesthesia versus local infiltration anesthesia in percutaneous endoscopic surgery for lumbar disc herniation

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Abstract: Objective: To compare the efficacy and safety of low-concentration ropivacaine epidural anesthesia with that of local infiltration anesthesia in patients undergoing percutaneous endoscopic surgery for lumbar disc herniation (LDH). Methods: From May 2023 to June 2025, 110 patients who underwent single-level percutaneous endoscopic lumbar discectomy were assigned to either a control group receiving local infiltration anesthesia with low-concentration ropivacaine or an observation group receiving epidural anesthesia with low-concentration ropivacaine. Surgical outcomes, hemodynamic parameters, Ramsay sedation scores, visual analog scale (VAS) pain scores, inflammatory markers, and pain mediators were compared between the two groups. Repeated measures analysis of variance (ANOVA) with Bonferroni post hoc correction was used for analysis of longitudinal data. Results: Compared with the control group, the observation group had a longer postoperative ambulation time but required a lower total dose of ropivacaine and a lower rate of rescue sufentanil analgesia ($P < 0.05$). Intraoperatively, the observation group showed lower mean arterial pressure (MAP) and heart rate (HR) at T1-T3, whereas Ramsay sedation scores at T1-T4 were significantly higher ($P < 0.05$). Postoperatively, VAS pain scores at T5-T9 were distinctly lower in the observation group. In addition, levels of inflammatory markers and pain mediators, including 5-hydroxytryptamine (5-HT) and prostaglandin E_2 (PGE_2), were reduced at 24 h and 72 h postoperatively ($P < 0.05$). Significant effects of time, group, and time × group interaction were observed for all hemodynamic, sedation, pain, and inflammatory variables ($P < 0.05$). Adverse event rates did not differ between groups ($P > 0.05$). Conclusion: Low-concentration ropivacaine epidural anesthesia provides better intraoperative hemodynamic stability, deeper sedation, more effective postoperative analgesia, and stronger suppression of inflammatory responses and pain mediator release compared with local infiltration anesthesia in patients undergoing percutaneous endoscopic surgery for LDH.

Keywords: Low concentration, ropivacaine, local infiltration anesthesia, epidural anesthesia, percutaneous endoscopy, lumbar disc herniation, adverse events

Introduction

Lumbar disc herniation (LDH) is a common degenerative spinal disorder associated with disc degeneration changes, trauma, and mechanical stress. Typical symptoms include low back pain, lower limb numbness, and radiating pain, with severe cases presenting with cauda equina syndrome. For patients who fail conservative management, percutaneous endoscopic lumbar discectomy adequately relieves pain and restores lumbar function [1]. The best anesthetic strategy for this procedure, however, remains controversial. Various anesthetic techniques, including local anesthesia combined with sedation, subarachnoid block, and general anesthesia, have been applied in clinical practice [2].

Local infiltration anesthesia offers advantages such as ease of administration, low cost, and ability to maintain intraoperative communica-

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tion with patients. Nevertheless, inadequate analgesia and incomplete neural blockade remain important limitations [3]. In contrast, epidural anesthesia provides a more controllable sensory blockade. Previous studies have indicated that low-concentration ropivacaine epidural anesthesia can selectively block sensory nerves while largely preserving motor function, achieving a "sensory-motor dissociation" state, along with low cardiotoxicity and favorable controllability [4]. Therefore, this study compared the clinical effects and safety of low-concentration ropivacaine epidural anesthesia versus local infiltration anesthesia in patients undergoing percutaneous endoscopic surgery for LDH, aiming to provide evidence to guide anesthetic selection in clinical practice.

Materials and methods

General information

Clinical data from 412 patients diagnosed with LDH who underwent single-level percutaneous endoscopic lumbar discectomy at our hospital between May 2023 and June 2025 were retrospectively collected. Patients were divided based on the intraoperative anesthetic method into a control group (n=239), which received local infiltration anesthesia with low-concentration ropivacaine, and an observation group (n=173), which received epidural anesthesia with low-concentration ropivacaine. To minimize baseline confounding, a logit-based propensity score matching (PSM) using a 1:1 nearest-neighbor matching algorithm was implemented. The caliper width was set at 0.2 × standard deviation (SD) of the logit-transformed propensity score. PSM covariates included age, sex, body mass index (BMI), disease duration, affected spinal segment, and comorbidities. After matching, 55 patients were included in each group, totaling 110. Post-matching assessment demonstrated that the standardized mean differences (SMD) for all covariates were < 0.1, indicating a well-balanced baseline. Sample size adequacy was evaluated based on the primary outcome measures. Using an independent samples t-test with α=0.05 (two-sided) and 55 cases per group, the observed effect size (Cohen's d≈0.6-0.8) corresponded to a statistical power (1-β) > 90%. Consequently, the sample size of this study was considered adequate for detecting clinically meaningful differences.

Inclusion and exclusion criteria

Inclusion criteria: ① Diagnosis consistent with the *Chinese Pain Expert Consensus on the Diagnosis and Treatment of Lumbar Disc Herniation* [5], with magnetic resonance imaging (MRI) or computed tomography (CT) findings and clinical manifestations confirming single-level LDH; ② Age between 18 and 75 years old; ③ American Society of Anesthesiologists (ASA) classification I-III; ④ Underwent single-level percutaneous endoscopic lumbar discectomy with either low-concentration ropivacaine local infiltration or low-concentration ropivacaine epidural anesthesia; ⑤ Complete medical records, including anesthesia records, operative reports, postoperative recovery data, and major adverse event documentation.

Exclusion criteria: ① Severe dysfunction of major organs, including the cardiovascular, cerebrovascular, pulmonary, hepatic, or renal systems; ② A history of opioid or analgesic abuse; ③ Coagulopathy, infection of the skin or soft tissue in the lumbosacral region, or neuropsychiatric disorders; ④ Previous lumbar surgery at the target segment; ⑤ Severe lumbar spinal stenosis, spinal deformity, spondylolisthesis, or spinal instability requiring fusion surgery; ⑥ Pre-existing cognitive impairment, psychiatric disorders, or language barriers affecting communication.

Anesthesia methods

All patients underwent percutaneous endoscopic surgery. Preoperative fasting for 8 hours and fluid restriction for 2 hours were required. Upon entering the operating room, routine monitoring was initiated, and peripheral intravenous access was established. Dexmedetomidine hydrochloride (Hengrui Medicine Co., Ltd., Jiangsu, China; specification 1 mL: 100 μg; approval number H20130093) was administered intravenously at a loading dose of 0.5-1.0 μg/kg over approximately 15 minutes, followed by continuous infusion at 0.2-0.5 μg/(kg.h). Lidocaine (Hunan Kelun Pharmaceutical Co., Ltd., China; specification 5 mL:0.1 g, approval number: H20057816) and ropivacaine (ASZAAB, specification 10 mL:75 mg, Import Drug Registration Number 20140764) were prepared at the required concentrations before surgery.

In the control group, patients received local infiltration anesthesia with low-concentration ropivacaine. After localization under C-arm fluoroscopy, 3-5 mL of 1% lidocaine was injected locally by the surgeon. Subsequently, 0.5% ropivacaine was infiltrated layer by layer into the subcutaneous tissue, fascia, and muscle along the endoscopic approach. The injection volume was adjusted based on the patient's condition and did not exceed 40 mL.

In the observation group, patients received epidural anesthesia with low-concentration ropivacaine. Patients were placed in the lateral decubitus position with lumbar flexion. The needle bevel was directed toward the sacrococcygeal region, and a test dose of 4 mL of 1% lidocaine was slowly injected. After confirming the absence of adverse reactions, 10-20 mL of 0.2% ropivacaine was administered. The needle was subsequently rotated with the bevel facing upward, and an epidural catheter was retained in place. The sensory block level was maintained below the T6 dermatome. Patients were repositioned prone for the percutaneous endoscopic procedure. Additional 0.2% ropivacaine was administered as needed based on surgical duration and anesthesia level. Rescue analgesia with sufentanil was provided in cases of severe intraoperative pain.

Outcome measures

Surgical outcomes (operative time, fluoroscopy frequency, postoperative time to ambulation, ropivacaine dosage, and rate of rescue sufentanil analgesia), hemodynamic parameters, Ramsay sedation scores, visual analog scale (VAS) pain scores, pain mediators, and incidence of adverse events (e.g., pruritus and urinary retention) were compared between the two groups.

Hemodynamic parameters: Upon entry to the operating room, all patients were connected to a vital signs monitor (model VS-600, Guangdong Medical Device Approval Number 20162070982). Mean arterial pressure (MAP), heart rate (HR), and peripheral oxygen saturation (SpO_2) were recorded at baseline (T0), during percutaneous puncture (T1), foramenoplasty (T2), nucleus pulposus removal (T3), and skin suturing (T4).

Ramsay sedation score: Ramsay sedation scores at T0-T4 were extracted from medical records. The Ramsay sedation scale was defined as follows: Level 1: the patient exhibits signs of anxiety, agitation, or restlessness; Level 2: the patient is calm, cooperative, and aware of their surroundings; Level 3: the patient responds exclusively to verbal commands; Level 4: the patient is in a state of sleep but demonstrates a rapid reaction upon light tapping of the glabella or exposure to a loud sound; Level 5: the patient is asleep, with a noticeably delayed reaction to light glabellar tapping or a loud auditory stimulus; and Level 6: the patient is asleep and unresponsive to any form of stimulation [6].

VAS pain score: VAS pain scores at T0, 2 hours postoperatively (T5), 6 hours postoperatively (T6), 24 hours postoperatively (T7), 48 hours postoperatively (T8), and 72 hours postoperatively (T9) were recorded. Higher scores indicates more severe pain [7].

Laboratory indicators: Peripheral venous blood samples (5 mL) were collected preoperatively, and at 24 h and 72 h postoperatively. After standing at room temperature for 30 minutes, samples were centrifuged at 3,500 rpm (centrifugal radius 12.5 cm) for 10 minutes. Serum levels of tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), C-X-C motif chemokine ligand 8 (CXCL8), 5-hydroxytryptamine (5-HT), and prostaglandin E_2 (PGE_2) were measured using enzyme-linked immunosorbent assay kits (Shenzhen Jingmei Biotechnology, China). Absorbance was measured using a microplate reader (Shenzhen Mindray, model MR-96A, Guangdong Medical Device Approval Number 20192220393).

Statistical analysis

Statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro-Wilk normality test and for homogeneity of variance using Levene's test. Normally distributed data with homogeneous variance were expressed as mean ± standard deviation ($\bar{x} \pm s$). For repeated measurement data at multiple time points, intergroup comparisons were performed using repeated measures analysis of variance (ANOVA). When significant interaction effects were detected, simple effects analysis was further conducted. *P*-values for intergroup compari-

Table 1. Comparison of baseline data after matching ($\bar{x} \pm s$)

Baseline Characteristic	Control group (n=55)	Observation group (n=55)	t/χ^2	P
Gender [n (%)]			0.152	0.697
Male	32 (58.18)	34 (61.82)		
Female	23 (41.82)	21 (38.18)		
Age [($\bar{x} \pm s$), years]	48.56±12.34	50.02±11.87	-0.632	0.528
BMI [($\bar{x} \pm s$), kg/m^2]	24.12±2.85	23.89±2.96	0.415	0.679
Disease duration [($\bar{x} \pm s$), months]	8.56±4.23	9.02±5.01	-0.520	0.604
Affected segment [n (%)]			0.411	0.814
L3/4	5 (9.09)	6 (10.91)		
L4/5	31 (56.36)	33 (60.00)		
L5/S1	19 (34.55)	16 (29.09)		
Herniation type [n (%)]			0.200	0.905
Paracentral	15 (27.27)	13 (23.64)		
Posterolateral	34 (61.82)	36 (65.45)		
Sequestrated/Extruded	6 (10.91)	6 (10.91)		
Comorbidities [n (%)]				
Hypertension	11 (20.00)	9 (16.36)	0.244	0.621
Diabetes Mellitus	5 (9.09)	6 (10.91)	0.101	0.751

Note: BMI, body mass index. *P* values were obtained by independent t-test for continuous variables and χ^2 test for categorical variables. After 1:1 propensity score matching (caliper = 0.2 × standard deviation of the logit), the standardized mean differences (SMD) for all covariates were < 0.1, indicating a well-balanced baseline between the control group and the observation group (all P > 0.05).

sons at each time point were corrected for multiple comparisons using the Bonferroni method. Within-group comparisons were performed using paired samples t-tests. Categorical data were expressed as n (%) and analyzed using the χ^2 test. A *P*-value < 0.05 was considered statistically significant.

Results

Comparison of baseline characteristics after PSM

After PSM, no significant differences were observed between the observation group and the control group in baseline characteristics such as sex, age, BMI, disease duration, affected segment, herniation type, or comorbidities (all P > 0.05; **Table 1**).

Comparison of surgical outcomes

The observation group had a longer postoperative time to ambulation, lower ropivacaine dosage, and a lower rate of rescue sufentanil analgesia compared with the control group (all P < 0.05). However, no statistically significant differences were observed between the two groups in operative time or fluoroscopy frequency (both P > 0.05). See **Table 2**.

Comparison of hemodynamic parameters

Repeated measures ANOVA demonstrated significant time effects, between-group effects, and time × group interaction effects for MAP and HR (P < 0.05). In contrast, no significant time, between-group, or time × group interaction effects were observed for SpO_2 (P > 0.05). Within-group comparisons showed that MAP and HR at T1-T3 were significantly higher than baseline values (T0) in both groups (P < 0.05). Between-group comparisons further revealed that MAP and HR at T1-T3 were significantly lower in the observation group than that in the control group (P < 0.05, Bonferroni corrected). The significant time × group interaction indicated that the temporal changes of MAP and HR differed statistically between the two groups (P < 0.05). See **Table 3**.

Comparison of Ramsay sedation scores

Repeated measures ANOVA showed significant time effects, between-group effects, and time × group interaction effects for Ramsay sedation scores (P < 0.05). Within-group comparisons showed that Ramsay scores at T1-T4 were significantly higher than those at T0 in both

Table 2. Comparison of surgical outcomes ($\bar{x}\pm s$)

Group	n	Operative Time [($\bar{x}\pm s$), min]	Fluoroscopy frequency [($\bar{x}\pm s$), n]	Time to Ambulation [($\bar{x}\pm s$), h]	Ropivacaine Dosage [($\bar{x}\pm s$), mg]	Rate of sufentanil rescue analgesia [n (%)]
Control Group	55	66.47±13.41	7.24±2.13	7.51±2.02	73.52±10.43	14 (25.45)
Observation Group	55	65.56±14.83	7.67±2.45	9.18±1.47	42.36±8.15	5 (9.09)
t/χ^2		0.338	-0.982	-4.957	17.458	5.153
P		0.736	0.328	0.000	0.000	0.023

Table 3. Comparison of hemodynamics ($\bar{x}\pm s$)

Indicator	Time	Control Group (n=55)	Observation Group (n=55)	F	P
MAP (mmHg)	T0	94.52±3.63	93.74±4.12	F_{time}=82.214, F_{group}=18.626, $F_{time \times group}$=12.362	P_{time} < 0.001, P_{group} < 0.001, $P_{time \times group}$ < 0.001
	T1	101.23±5.96*	96.88±4.87*,#		
	T2	104.74±5.26*	98.86±6.02*,#		
	T3	110.58±6.92*	103.23±7.11*,#		
	T4	96.36±5.77	95.63±6.34		
HR (beats/min)	T0	74.52±3.16	73.98±3.57	F_{time}=42.158, F_{group}=42.036, $F_{time \times group}$=9.528	P_{time} < 0.001, P_{group} < 0.001, $P_{time \times group}$ < 0.001
	T1	77.96±4.02*	75.93±3.86*,#		
	T2	78.17±3.85*	76.32±4.12*,#		
	T3	81.27±4.02*	76.52±3.88*,#		
	T4	73.41±4.26	74.22±4.09		
SpO_2 (%)	T0	98.21±1.05	97.98±1.12	F_{time}=0.845, F_{group}=0.369, $F_{time \times group}$=0.291	P_{time}=0.472, P_{group}=0.546, $P_{time \times group}$=0.883
	T1	98.02±1.17	98.23±1.06		
	T2	97.96±1.15	98.25±1.27		
	T3	98.23±0.94	98.09±1.17		
	T4	98.41±0.89	98.50±0.93		

Note: Data are presented as mean ± SD. MAP, mean arterial pressure; HR, heart rate; SpO_2, oxygen saturation. T0, baseline (before surgery); T1, during percutaneous puncture; T2, during foraminoplasty; T3, during nucleus pulposus removal; T4, at skin closure. Repeated measures ANOVA was performed for each parameter; the corresponding *F* values and *P* values for time effects, group effects, and time × group interactions are displayed in the table. Between-group comparisons at each time point were corrected for multiple testing using the Bonferroni method. *P < 0.05 compared with T0; #P < 0.05 compared with the control group.

Table 4. Comparison of Ramsay sedation scores ($\bar{x}\pm s$)

Group	n	Comparison of Ramsay Sedation Scores (points)				
		T0	T1	T2	T3	T4
Control Group	55	1.93±0.34	2.37±0.39*	2.26±0.41*	2.43±0.39*	2.17±0.37*
Observation Group	55	1.96±0.38	2.92±0.43*,#	2.87±0.48*,#	2.88±0.41*,#	2.63±0.45*,#
F		F_{time}=68.431, F_{group}=24.582, $F_{time \times group}$=15.863				
P		P_{time} < 0.001, P_{group} < 0.001, $P_{time \times group}$ < 0.001				

Note: Data are presented as mean ± SD. T0, baseline; T1, during percutaneous puncture; T2, during foraminoplasty; T3, during nucleus pulposus removal; T4, at skin closure. Between-group comparisons at each time point were corrected using the Bonferroni method. *P < 0.05 compared with T0; #P < 0.05 compared with the control group at the same time point.

groups (P < 0.05). Moreover, the observation group exhibited significantly higher Ramsay sedation scores at T1-T4 than the control group (P < 0.05, Bonferroni corrected). The significant time × group interaction further indicated distinct temporal patterns of sedation between the two groups (P < 0.05). See **Table 4**.

Comparison of VAS pain scores

Repeated measures ANOVA showed significant time effects, between-group effects, and time × group interaction effects for VAS pain scores (P < 0.05). Pain scores at T5-T9 were significantly lower than at T0 in both groups (P <

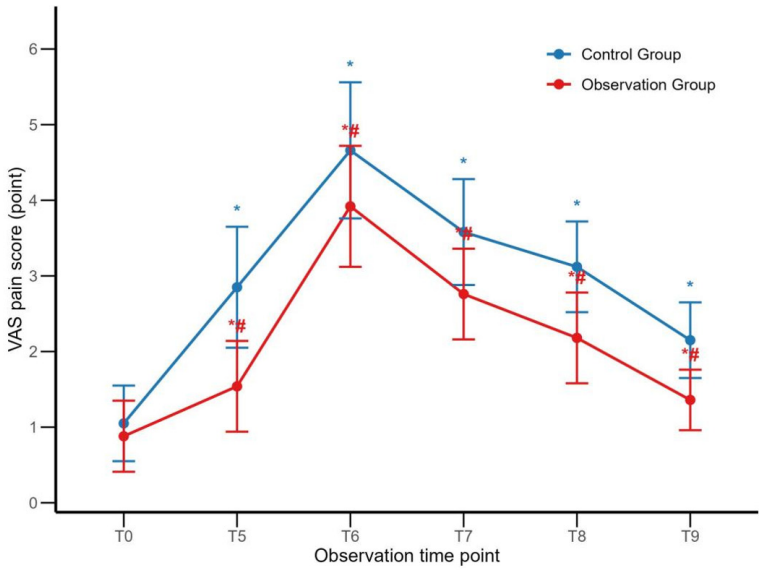

Figure 1. Comparison of VAS pain scores between the two groups. Note: T0, before surgery; T5, 2 h after surgery; T6, 6 h after surgery; T7, 24 h after surgery; T8, 48 h after surgery; T9, 72 h after surgery. *P < 0.05 compared with T0; #P < 0.05 compared with the control group. Repeated measures ANOVA revealed significant time effect (F_{time}=210.252, P < 0.001), group effect (F_{group}=32.698, P < 0.001), and time × group interaction ($F_{time \times group}$=28.415, P < 0.001).

0.05). The observation group had significantly lower VAS scores at T5-T9 compared to the control group (P < 0.05, Bonferroni corrected). The significant time × group interaction indicated that postoperative pain trajectories differed significantly between the two groups (P < 0.05; **Figure 1**).

Comparison of inflammatory markers

Repeated measures ANOVA demonstrated significant time effects, between-group effects, and time × group interaction effects for TNF-α, IL-1β, and CXCL8 (all P < 0.05). In both groups, serum levels of these inflammatory markers at 24 h and 72 h postoperatively were significantly increased compared with preoperative values (P < 0.05). The observation group had significantly lower levels of TNF-α, IL-1β, and CXCL8 at both postoperative time points compared to the control group (P < 0.05, Bonferroni corrected). The significant time × group interactions indicated distinct temporal changes in inflammatory responses between the two groups (P < 0.05). See **Table 5**.

Comparison of pain mediators

Repeated measures ANOVA showed significant time effects, between-group effects, and time × group interaction effects for 5-HT and PGE_2 levels (all P < 0.05). In both groups, serum 5-HT and PGE_2 levels increased at 24 h postoperatively compared with preoperative values and subsequently decreased at 72 h postoperatively (P < 0.05). The observation group exhibited significantly lower levels of 5-HT and PGE_2 at both 24 h and 72 h postoperatively compared to the control group (P < 0.05, Bonferroni corrected). Significant time × group interactions were observed, indicating different temporal patterns of pain mediator changes between the two groups (P < 0.05). See **Table 6**.

Comparison of adverse events

No statistically significant difference was observed in the overall incidence of adverse events between the two groups (P > 0.05). See **Table 7**.

Discussion

The incidence of LDH is increasing annually, with reported prevalence rates of approximately 3.0%-3.1% in males and 1%-1.3% in females. In addition, the disease has shown a trend toward younger onset, with the highest incidence occurring between 30-50 years [8]. The pathogenesis of LDH is complex, with existing research suggesting strong associations with genetics, lifestyle habits, and occupation [9]. Surgical intervention is recommended for patients who fail to respond to conservative treatment for more than 3 months, experience worsening symptoms, or develop severe manifestations such as foot drop or urinary incontinence [10]. Currently, commonly used anesthetic techniques for percutaneous endoscopic surgery include local anesthesia combined with sedation or epidural anesthesia [11-13].

Ropivacaine, an amide local anesthetic, produces a sensory-motor dissociation effect at low concentrations, offering effective sensory blockade while largely preserving motor function [14]. During percutaneous endoscopic pro-

Table 5. Comparison of inflammatory markers ($\bar{x} \pm s$)

Group	n	TNF-α (ng/L)			IL-1β (ng/L)			CXCL8 (ng/L)		
		Preoperative	Postoperative 24 h	Postoperative 72 h	Preoperative	Postoperative 24 h	Postoperative 72 h	Preoperative	Postoperative 24 h	Postoperative 72 h
Control Group	55	6.85±1.74	18.98±4.74*	10.74±3.11*	13.20±3.31	42.56±7.85*	24.12±4.65*	1.25±0.29	9.85±1.74*	4.74±1.21*
Observation Group	55	6.91±1.66	13.11±3.28*,#	8.52±2.69*,#	13.28±3.17	36.62±5.29*,#	18.85±3.79*,#	1.22±0.36	6.98±1.58*,#	3.23±0.76*,#
F		F_{time}=125.632, F_{group}=20.481, $F_{time \times group}$=18.326			F_{time}=230.417, F_{group}=35.629, $F_{time \times group}$=29.853			F_{time}=90.541, F_{group}=15.869, $F_{time \times group}$=13.209		
P		P_{time} < 0.001, P_{group} < 0.001, $P_{time \times group}$ < 0.001			P_{time} < 0.001, P_{group} < 0.001, $P_{time \times group}$ < 0.001			P_{time} < 0.001, P_{group} < 0.001, $P_{time \times group}$ < 0.001		

Note: Data are presented as mean ± SD. TNF-α, tumor necrosis factor-alpha; IL-1β, interleukin-1 beta; CXCL8, C-X-C motif chemokine ligand 8. Repeated measures ANOVA was performed for each marker; the *F* values and corresponding *P* values for time effects, group effects, and time × group interactions are displayed in the table (all interaction P < 0.001). Between-group comparisons at each time point were corrected for multiple testing using the Bonferroni method. *P < 0.05 compared with preoperative values; #P < 0.05 compared with the control group.

Table 6. Comparison of pain mediators ($\bar{x} \pm s$)

Group	n	5-HT (ng/mL)			PGE_2 (ng/L)		
		Preoperative	Postoperative 24 h	Postoperative 72 h	Preoperative	Postoperative 24 h	Postoperative 72 h
Control Group	55	421.25±62.58	578.96±73.63*	379.52±51.02*	352.32±51.85	458.96±62.96*	317.85±45.62*
Observation Group	55	417.96±68.63	512.85±68.95*,#	339.65±46.74*,#	346.98±56.87	422.12±50.78*,#	269.63±41.79*,#
F		F_{time}=150.214, F_{group}=22.639, $F_{time \times group}$=20.532			F_{time}=138.474, F_{group}=19.637, $F_{time \times group}$=17.598		
P		P_{time} < 0.001, P_{group} < 0.001, $P_{time \times group}$ < 0.001			P_{time} < 0.001, P_{group} < 0.001, $P_{time \times group}$ < 0.001		

Note: Data are presented as mean ± SD. 5-HT, 5-hydroxytryptamine; PGE2, prostaglandin E2. Between-group comparisons at each time point were corrected using the Bonferroni method. *P < 0.05 compared with preoperative values; #P < 0.05 compared with the control group at the same time point.

Table 7. Comparison of adverse reactions [n (%)]

Group	n	Pruritus	Urinary Retention	Nausea/Vomiting	Hypotension	Total
Control Group	55	2 (3.64)	1 (1.82)	2 (3.64)	0 (0.00)	5 (9.09)
Observation Group	55	1 (1.82)	1 (1.82)	3 (5.45)	1 (1.82)	6 (10.91)
χ^2						0.101
P						0.751

cedures, steps such as puncture localization and removal of the nucleus pulposus carry a risk of injury to the nerve roots or the dural sac, making intraoperative patient feedback important for surgical safety [15]. Low-concentration ropivacaine can satisfy the dual requirements of adequate anesthesia and preservation of patient responsiveness during surgery [16]. However, there remains no consensus regarding the optimal route of administration for low-concentration ropivacaine, particularly whether epidural anesthesia or local infiltration anesthesia provides superior clinical outcomes.

In the present study, compared with local infiltration anesthesia, epidural anesthesia using low-concentration ropivacaine was associated with prolonged postoperative ambulation time, lower total ropivacaine dose, lower rate of rescue sufentanil analgesia, higher intraoperative Ramsay sedation scores, and lower postoperative pain scores. The pharmacological mechanism underlying these findings may be related to the selective blockade characteristics of ropivacaine. Low-concentration ropivacaine reversibly blocks voltage-gated sodium channels on nerve cell membranes, which inhibits action potential conduction and interrupts pain signal transmission. Its blockade of unmyelinated C fibers which carry pain signals occurs more quickly than that of A fibers which are involved in motor function, resulting in the sensory-motor dissociation effect [17].

Under local infiltration anesthesia, ropivacaine is injected directly into the surgical area, where it diffuses locally to nerve endings and suppresses impulse conduction. While this approach offers a rapid onset, its duration is relatively short. It often requires repeated layer-by-layer infiltration through the endoscope, leading to higher ropivacaine consumption and more pronounced postoperative pain, which in turn frequently necessitates rescue analgesia [18]. In contrast, epidural anesthesia delivers ropivacaine directly into the epidural space, where it blocks sodium channels on nerve roots and inhibits pain signal transmission. This approach provides a broader and more sustained analgesic effect, while allowing additional doses to be administered through an indwelling epidural catheter as needed during surgery. As a result, total ropivacaine consumption and the requirement for rescue analgesia are reduced, and postoperative analgesia is prolonged [19]. However, because the drug is metabolized more slowly in the epidural space, lower limb sensory and motor function may be temporarily affected, which can delay ambulation after surgery [20].

Maintaining intraoperative hemodynamic stability is essential for patient safety and for reducing the risk of adverse cardiovascular or cerebrovascular events. In this study, patients receiving epidural anesthesia with low-concentration ropivacaine exhibited more stable intraoperative hemodynamics, as reflected by smaller fluctuations in MAP and HR during surgery. Local infiltration anesthesia acts primarily at the surgical site and provides a relatively limited range and depth of neural blockade, which may allow nociceptive stimulation during surgery to trigger sympathetic activation and hemodynamic fluctuations [21]. Epidural anesthesia, by contrast, can block sympathetic nerves and reduce vasoconstrictor responses, thereby contributing to better blood pressure stability. The ability to give supplemental medication and maintain a controllable sensory level also helps keep MAP and HR stable [22]. We observed no significant difference in SpO_2 between the two groups, likely because neither low-concentration local infiltration nor low-concentration epidural anesthesia impairs the respiratory muscles or central ventilatory drive, so oxygen saturation generally remains unaffected.

Surgery trauma inevitably induces a systemic stress response. In percutaneous endoscopic

lumbar disc procedures, the local tissue trauma triggers a cascade of physiological responses. Cell membranes break down, releasing arachidonic acid, which cyclooxygenase then converts into PGE_2. At the same time, surgical stress activates the neuroimmune system, leading to the release of 5-HT, TNF-α, and other pain-related mediators. TNF-α, in turn, stimulates the production of proinflammatory cytokines such as IL-1β and CXCL8, amplifying the inflammatory response [23]. These inflammatory mediators directly stimulate peripheral nociceptors and contribute to postoperative pain, which may adversely affect functional recovery [24]. Moreover, serum levels of inflammatory cytokines and pain mediators increased postoperatively in both groups; however, the magnitude of increase was significantly lower in the epidural anesthesia group. Compared with local infiltration, epidural anesthesia provides a broader area of anesthesia, more complete analgesia, and superior muscle relaxation. More importantly, the use of an indwelling epidural catheter enables continuous administration of anesthetic agents throughout the procedure, resulting in more effective suppression of nociceptive signal transmission. By suppressing the transmission of pain signals, epidural anesthesia appears to attenuate the overall surgical stress response, which in turn reduces the release of inflammatory factors and pain mediators [25].

Conclusion

To sum up, for percutaneous endoscopic lumbar disc herniation surgery, low-concentration ropivacaine delivered via epidural anesthesia has clear advantages over local infiltration. Specifically, epidural anesthesia provided better intraoperative hemodynamic stability, deeper sedation, improved postoperative analgesia, and greater suppression of inflammatory cytokines and pain mediators. These findings suggest that low-concentration ropivacaine epidural anesthesia may represent a more advantageous anesthetic strategy for percutaneous endoscopic surgery. Nevertheless, several limitations of this study should be acknowledged. First, the retrospective design carries an inherent risk of bias; while PSM balanced key baseline variables, residual confounding cannot be fully excluded. Second, intraoperative variables such as blood loss and fluid infusion volume were not recorded and controlled, and these could potentially influence hemodynamic and inflammatory outcomes. Third, patients' preoperative psychological status was not assessed, which could be a confounding source for intraoperative hemodynamic responses and subjective postoperative pain scores. Fourth, correlation analyses between hemodynamic parameters and sedation scores, as well as between inflammatory factors and VAS pain scores, were not performed; likewise, multivariate regression analysis was not conducted to identify independent predictors of postoperative pain. These omissions limit the depth of statistical inference. However, the absence of significant difference in operative time and baseline levels of VAS, 5-HT, and PGE_2 between the two groups may partially mitigate concerns regarding these uncontrolled confounders. Future prospective, multicenter, randomized controlled trials with larger sample sizes are warranted to further validate the findings of the present study.

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Disclosure of conflict of interest

None.

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