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Original Article

Effect of ropivacaine-dexamethasone combination on postpartum uterine pain, inflammation, and breastfeeding in multiparous women

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Abstract: Objective: To investigate the effects of epidural ropivacaine combined with dexamethasone on postpartum uterine cramping pain, inflammatory markers, and breastfeeding behavior in multiparous women. Methods: This retrospective cohort study included 336 women who underwent vaginal delivery and received postpartum epidural analgesia between May 2022 and November 2024, including 211 multiparous women. The observation group (n = 172) received 0.15% ropivacaine combined with 10 mg dexamethasone, while the control group (n = 164) received only 0.15% ropivacaine. The severity of uterine cramping pain was assessed using the Visual Analogue Scale (VAS) at 2, 6, 12, 24, and 48 hours postpartum. Serum levels of tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and interleukin-10 (IL-10) were measured using ELISA, and C-reactive protein (CRP) was measured using a quantitative laboratory assay, at 6, 24, and 72 hours postpartum. Breastfeeding indicators and adverse events were recorded. Results: The VAS scores at all time points in the observation group were significantly lower than those of the control group (all $P < 0.001$), with a lower rescue analgesia rate (10.47% vs. 21.34%, $P = 0.006$). At 24 hours postpartum, the observation group showed significantly lower levels of TNF-α, IL-6, and CRP, and significantly higher levels of IL-10 (all $P < 0.001$); shorter first breastfeeding time (38.00 minutes vs. 52.00 minutes, $P < 0.001$); higher frequency of breastfeeding within 48 hours postpartum; and a higher rate of exclusive breastfeeding on day 7 postpartum (84.30% vs. 68.29%, $P < 0.001$). There were no significant differences in adverse events or neonatal outcome between the two groups (both $P > 0.05$). Conclusions: Epidural analgesia with ropivacaine combined with dexamethasone effectively alleviates postpartum uterine cramping pain in multiparous women, regulates the inflammatory responses, improves breastfeeding outcomes, and has a good safety profile.

Keywords: Postpartum analgesia, dexamethasone, ropivacaine, epidural analgesia, uterine cramping pain, inflammatory markers, breastfeeding

Introduction

Postpartum uterine cramping pain, caused by contractions during uterine involution, is one of the most common causes of pain during the puerperium [1]. Studies indicate that approximately 47% of women experience significant cramping pain within 48 hours after delivery. Multiparous women typically experience more severe pain due to increased sensitivity of uterine muscle fibers after repeated stretching [2]. Breastfeeding may further exacerbate this pain [2]. Postpartum pain reduces maternal comfort and quality of life and may also trigger anxiety and depression, thereby affecting early mother-infant interaction and the establishment of attachment [3]. In recent years, the effect of postpartum pain on breastfeeding has received increasing attention. Pain-induced stress responses inhibit the function of the hypothalamus-pituitary axis, leading to a decrease in oxytocin and prolactin secretion, impairing the initiation of lactation and the milk ejection reflex [4]. Establishing breastfeeding

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behavior as early as possible helps to achieve better breastfeeding outcomes. Women who start breastfeeding within one hour of delivery are twice as likely to achieve exclusive breastfeeding within six weeks postpartum compared to those who initiate later (OR = 2.01, 95% CI: 1.12-3.61) [5]. Childbirth-induced bodily damage triggers inflammatory responses and participates in the aforementioned adverse physiologic processes. In the early postpartum period, elevated levels of pro-inflammatory cytokines such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6), as well as systemic inflammatory markers such as C-reactive protein (CRP), may contribute to pain sensitization, further altering the local microenvironment of the breast and thus interfering with lactation [6]. Interleukin-10 (IL-10) is a potentially protective anti-inflammatory cytokine. Studies have found that increasing IL-10 levels and decreasing IL-6, TNF-α, and CRP levels postpartum may help relieve pain and improve maternal physical recovery and well-being [7]. Therefore, exploring methods that can effectively manage pain and regulate inflammatory responses is of great significance for postpartum recovery and successful breastfeeding. Currently, analgesics used to relieve postpartum uterine cramping pain mainly include nonsteroidal anti-inflammatory drugs (NSAIDs), opioids, and regional block anesthesia techniques. A Cochrane systematic review showed that NSAIDs were more effective than placebo in treating postpartum colic and may be more effective than opioids, but their analgesic effect was limited [1]. Although opioids have significant analgesic effects, they can be passed to newborns through breast milk, causing adverse reactions such as nausea, vomiting, and drowsiness [8]. The regional block effect produced by local anesthetics when used alone is short-lived, often requiring repeated administration or continuous infusion [9]. Therefore, the development of adjuvants that can prolong the duration of analgesia, improve the analgesic efficacy, and ensure maternal and infant safety has become a focus of clinical research.

Dexamethasone is a long-acting glucocorticoid with potent anti-inflammatory and immunomodulatory effects. Its anti-inflammatory mechanism primarily involves inducing the synthesis of lipocorticoid proteins and inhibiting the activity of phospholipase A2, thereby reducing the release of arachidonic acid and the production of inflammatory mediators such as prostaglandins [10]. Recent studies have confirmed that dexamethasone, as an adjuvant to local anesthetics, can significantly prolong the duration of nerve block. A Cochrane systematic review involving 35 randomized controlled trials and 2,702 patients showed that perineural administration of dexamethasone could prolong the duration of sensory block by approximately 6.70 hours (95% CI: 5.54-7.85) and significantly reduced pain scores at 12 and 24 hours postoperatively [11]. In the field of obstetrics, studies have reported that ropivacaine combined with dexamethasone for epidural analgesia in preeclamptic women can achieve better analgesia and sedation while stabilizing hemodynamic indices [12]. Given that dexamethasone has both analgesic and anti-inflammatory effects, its use in the management of postpartum uterine colic may optimize pain control through its anti-inflammatory effect and promote early establishment of breastfeeding behavior. A future cohort study of 468 women who underwent cesarean section showed that primiparity, hypoalbuminemia, and postpartum depression were independent risk factors for delayed milk production, while breastfeeding frequency was negatively correlated with the risk of delayed milk production [13].

Based on this background, this study aimed to observe the clinical effects of ropivacaine alone or in combination with dexamethasone for postpartum analgesia among multiparous women, analyze its effects on postpartum pain, inflammatory factor levels, and breastfeeding behavior, and explore the association among the three factors, providing evidence-based support for the management of postpartum uterine cramping pain and the achievement of successful breastfeeding.

Materials and methods

Study population

This retrospective cohort study included women who underwent vaginal delivery and received postpartum epidural analgesia at Xi'an Savaid Obstetrics-Gynecology Hospital, Northwest Women's and Children's Hospital and Shaanxi Provincial Nuclear Industry 215 Hospital between May 2022 and November 2024. A total of 336 patients were divided into

two groups based on the analgesia regimen recorded in their medical records: the observation group (n = 172) received epidural analgesia with ropivacaine combined with dexamethasone, and the control group (n = 164) received epidural analgesia with ropivacaine only. This study was approved by the Ethics Committee of the Shaanxi Provincial Nuclear Industry 215 Hospital.

Inclusion criteria: (1) age 18-45 years; (2) gestational age ≥ 37 weeks with singleton cephalic presentation; (3) vaginal delivery; (4) American Society of Anesthesiologists (ASA) classification I-II; (5) receipt of postpartum epidural analgesia; and (6) complete medical records.

Exclusion criteria: (1) multiple pregnancy; (2) abnormal fetal position; (3) macrosomia (birth weight ≥ 4000 g); (4) severe pregnancy complications such as severe preeclampsia or poorly controlled gestational diabetes mellitus; (5) allergy to the drugs used in the study; (6) coagulation disorders or use of anticoagulants; (7) history of chronic pain or long-term use of analgesics; (8) infection at the puncture site; (9) mental illness or cognitive impairment; (10) conversion to cesarean section; (11) incomplete medical records or missing follow-up data.

Analgesia protocol

All subjects had an epidural catheter retained after delivery, and patient-controlled epidural analgesia (PCEA) was administered by an anesthesiologist using a disposable 100 mL infusion pump [14]. According to the analgesia protocol recorded in the medical records, patients in the control group received 100 mL of 0.15% ropivacaine hydrochloride, while patients in the observation group received 0.15% ropivacaine combined with 10 mg (2 mL) of dexamethasone sodium phosphate. The PCEA parameters were set identically for both groups: background infusion rate of 6 mL/h, patient-controlled bolus of 4 mL, lockout interval of 30 min, and a maximum hourly dose of 14 mL. PCEA was initiated immediately after placental delivery and continued for 48 hours until catheter removal. Uterine cramping pain was assessed using the Visual Analogue Scale (VAS) at 2, 6, 12, 24, and 48 hours postpartum. When the VAS score was ≥ 4 and a single PCEA dose provided inadequate relief, 50 mg of tramadol hydrochloride was administered intravenously as rescue analgesia. The dose could be repeated at intervals of at least 4 hours, with a maximum daily dose not exceeding 200 mg. During analgesia, vital signs and uterine contractions were routinely monitored, and patients were encouraged to ambulate and breastfeed as early as possible.

Outcome measures

Data were collected from electronic medical records, nursing documentation, and telephone follow-ups.

Baseline characteristics included maternal age, gestational age, body mass index (BMI), parity, duration of labor, newborn birth weight, and Apgar scores.

Primary outcomes were: (1) exclusive breastfeeding status on day 7 postpartum; and (2) VAS score at 24 hours postpartum.

Secondary outcomes included: (1) VAS scores at 2, 6, 12, and 48 hours postpartum; (2) time to first rescue analgesia and total dose of rescue analgesics within 24 hours; (3) analgesia satisfaction score (5-point Likert scale); (4) serum levels of TNF-α, IL-6, IL-10, and CRP, measured at 6, 24, and 72 hours postpartum; (5) breastfeeding-related indicators, including time to first breastfeeding, breastfeeding frequency within 48 hours postpartum, and time to lactation initiation; (6) adverse events, including nausea, vomiting, dizziness, pruritus, and hypotension.

Pain assessment

Pain was assessed using a standard 100 mm Visual Analog Scale (VAS) [15], where 0 mm = “no pain” and 100 mm = “most severe pain” (converted to a 0-10 score). Higher scores indicate more severe pain. Rescue analgesia (intravenous tramadol 50 mg) was administered when the VAS score was ≥ 4 and a single PCEA dose was inadequate. Assessments were performed by obstetric nurses at each time point and recorded in the nursing documentation.

Breastfeeding assessment

Breastfeeding indicators were recorded by obstetric nurses in a breastfeeding log. Breastfeeding status on day 7 was obtained via telephone follow-up after discharge and recorded in a follow-up logbook.

Laboratory methods

Blood samples were collected at 6, 24, and 72 hours postpartum. Serum levels of TNF-α, IL-6, and IL-10 were measured using commercially available double-antibody sandwich ELISA kits (Shanghai Enzyme-Link Biotechnology Co., Ltd.; batch numbers: TNF-α ml058097, IL-6 ml064292, and IL-10 ml058098). CRP was measured using a quantitative laboratory assay in the hospital laboratory. Blood samples were centrifuged at 3000 rpm for 10 minutes, and serum was stored at -80°C until analysis. Absorbance for ELISA was measured at 450 nm using an ELISA reader.

Statistical analysis

Data analysis was performed using R version 4.5.1 and SPSS version 27.0. Normality of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed continuous data were presented as mean ± standard deviation, and independent samples t-tests were used for comparisons between groups. Non-normally distributed continuous data were expressed as median (interquartile range) [M (Q1, Q3)], and the Mann-Whitney U test was used for comparisons between groups. Repeated measures data, including VAS scores at multiple time points and inflammatory marker levels (TNF-α, IL-6, IL-10, and CRP), were analyzed using repeated measures ANOVA to test for between-group effects, time effects, and interaction effects. When significant main effects or interaction effects were observed, post hoc pairwise comparisons were performed using the Bonferroni correction to adjust for multiple comparisons. Categorical data were expressed as frequency (percentage) [n (%)], and the χ^2 test or Fisher's exact test was used for comparisons between groups. The Mann-Whitney U test was used for comparisons of ordinal data between groups. Before correlation analysis, skewness (|skewness| < 1) and excess kurtosis (|kurtosis| < 2) were used as the primary criteria to assess the normality of continuous variables, supplemented by the Shapiro-Wilk test. Pearson correlation analysis was used when both variables approximately follow a normal distribution; otherwise, Spearman rank correlation analysis was applied. The rate of exclusive breastfeeding on day 7 postpartum did not meet the normality criterion (skewness = 1.240), therefore Spearman rank correlation analysis was used. All other correlation analyses used Pearson correlation analysis. Repeated measures ANOVA was performed before the Mauchly test for sphericity. If the sphericity was not established ($P < 0.05$), Greenhouse-Geisser correction was used. The significance level was set at $\alpha = 0.05$, and $P < 0.05$ indicated significance.

Results

Baseline characteristics

There were no significant differences between the two groups in terms of age ($P = 0.859$), body mass index at delivery ($P = 0.981$), insurance type ($P = 0.568$), parity ($P = 0.368$), gestational age at delivery ($P = 0.929$), gestational diabetes mellitus ($P = 0.829$), gestational hypertension ($P = 0.649$), total labor duration ($P = 0.377$), or episiotomy rate ($P = 0.525$). Neonatal-related indicators, including birth weight, sex, length, and head circumference, were also comparable between groups (all $P > 0.05$). Baseline characteristics were well-balanced, confirming the comparability of the two groups (**Table 1**).

Postpartum VAS scores

The VAS scores in the observation group were significantly lower than those of the control group at all postpartum time points (2, 6, 12, 24, and 48 hours) (all $P < 0.001$). Within-group analysis showed that the VAS scores in the observation group progressively decreased across consecutive time points, and all differences were significant (all $P < 0.05$). In the control group, the VAS scores remained stable from 2 to 6 hours postpartum, after which they began to decline. The results indicate that the decrease in VAS scores in the observation group was significantly greater than that in the control group from 2 to 6 hours postpartum ($P = 0.024$), whereas there were no significant differences between groups at 6-12 hours ($P = 0.904$), 12-24 hours ($P = 0.555$), or 24-48 hours ($P = 0.289$) (**Figure 1**).

Rescue analgesia

Compared to the control group, the rescue analgesia rate in the observation group was significantly lower ($P < 0.05$). In subjects requir-

Table 1. Comparison of baseline characteristics between groups [n (%) or $\bar{x} \pm s$]

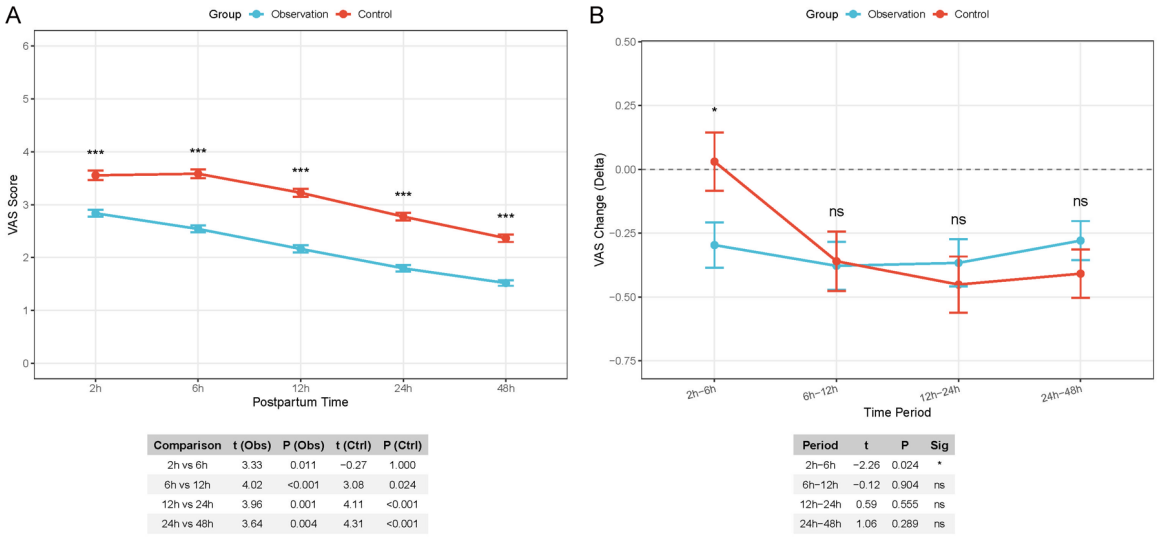
Variable	Total	Control Group (n = 164)	Observation Group (n = 172)	t/χ^2	*P* value
Age				0.303	0.859
≤ 30 years	188 (55.95%)	90 (54.88%)	98 (56.98%)		
31-35 years	114 (33.93%)	56 (34.15%)	58 (33.72%)		
> 35 years	34 (10.12%)	18 (10.98%)	16 (9.30%)		
BMI at delivery				0.038	0.981
< 24 kg/m^2	100 (29.76%)	48 (29.27%)	52 (30.23%)		
24-28 kg/m^2	167 (49.70%)	82 (50.00%)	85 (49.42%)		
> 28 kg/m^2	69 (20.54%)	34 (20.73%)	35 (20.35%)		
Insurance type				0.326	0.568
Urban employee insurance	206 (61.31%)	98 (59.76%)	108 (62.79%)		
Other	130 (38.69%)	66 (40.24%)	64 (37.21%)		
Parity				0.811	0.368
Primiparous	125 (37.20%)	65 (39.63%)	60 (34.88%)		
Multiparous	211 (62.80%)	99 (60.37%)	112 (65.12%)		
Gestational age at delivery				0.147	0.929
37-38 weeks	60 (17.86%)	28 (17.07%)	32 (18.60%)		
39-40 weeks	247 (73.51%)	122 (74.39%)	125 (72.67%)		
> 40 weeks	29 (8.63%)	14 (8.54%)	15 (8.72%)		
Gestational diabetes mellitus				0.046	0.829
Yes	34 (10.12%)	16 (9.76%)	18 (10.47%)		
No	302 (89.88%)	148 (90.24%)	154 (89.53%)		
Gestational hypertension				0.207	0.649
Yes	14 (4.17%)	6 (3.66%)	8 (4.65%)		
No	322 (95.83%)	158 (96.34%)	164 (95.35%)		
Total labor duration (h)				0.781	0.377
≥ 7	172 (51.19%)	88 (53.66%)	84 (48.84%)		
< 7	164 (48.81%)	76 (46.34%)	88 (51.16%)		
Episiotomy				0.404	0.525
Yes	93 (27.68%)	48 (29.27%)	45 (26.16%)		
No	243 (72.32%)	116 (70.73%)	127 (73.84%)		
Neonatal birth weight (g)				0.3	0.861
< 3000	67 (19.94%)	32 (19.51%)	35 (20.35%)		
3000-4000	243 (72.32%)	118 (71.95%)	125 (72.67%)		
> 4000	26 (7.74%)	14 (8.54%)	12 (6.98%)		
Neonatal sex				0.409	0.522
Male	174 (51.79%)	82 (50.00%)	92 (53.49%)		
Female	162 (48.21%)	82 (50.00%)	80 (46.51%)		
Neonatal length (cm)	49.82±1.61	49.79±1.56	49.85±1.67	0.373	0.709
Neonatal head circumference (cm)	33.65±1.18	33.68±1.29	33.62±1.06	-0.504	0.615

Note: BMI, Body Mass Index; GDM, Gestational Diabetes Mellitus; PIH, Pregnancy-Induced Hypertension; SD, Standard Deviation.

ing rescue analgesia, the observation group had a significantly later onset of rescue analgesia and a significantly lower total dosage of rescue analgesics (both P < 0.001, **Table 2**).

Inflammatory marker levels

At 6, 24, and 72 hours postpartum, the levels of TNF-α, IL-6, and CRP in the observation



Comparison	t (Obs)	P (Obs)	t (Ctrl)	P (Ctrl)
2h vs 6h	3.33	0.011	−0.27	1.000
6h vs 12h	4.02	<0.001	3.08	0.024
12h vs 24h	3.96	0.001	4.11	<0.001
24h vs 48h	3.64	0.004	4.31	<0.001

Period	t	P	Sig
2h–6h	−2.26	0.024	*
6h–12h	−0.12	0.904	ns
12h–24h	0.59	0.555	ns
24h–48h	1.06	0.289	ns

Figure 1. Dynamic changes in postpartum VAS scores between groups. A. Trends in VAS scores at each postpartum time point between groups. B. Comparison of VAS score changes between consecutive time points. Note: VAS, visual analogue scale; Obs, observation group; Ctrl, control group; Sig, significance; ns, not significant. *P < 0.05, ***P < 0.001.

Table 2. Comparison of rescue analgesia between groups [n (%) or M (Q1, Q3)]

Variable	Control Group (n = 164)	Observation Group (n = 172)	χ^2/t	*P* value
Rescue analgesia rate [n (%)]	35 (21.34%)	18 (10.47%)	7.475	0.006
Time to first rescue (h)	12.45 [9.88, 15.62]	18.65 [13.60, 22.20]	7.839	< 0.001
Total rescue medication (mg)	48.00 [34.00, 56.00]	32.00 [24.00, 40.00]	8.665	< 0.001

Note: M, Median; Q, Quartile.

group were significantly lower than those of the control group (all P < 0.001). In the control group, the levels of TNF-α and CRP initially increased and then decreased from 6 to 24 hours postpartum. Rate of change analysis showed that from 6 to 24 hours postpartum, the decreases in TNF-α, IL-6 and CRP were significantly greater in the observation group (all P < 0.001); however, there were no significant differences between the two groups from 24 to 72 hours (all P > 0.05).

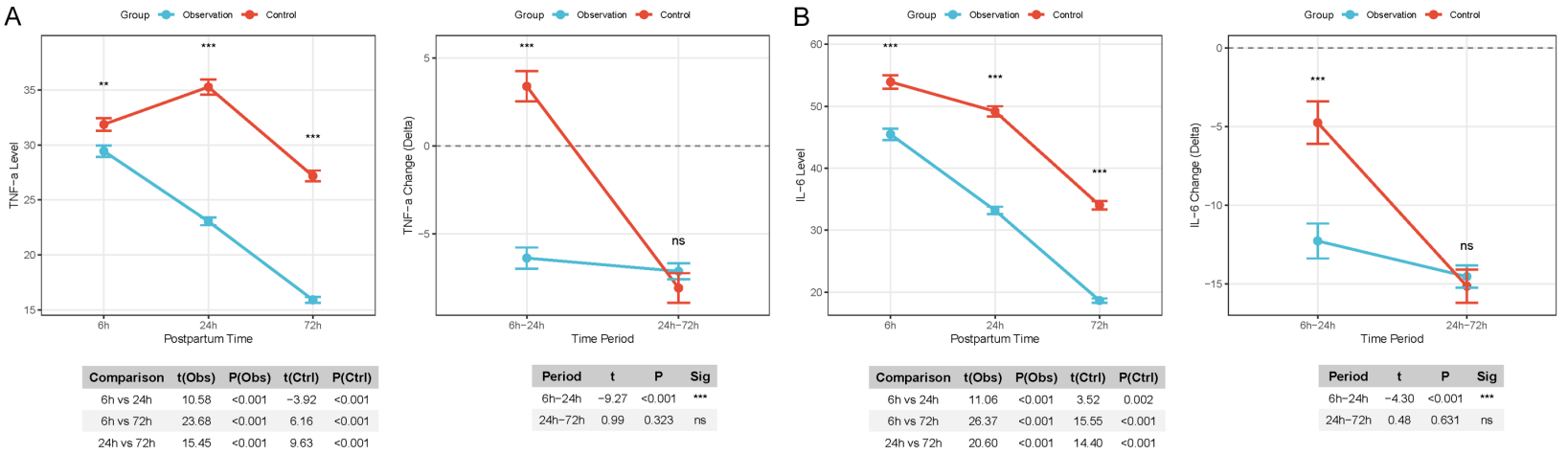
Regarding anti-inflammatory markers, IL-10 levels were significantly higher in the observation group at all postpartum time points (all P < 0.001). Both groups showed progressive increases in IL-10, but the observation group demonstrated significantly greater increases during both the 6-24 hour (P < 0.001) and 24-72 hour (P = 0.001) periods (**Figure 2**).

Breastfeeding behavior

The observation group had a significantly shorter first breastfeeding time, a significantly higher frequency of breastfeeding within 48 hours postpartum, a significantly shorter time to lactation initiation, and a significantly higher breastfeeding self-efficacy score (all P < 0.001). On day 7 postpartum, the observation group had a significantly higher rate of exclusive breastfeeding (P < 0.001, **Table 3**).

Correlation between VAS scores, inflammatory markers, and breastfeeding behavior

Pearson or Spearman correlation analyses showed that the VAS score at 24 hours postpartum was positively correlated with TNF-α (r = 0.472, P < 0.001), IL-6 (r = 0.442, P < 0.001), and CRP (r = 0.442, P < 0.001), and negatively correlated with IL-10 (r = -0.412, P < 0.001). In addition, higher VAS scores were associated with a longer time to first breastfeeding, lower breastfeeding frequency within 48 hours postpartum, and a lower likelihood of exclusive breastfeeding on day 7 postpartum (**Figure 3**).



Comparison	t(Obs)	P(Obs)	t(Ctrl)	P(Ctrl)
6h vs 24h	10.58	<0.001	−3.92	<0.001
6h vs 72h	23.68	<0.001	6.16	<0.001
24h vs 72h	15.45	<0.001	9.63	<0.001

Period	t	P	Sig
6h−24h	−9.27	<0.001	***
24h−72h	0.99	0.323	ns

Comparison	t(Obs)	P(Obs)	t(Ctrl)	P(Ctrl)
6h vs 24h	11.06	<0.001	3.52	0.002
6h vs 72h	26.37	<0.001	15.55	<0.001
24h vs 72h	20.60	<0.001	14.40	<0.001

Period	t	P	Sig
6h−24h	−4.30	<0.001	***
24h−72h	0.48	0.631	ns

Comparison	t(Obs)	P(Obs)	t(Ctrl)	P(Ctrl)
6h vs 24h	−12.31	<0.001	−5.00	<0.001
6h vs 72h	−17.99	<0.001	−10.82	<0.001
24h vs 72h	−8.72	<0.001	−6.01	<0.001

Period	t	P	Sig
6h−24h	5.75	<0.001	***
24h−72h	3.24	0.001	**

Comparison	t(Obs)	P(Obs)	t(Ctrl)	P(Ctrl)
6h vs 24h	12.81	<0.001	−2.42	0.050
6h vs 72h	25.68	<0.001	8.54	<0.001
24h vs 72h	18.09	<0.001	10.69	<0.001

Period	t	P	Sig
6h−24h	−9.31	<0.001	***
24h−72h	1.76	0.080	ns

Figure 2. Dynamic changes in postpartum inflammatory marker levels between groups. A. Trends in TNF-α levels and comparison of changes between consecutive time points. B. Trends in IL-6 levels and comparison of changes between consecutive time points. C. Trends in IL-10 levels and comparison of changes between consecutive time points. D. Trends in CRP levels and comparison of changes between consecutive time points. Note: TNF-α, tumor necrosis factor-alpha; IL-6, interleukin-6; IL-10, interleukin-10; CRP, C-reactive protein; Obs, observation group; Ctrl, control group; Sig, significance; ns, not significant. **P < 0.01, ***P < 0.001.

Table 3. Comparison of breastfeeding behavior between groups [n (%) or M (Q1, Q3)]

Variable	Control Group (n = 164)	Observation Group (n = 172)	t/χ^2	*P* value
Time to first breastfeeding (min)	52.00 [39.75, 63.00]	38.00 [30.00, 45.00]	7.692	< 0.001
Breastfeeding frequency within 48 h (times)	8.00 [7.00, 9.25]	10.00 [9.00, 12.00]	8.427	< 0.001
Exclusive breastfeeding rate at day 7 [n (%)]	112 (68.29%)	145 (84.30%)	11.965	< 0.001
Lactogenesis onset time (h)	37.60 [30.58, 45.30]	28.30 [23.87, 32.35]	8.343	< 0.001
Breastfeeding self-efficacy score (points)	47.00 [40.00, 52.00]	53.00 [48.00, 58.00]	6.989	< 0.001

Note: M, Median; Q, Quartile.

Correlation between inflammatory markers and breastfeeding behavior

Correlation analysis showed that higher levels of pro-inflammatory markers were associated with delayed initiation of breastfeeding and lower breastfeeding frequency within 48 hours postpartum, whereas higher IL-10 levels were associated with earlier first breastfeeding. However, inflammatory markers were not directly associated with exclusive breastfeeding on day 7 postpartum. These findings suggest that inflammatory responses may influence longer-term breastfeeding outcomes primarily through their effects on early breastfeeding behaviors (**Figure 4**).

Multivariate logistic regression analysis of exclusive breastfeeding on day 7 postpartum

Using the exclusive breastfeeding rate on day 7 postpartum as the dependent variable, a logistic regression analysis was performed, incorporating potential factors that might influence breastfeeding outcomes. Multicollinearity tests revealed severe collinearity between the analgesia regimen and the 24-hour postpartum VAS score. After excluding the 24-hour VAS score, the variance inflation factor for all variables was less than 5, allowing them to be included in the regression model.

Univariate analysis showed that analgesia regimen ($P < 0.001$), age ($P < 0.001$), BMI at delivery ($P < 0.001$), parity ($P = 0.005$), episiotomy ($P = 0.042$), and rescue analgesia rate ($P = 0.023$) were significantly associated with the rate of exclusive breastfeeding on day 7 postpartum; while inflammatory markers TNF-α ($P = 0.756$), IL-6 ($P = 0.336$), IL-10 ($P = 0.408$), and CRP ($P = 0.176$) showed no significant association.

Variables with $P < 0.1$ in the univariate analysis were included in the multivariate logistic regression model. The results showed that, after adjusting for confounding factors, analgesia regimen ($P = 0.001$), age ($P < 0.001$), and BMI at delivery ($P < 0.001$) remained independent factors influencing the rate of exclusive breastfeeding on day 7 postpartum; parity ($P = 0.084$), episiotomy ($P = 0.100$), and rescue analgesia rate ($P = 0.320$) were no longer significant (**Tables 4** and **5**).

Subgroup analysis of analgesic effects and breastfeeding outcomes

To explore the consistency of intervention effects across different populations, subgroup analyses were conducted based on parity and BMI.

In parity subgroups, VAS scores at 24 hours postpartum were significantly lower in the observation group regardless of parity status (both $P < 0.001$). In the multiparous subgroup, the observation group showed significantly better rates of emergency analgesia ($P < 0.001$) and exclusive breastfeeding ($P = 0.004$); while in the primiparous subgroup, there were no significant differences in emergency analgesia rates ($P = 1.000$) and exclusive breastfeeding rates ($P = 0.156$) between the two groups. Interaction analysis showed no significant interaction between the intervention and parity on VAS scores ($P = 0.746$), suggesting that the intervention's effect was consistent across different parity groups.

In different BMI subgroups, within each BMI stratum (BMI < 24: $P < 0.001$; BMI 24-28: $P < 0.001$; BMI > 28: $P = 0.002$), the observation group consistently had significantly lower VAS scores at 24 hours postpartum. There were no significant differences in emergency analgesia

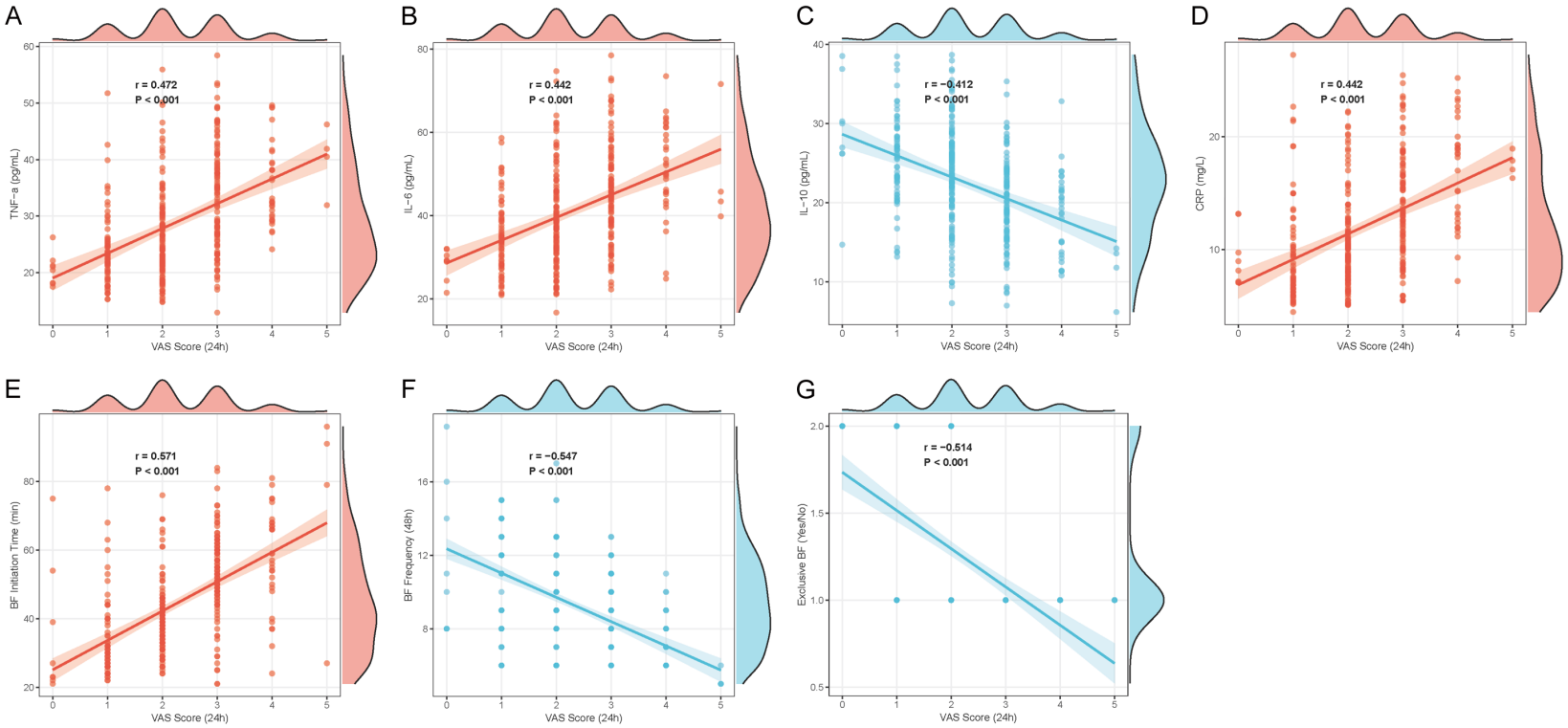


Figure 3. Scatter plots of correlations between 24-hour postpartum vas scores and inflammatory markers and breastfeeding behavior. (A) Correlation between VAS and TNF-α. (B) Correlation between VAS and IL-6. (C) Correlation between VAS and IL-10. (D) Correlation between VAS and CRP. (E) Correlation between VAS and time to first breastfeeding. (F) Correlation between VAS and breastfeeding frequency within 48 hours. (G) Correlation between VAS and exclusive breastfeeding at day 7. Note: VAS, visual analogue scale; TNF-α, tumor necrosis factor-alpha; IL-6, interleukin-6; IL-10, interleukin-10; CRP, C-reactive protein; BF, breastfeeding. (A-F) present Pearson correlation coefficients (r); (G) presents Spearman rank correlation coefficient (ρ), as the exclusive breastfeeding rate at day 7 did not satisfy the normality criterion. Red indicates significant positive correlation; blue indicates significant negative correlation.

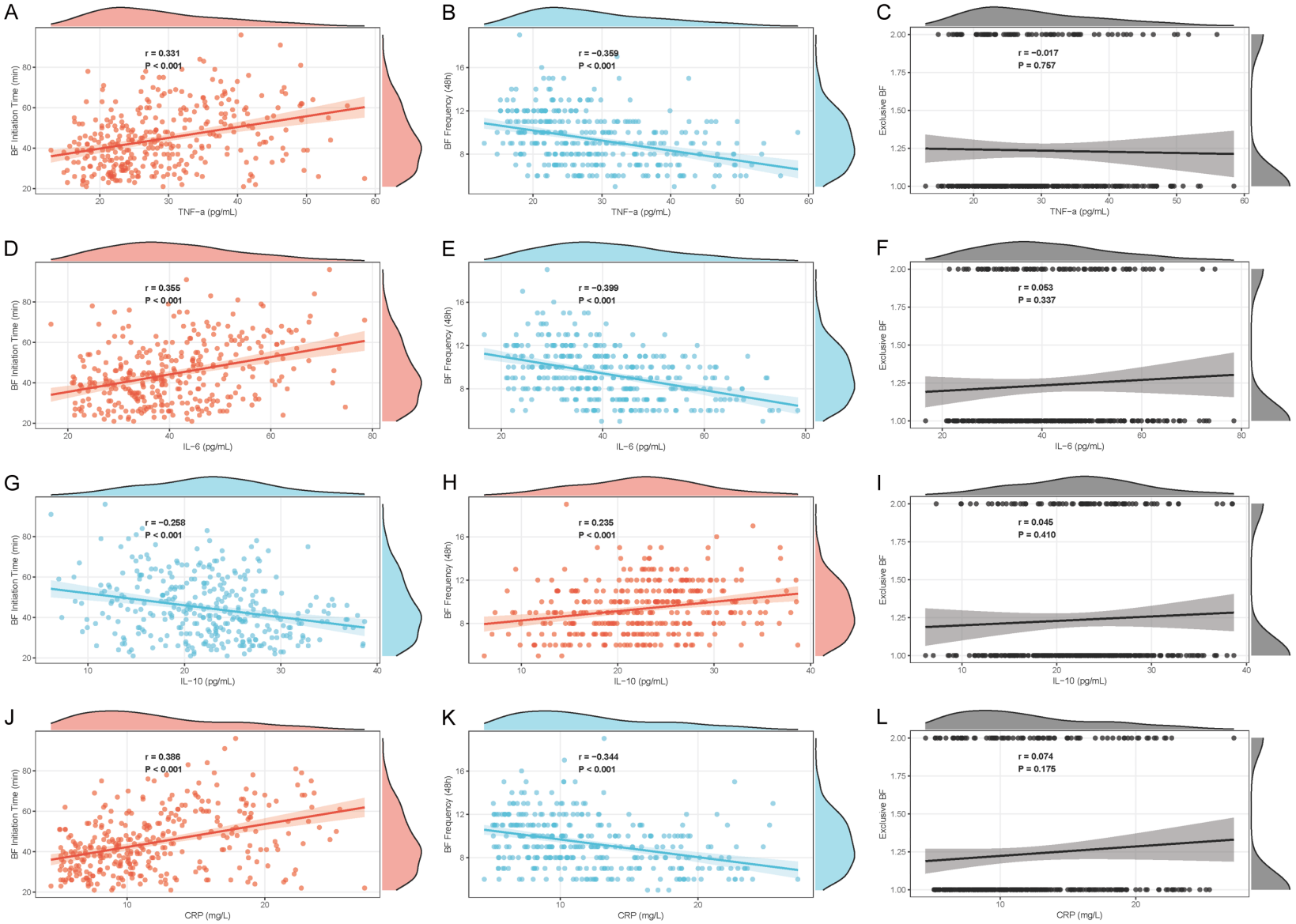


Figure 4. Scatter plots of correlations between inflammatory markers and breastfeeding behavior. (A-C) Correlations between TNF-α and breastfeeding behavior. (D-F) Correlations between IL-6 and breastfeeding behavior. (G-I) Correlations between IL-10 and breastfeeding behavior. (J-L) Correlations between CRP and breast-

feeding behavior. Note: TNF-α, tumor necrosis factor-alpha; IL-6, interleukin-6; IL-10, interleukin-10; CRP, C-reactive protein; BF, breastfeeding; ns, not significant. (A, B, D, E, G, H, and J, K) present Pearson correlation coefficients (r); (C, F, I, and L) present Spearman rank correlation coefficients (ρ), as the exclusive breastfeeding rate at day 7 did not satisfy the normality criterion. Red indicates significant positive correlation; blue indicates significant negative correlation; black indicates non-significant correlation.

Table 4. Variable assignment and multicollinearity testing for logistic regression analysis

Variable	Assignment	Adjusted VIF
Analgesia regimen	Observation group = 1, Control group = 0	3.947
Age	≤ 30 years = 1, 31-35 years = 2, > 35 years = 3	1.122
BMI at delivery	< 24 kg/m^2 = 1, 24-28 kg/m^2 = 2, > 28 kg/m^2 = 3	1.116
Parity	Primiparous = 1, Multiparous = 0	1.128
Gestational age at delivery	37-38 weeks = 1, 39-40 weeks = 2, > 40 weeks = 3	1.053
Gestational diabetes mellitus	Yes = 1, No = 0	1.078
Gestational hypertension	Yes = 1, No = 0	1.047
Total labor duration	≥ 7 = 1, < 7 = 0	1.084
Episiotomy	Yes = 1, No = 0	1.137
Neonatal birth weight	< 3000 = 1, 3000-4000 = 2, > 4000 = 3	1.072
Rescue analgesia rate	Rescue = 1, No rescue = 0	1.103
Neonatal jaundice	Yes = 1, No = 0	1.131
24 h VAS	Raw data	-
24 h TNF-α	Raw data	1.849
24 h IL-6	Raw data	1.802
24 h IL-10	Raw data	1.582
24 h CRP	Raw data	1.764
Exclusive breastfeeding rate at day 7	Success = 1, Failure = 0	-

Note: VIF, Variance Inflation Factor; VAS, Visual Analogue Scale; TNF-α, Tumor Necrosis Factor-α; IL-6, Interleukin-6; IL-10, Interleukin-10; CRP, C-Reactive Protein.

rates among the BMI subgroups (all P > 0.05). Regarding exclusive breastfeeding rates, the observation group showed a significantly higher rate only in the BMI 24-28 subgroup (P = 0.006), while there was no significant difference between the BMI < 24 (P = 0.259) and BMI > 28 (P = 0.169) subgroups. Interaction tests showed no significant interaction between the intervention and BMI on VAS scores (P = 0.313), suggesting that the intervention's effect is consistent across different BMI populations (**Figures 5** and **6**).

Adverse events

The intergroup comparison of adverse events showed no significant differences in the incidence of nausea and vomiting (P = 0.745), dizziness (P = 0.649), pruritus (P = 1.000), urinary retention (P = 0.821), hypotension (P = 1.000), lower limb numbness (P = 1.000), surgical site infection (P = 0.678), and postpartum hemorrhage (P = 0.718). The overall incidence of adverse events also showed no significant difference (P = 0.677). These results indicate that the addition of dexamethasone to ropivacaine does not increase the risk of adverse events in parturient women (see **Table 6**).

Neonatal outcomes

The intergroup comparison of neonatal outcomes showed no significant differences in 1-minute Apgar scores (P = 1.000), 5-minute Apgar scores (P = 0.153), neonatal jaundice rate (P = 0.623), neonatal hypoglycemia rate (P = 1.000), neonatal infection rate (P = 0.949), neonatal hospital stay duration (P = 0.125), or neonatal intensive care unit admission rate (P = 0.942, **Table 7**).

Interaction analysis of intervention effects

To further evaluate the robustness of the intervention effect across different populations, an interaction analysis was conducted based on

Table 5. Logistic regression analysis of factors associated with exclusive breastfeeding rate at postpartum day 7

Variable	Univariable OR	Univariable P	Univariable 95% CI	Multivariable OR	Multivariable P	Multivariable 95% CI
Analgesia regimen	0.401	< 0.001	0.237-0.679	0.387	0.001	0.214-0.686
Age	0.402	< 0.001	0.278-0.583	0.375	< 0.001	0.248-0.558
BMI at delivery	0.466	< 0.001	0.319-0.680	0.44	< 0.001	0.284-0.666
Parity	0.482	0.005	0.289-0.804	0.6	0.084	0.336-1.072
Gestational age at delivery	1.191	0.491	0.724-1.958			
Gestational diabetes mellitus	1.488	0.398	0.593-3.734			
Gestational hypertension	1.886	0.413	0.413-8.611			
Total labor duration	0.964	0.886	0.582-1.596			
Episiotomy	0.572	0.042	0.334-0.979	0.599	0.1	0.326-1.109
Neonatal birth weight	1.09	0.732	0.666-1.782			
Rescue analgesia rate	0.481	0.023	0.257-0.904	0.694	0.32	0.341-1.448
Neonatal jaundice	1.84	0.158	0.788-4.294			
24 h TNF-α	1.004	0.756	0.977-1.032			
24 h IL-6	0.99	0.336	0.970-1.010			
24 h IL-10	0.984	0.408	0.947-1.023			
24 h CRP	0.967	0.176	0.921-1.015			

Note: VAS, Visual Analogue Scale; TNF-α, Tumor Necrosis Factor-α; IL-6, Interleukin-6; IL-10, Interleukin-10; CRP, C-Reactive Protein; OR, Odds Ratio; CI, Confidence Interval. Variables with P < 0.1 in univariate analysis were included in multivariate analysis.

parity, BMI, and gestational diabetes mellitus. Forest plot analysis showed that, overall, the rate of exclusive breastfeeding on day 7 postpartum was significantly higher in the observation group (OR = 2.49, 95% CI: 1.48-4.27, P < 0.001).

Stratified analysis revealed that the intervention was statistically significant in multiparous women (OR = 3.13, 95% CI: 1.51-6.79, P = 0.003) and the BMI 24-28 subgroup (OR = 2.73, 95% CI: 1.39-5.55, P = 0.004); however, the intervention was not significant in primiparous women (P = 0.109), women with BMI > 28 (P = 0.106), and women with gestational diabetes mellitus (P = 0.300). However, the OR values in all subgroups were greater than 1, suggesting that the intervention tended to benefit the observation group in all subgroups.

Rate difference analysis indicated that the exclusive breastfeeding rate in the observation group was on average 16.01% higher than that in the control group (95% CI: 7.05%-24.97%), with the largest increase in the BMI 24-28 subgroup, reaching 20.48% (95% CI: 6.93%-34.02%).

Interaction test showed no significant interaction effect between the intervention and parity (P = 0.350), BMI (P = 0.970), or gestational diabetes mellitus (P = 0.940), suggesting that the intervention effect was consistent across different populations (**Figure 7**).

Discussion

This study found that in multiparous women, epidural analgesia using ropivacaine combined with dexamethasone was associated with lower VAS scores at all postpartum time points, fewer needs for rescue analgesia, improved inflammatory marker profiles, and better early breastfeeding outcomes. The observation group had a higher rate of exclusive breastfeeding on day 7 postpartum without an increase in maternal and infant adverse events.

The observation group showed significantly lower VAS scores at all time points, particularly between 2 and 6 hours postpartum, suggesting that dexamethasone as an adjunct to regional anesthesia may help alleviate pain. Previous meta-analyses have consistently confirmed that perineurial administration of dexamethasone prolongs sensory blockade time and reduces postoperative pain scores [11, 16]. The mechanisms may include: inducing local vasoconstriction by activating glucocorticoid receptors, reducing systemic absorption of local anesthetics [17]; inhibiting nociceptive C-fiber transmission by acting on potassium

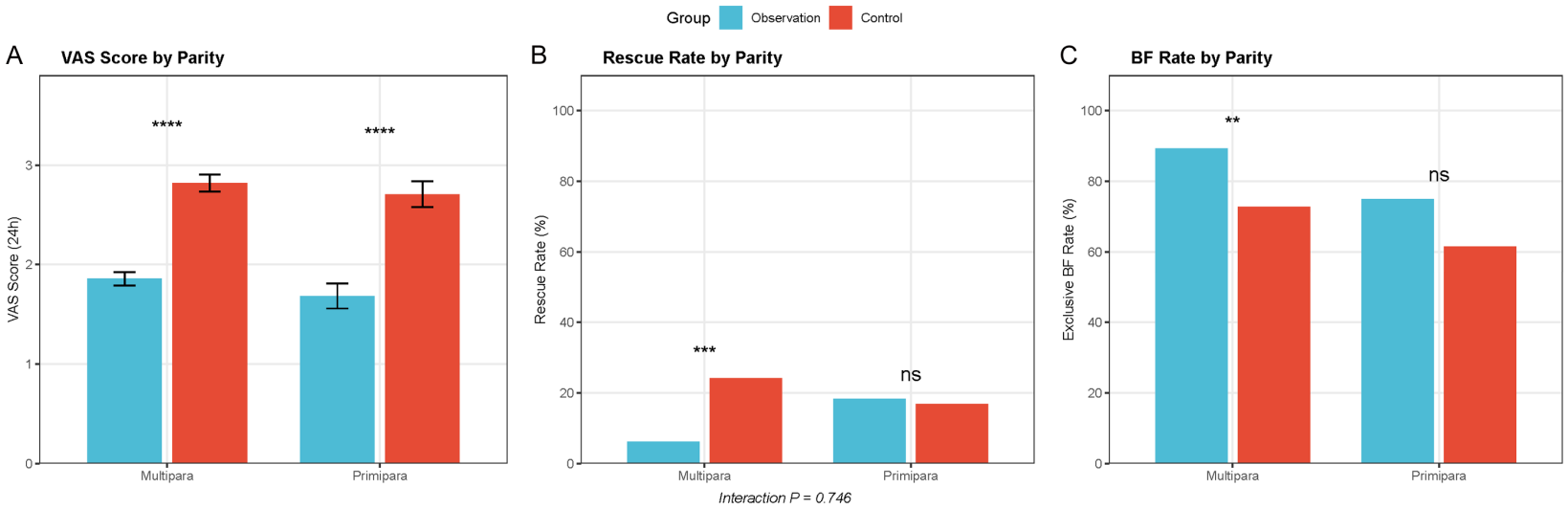


Figure 5. Comparison of analgesia efficacy and breastfeeding outcomes between groups based on parity subgroups. A. Comparison of 24-hour postpartum VAS scores between groups based on parity subgroups. B. Comparison of rescue analgesia rates between groups based on parity subgroups. C. Comparison of exclusive breastfeeding rates between groups based on parity subgroups. Note: VAS, visual analogue scale; BMI, body mass index; BF, breastfeeding; ns, not significant. **P < 0.01, ***P < 0.001, ****P < 0.0001.

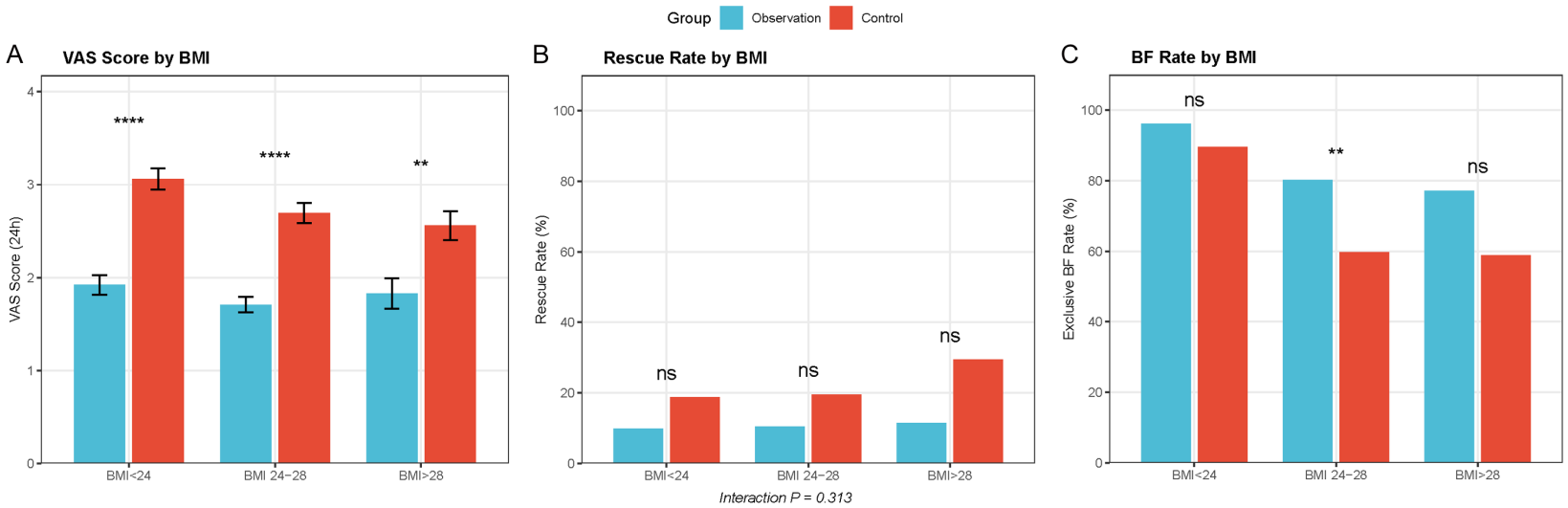


Figure 6. Comparison of analgesic effects and breastfeeding outcomes between groups based on BMI subgroups. A. Comparison of 24-hour postpartum VAS scores between groups based on BMI subgroups. B. Comparison of rescue analgesia rates between groups based on BMI subgroups. C. Comparison of exclusive breastfeeding rates between groups based on BMI subgroups. Note: VAS, visual analogue scale; BMI, body mass index; BF, breastfeeding; ns, not significant. **P < 0.01, ****P < 0.0001.

Table 6. Comparison of adverse events between groups [n (%)]

Adverse Event	Control Group (n = 164)	Observation Group (n = 172)	χ^2	*P* value
Nausea/vomiting	10 (6.10%)	12 (6.98%)	0.106	0.745
Dizziness	6 (3.66%)	8 (4.65%)	0.207	0.649
Pruritus	4 (2.44%)	5 (2.91%)	-	1
Urinary retention	5 (3.05%)	6 (3.49%)	0.051	0.821
Hypotension	3 (1.83%)	4 (2.33%)	-	1
Lower limb numbness	2 (1.22%)	3 (1.74%)	-	1
Surgical site infection	3 (1.83%)	2 (1.16%)	-	0.678
Postpartum hemorrhage	4 (2.44%)	3 (1.74%)	-	0.718
Overall incidence	24 (14.63%)	28 (16.28%)	0.174	0.677

Note: Fisher's exact test was used when expected frequency was less than 5.

Table 7. Comparison of neonatal outcomes between groups [n (%) or M (Q1, Q3)]

Variable	Control Group (n = 164)	Observation Group (n = 172)	t/χ^2	*P* value
1-min Apgar score (points)	10.00 [9.00, 10.00]	10.00 [9.00, 10.00]	< 0.001	1
5-min Apgar score (points)	10.00 [10.00, 10.00]	10.00 [10.00, 10.00]	1.428	0.153
Neonatal jaundice [n (%)]	24 (14.63%)	22 (12.79%)	0.241	0.623
Neonatal hypoglycemia [n (%)]	4 (2.44%)	5 (2.91%)	< 0.001	1
Neonatal infection [n (%)]	4 (2.44%)	3 (1.74%)	0.004	0.949
Neonatal hospital stay (d)	3.00 [3.00, 4.00]	3.00 [3.00, 4.00]	1.533	0.125
NICU transfer rate [n (%)]	5 (3.05%)	4 (2.33%)	0.005	0.942

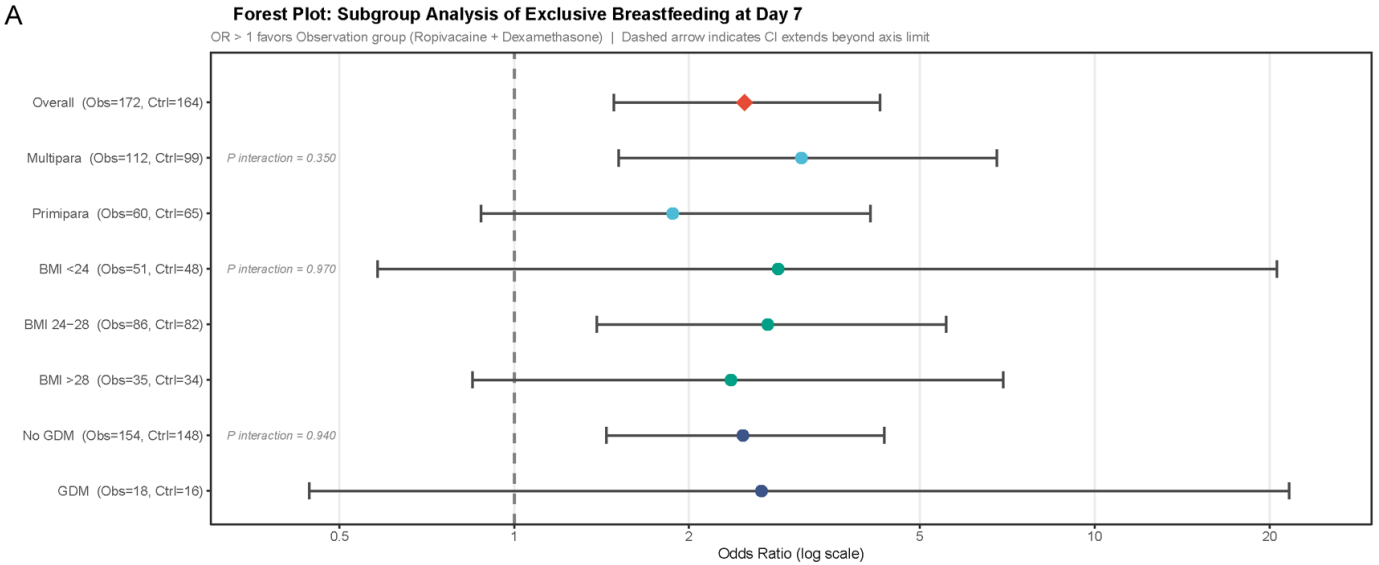
Note: NICU, Neonatal Intensive Care Unit; M, Median; Q, Quartile. Fisher's exact test was used when expected frequency was less than 5.

channels [17]; and reducing surgical trauma-induced inflammatory responses through systemic anti-inflammatory effects [20]. The more significant improvement in early postpartum pain in the observation group may reflect that these mechanisms were at work from the onset of analgesia.

This study used a dexamethasone dose of 10 mg. Pehora et al. [11] reported no significant dose-response difference in the range of 4-10 mg, suggesting a plateau in the dose-response relationship. A recent network meta-analysis confirmed that 4 mg of dexamethasone administered peripherally or 8 mg intravenously may be the appropriate clinical dose [18]. It is noteworthy that the existing evidence comes mainly from peripheral nerve block studies. Although the effect of glucocorticoids in prolonging local anesthetic analgesia is transferable in different regional anesthesia scenarios, the optimal dose and safety boundaries of epidural administration still need to be verified by prospective randomized controlled trials.

Labor itself represents an inflammatory stress state. Studies have shown that maternal serum levels of pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α are significantly elevated during labor and participate in uterine contractions and labor initiation [19]. This inflammatory response persists in the early postpartum period and contributes to uterine involution, but excessive inflammation may also lead to pain sensitization and tissue injury [20]. In the observation group, serum TNF-α, IL-6, and CRP levels were significantly lower, whereas IL-10 levels were significantly higher across postpartum time points, suggesting that dexamethasone may effectively regulate the direction and

A


Forest Plot: Subgroup Analysis of Exclusive Breastfeeding at Day 7
OR > 1 favors Observation group (Ropivacaine + Dexamethasone) | Dashed arrow indicates CI extends beyond axis limit
Overall (Obs=172, Ctrl=164)
Multipara (Obs=112, Ctrl=99)
Primipara (Obs=60, Ctrl=65)
BMI <24 (Obs=51, Ctrl=48)
BMI 24–28 (Obs=86, Ctrl=82)
BMI >28 (Obs=35, Ctrl=34)
No GDM (Obs=154, Ctrl=148)
GDM (Obs=18, Ctrl=16)
P interaction = 0.350
P interaction = 0.970
P interaction = 0.940
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1
2
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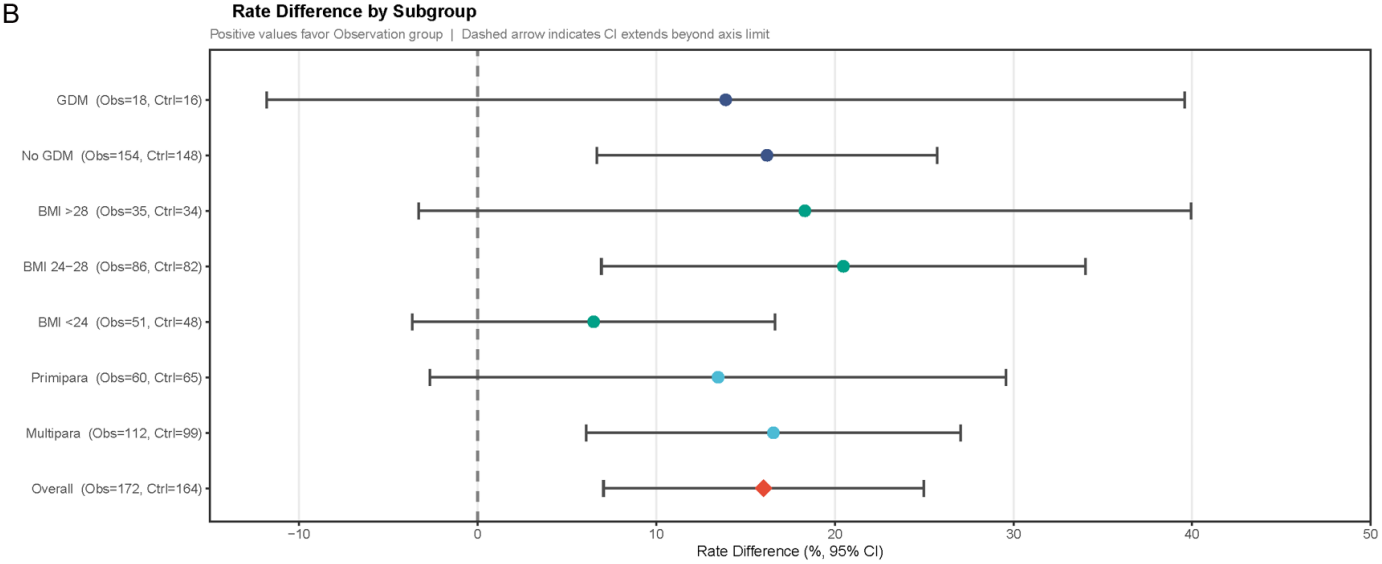
10
20
Odds Ratio (log scale)

B


Rate Difference by Subgroup
Positive values favor Observation group | Dashed arrow indicates CI extends beyond axis limit
GDM (Obs=18, Ctrl=16)
No GDM (Obs=154, Ctrl=148)
BMI >28 (Obs=35, Ctrl=34)
BMI 24–28 (Obs=86, Ctrl=82)
BMI <24 (Obs=51, Ctrl=48)
Primipara (Obs=60, Ctrl=65)
Multipara (Obs=112, Ctrl=99)
Overall (Obs=172, Ctrl=164)
−10
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Rate Difference (%, 95% CI)

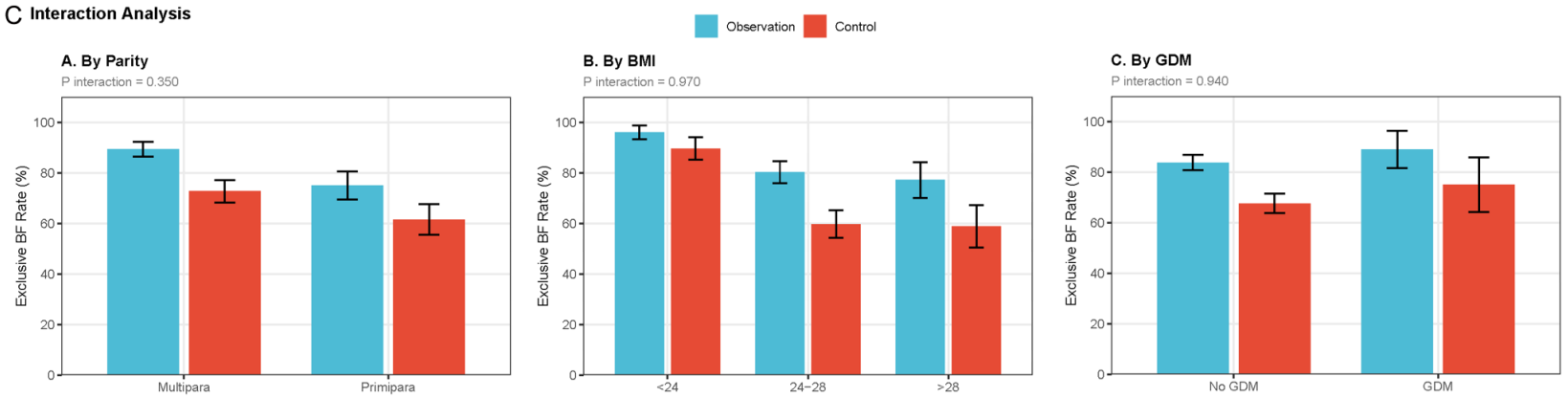

Figure 7. Interaction Analysis of Factors Associated with Exclusive Breastfeeding Rate on Day 7 Postpartum. (A) Forest plot of intervention effects across subgroups (OR and 95% CI). (B) Rate differences in exclusive breastfeeding (observation group minus control group) with 95% CI across subgroups. (C) Comparison of exclusive breastfeeding rates between groups stratified by parity, BMI, and gestational diabetes mellitus. Note: OR, odds ratio; CI, confidence interval; BMI, body mass index; GDM, gestational diabetes mellitus; BF, breastfeeding. Dashed line indicates OR = 1 (A) or rate difference = 0 (B).

intensity of postpartum inflammatory responses [21].

Dexamethasone exerts its anti-inflammatory effects through multiple mechanisms, inducing IκBα protein synthesis, causing active NF-κB to remain in the cytoplasm to form an inactive complex, thereby preventing the transcription of pro-inflammatory genes [22]; directly binding to transcription factors AP-1 or NF-κB through glucocorticoid receptors, reducing their DNA binding and transcriptional activation capacity [23]; and inhibiting phospholipase A2 activity, reducing arachidonic acid release and prostaglandin production [24].

The VAS score at 24 hours postpartum was positively correlated with TNF-α, IL-6, and CRP, and negatively correlated with IL-10, suggesting that inflammatory responses are involved in postpartum pain. Pro-inflammatory mediators may exacerbate pain by activating peripheral nociceptors, lowering the pain threshold, and promoting central sensitization, whereas IL-10 may exert a protective effect. Dexamethasone may therefore improve postpartum pain through both prolongation of local anesthetic effects and modulation of the inflammatory response.

The observation group achieved a shorter time to first breastfeeding, a higher feeding frequency within 48 hours postpartum, an earlier initiation of lactation, and a higher rate of exclusive breastfeeding on day 7 (84.30% vs. 68.29%). Correlation analysis showed that VAS scores were positively correlated with the time to first breastfeeding and negatively correlated with feeding frequency and exclusive breastfeeding rate. Postpartum pain affects breastfeeding through multiple pathways: pain-induced stress inhibits hypothalamic-pituitary axis function, reduces oxytocin and prolactin secretion, and affects lactation and milk ejection reflexes [25]; severe pain affects maternal mobility, limiting early mother-infant interaction and latching [25]; and negative emotions associated with pain reduce confidence and persistence in breastfeeding [26]. Furthermore, inflammatory markers were mainly associated with immediate breastfeeding indicators (time of first feeding, frequency of early feeding), but not directly with the rate of exclusive breastfeeding on day 7. Therefore, their effect on long-term breastfeeding outcomes may be indirectly achieved through the establishment of early feeding behavior. This mediating hypothesis needs to be verified in prospective studies through mediation analysis or structural equation modeling. The World Health Organization emphasizes that early contact, early suckling, and early initiation are crucial for successful breastfeeding in infants [27]. Moore et al., authors of a Cochrane systematic review including 46 randomized controlled trials, demonstrated that early skin-to-skin contact significantly improves breastfeeding rates at 1-4 months postpartum [28]. Similarly, Safari et al. found that skin-to-skin contact immediately after birth supports successful first breastfeeding and a longer duration of breastfeeding [29]. Based on these findings, the observation group in our study, having experienced effective analgesia and anti-inflammatory effects, may have benefited from better conditions for early breastfeeding, which could indirectly contribute to longer-term breastfeeding.

Multivariate logistic regression analysis with the control group as a reference and failure to achieve exclusive breastfeeding as the outcome event showed that the analgesia regimen was independently associated with the rate of exclusive breastfeeding on day 7 (OR = 0.387, 95% CI: 0.214-0.686), suggesting that the risk of exclusive breastfeeding failure in the observation group was 61.3% lower than that of the control group. After adjusting for confounding factors, inflammatory markers lost statistical significance, further supporting the possibility that they may indirectly affect breastfeeding outcomes through the establishment of early feeding behaviors. Subgroup analysis showed that VAS scores were significantly lower in all subgroups of the observation group. The intervention effect on breastfeeding rate was statistically significant in multiparous women, but not in primiparous women, although the effect direction was consistent. This may reflect the lack of statistical power due to insufficient sample size, or suggest that the reasons for breastfeeding failure in primiparous women are more related to other factors (such as lack of breastfeeding experience and technical knowledge). BMI subgroup analysis showed that the analgesic effect was generally better in the observation group, but the improvement in breastfeeding rate was seen mainly in the BMI 24-28 kg/m^2 subgroup. Breastfeeding in women with

higher BMI (> 28 kg/m^2) is affected by a variety of factors, including breast tissue morphology affecting latching, and delayed lactation initiation [30], and simply improving analgesia may not be enough to offset these adverse factors. Forest plot analysis showed that the overall exclusive breastfeeding rate in the observation group was about 16 percentage points higher than that in the control group, with consistent subgroup effects (all OR > 1), and no significant interaction between the intervention and parity or BMI was detected, suggesting that the observed associations were consistent across different populations.

There was no significant difference in the incidence of maternal adverse events between the two groups. There were also no significant differences in neonatal outcomes (Apgar score, jaundice, hypoglycemia, infection rate, NICU admission rate). No safety issues were observed in this study (336 cases) and during the 7-day postpartum follow-up period, but this does not equate to complete safety and requires further validation in larger samples and longer follow-ups. Pehora et al.'s Cochrane systematic review [17] did not find that perineurial use of dexamethasone increased the risk of nerve damage, which supported our results. The systemic effect of a single low-dose dexamethasone is limited, and epidural administration may further reduce systemic exposure.

This study has several limitations. First, its retrospective cohort design could not eliminate selection bias or unmeasured confounding, thus precluding causal inference. Second, the study excluded patients with poorly controlled gestational diabetes mellitus; therefore, dexamethasone should be used with greater caution in patients with abnormal glucose metabolism. Third, although multiparous women were the primary focus, the retrospective data did not permit detailed stratification within this group (e.g., by parity or history of cesarean section). Fourth, obstetric practices at the study hospital may differ from those in other regions, affecting the generalizability of the results. Fifth, breastfeeding data beyond 7 days postpartum (e.g., at 6 weeks or 3 months) were unavailable, and the absence of hormone measurements (e.g., oxytocin, prolactin) limited mechanistic interpretation. Additionally, subjective indicators (e.g., VAS scores, lactation initiation time) may introduce bias, and the proposed mediating role of inflammatory markers on breastfeeding behavior requires validation in prospective studies using appropriate mediation analysis techniques.

Conclusion

This retrospective cohort study demonstrated that epidural analgesia with ropivacaine combined with dexamethasone was associated with reduced postpartum uterine cramping pain, improved inflammatory profiles, including reductions in pro-inflammatory markers and CRP and an increase in IL-10, optimized early breastfeeding behavior, and an increased rate of exclusive breastfeeding on day 7 postpartum, without increasing maternal or infant adverse events. Dexamethasone, as an adjunct to epidural analgesia, may create favorable conditions for postpartum recovery and breastfeeding through a dual effect of enhanced analgesia and anti-inflammatory modulation.

Disclosure of conflict of interest

None.

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