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Original Research Article

Continuous Epidural Bupivacaine with Two Different Doses of Dexmedetomidine for Postoperative Analgesia in Infraumbilical Surgeries: A Randomized Double-Blind Comparative Study

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Abstract

Background: Effective postoperative analgesia is essential for reducing pain-related physiological stress and facilitating recovery. Dexmedetomidine, an α2-adrenergic agonist, has been used as an epidural adjuvant to enhance local-anesthetic analgesia, although its optimal dose remains uncertain.

Objective: To compare the postoperative analgesic efficacy and hemodynamic effects of continuous epidural infusion of 0.125% bupivacaine with two different concentrations of dexmedetomidine in patients undergoing infraumbilical surgeries.

Methods: This prospective, randomized, double-blind, hospital-based study included 96 adult patients undergoing surgical procedures below the level of the umbilicus. Participants were equally allocated to three groups (n=32 each): a control group receiving 0.125% bupivacaine alone, Group 1 receiving 0.125% bupivacaine with dexmedetomidine 1 µg/mL, and Group 2 receiving 0.125% bupivacaine with dexmedetomidine 2 µg/mL. Postoperative pain using the Visual Analogue Scale (VAS), systolic and diastolic blood pressure, mean arterial pressure, pulse rate, and peripheral oxygen saturation (SpO_2) were assessed from baseline to 24 hours. Data were analysed using IBM SPSS Statistics version 27.0.

Results: Mean age and distributions of sex, ASA physical status, and type of surgery were comparable among the groups. VAS scores were significantly different at all assessed time points ($p<0.001$). At 24 hours, mean VAS scores were 3.16±0.72, 1.91±0.64, and 1.66±0.48 in the control, Group 1, and Group 2, respectively. Group 2 showed the greatest reductions in systolic and diastolic blood pressure and pulse rate; at 24 hours, systolic blood pressure was 89.47±8.21 mmHg and pulse rate was 60.84±5.35 beats/min. SpO_2 remained approximately 98% in all groups throughout follow-up.

Conclusion: Epidural dexmedetomidine enhanced the analgesic effect of 0.125% bupivacaine in a dose-related manner. Although 2 µg/mL provided the greatest analgesia, it produced more pronounced hemodynamic reductions, whereas 1 µg/mL appeared to provide a more balanced analgesic and hemodynamic profile.

Keywords: Postoperative analgesia, Epidural analgesia, Bupivacaine, Dexmedetomidine, Visual Analogue Scale.

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Introduction

Acute postoperative pain remains clinically important despite advances in multimodal analgesia. Gan et al. reported that approximately 86% of 300 surveyed surgical patients experienced postoperative pain; among those with pain, 75% described it as moderate to extreme during the immediate postoperative period and 74% continued to report moderate-to-extreme pain after discharge. [1] Persistent nociceptive input after surgery can increase sympathetic activity, impede mobilization and rehabilitation, and increase analgesic requirements, emphasizing the need for effective

regional analgesic strategies. Epidural analgesia enables segmental delivery of local anesthetic close to spinal nerve roots and is particularly useful for prolonged analgesia after abdominal, pelvic, and lower-limb procedures. Bupivacaine is a long-acting amide local anesthetic that reversibly blocks voltage-gated sodium channels, thereby preventing neuronal depolarization and propagation of nociceptive impulses. Its prolonged sensory action favors postoperative epidural use, although greater local-anesthetic exposure may increase motor blockade, hypotension, and, if systemic toxicity occurs, cardiovascular complications. [2]

Dexmedetomidine is a highly selective α2-adrenoceptor agonist with an α2:α1 selectivity ratio of approximately 1620:1. Weerink et al. described its sedative, anxiolytic, sympatholytic, and analgesic-sparing actions with minimal respiratory depression, while also identifying bradycardia and hypotension as clinically relevant hemodynamic effects. [3] These pharmacological characteristics make dexmedetomidine attractive as a neuraxial adjuvant because it may enhance analgesia without the respiratory-depressant profile associated with opioid supplementation, although dose selection remains important. Clinical studies demonstrate potentiation of neuraxial local anesthetics by dexmedetomidine. Bajwa et al. found that epidural dexmedetomidine produced faster progression of sensory blockade than clonidine, with the time to maximum sensory level averaging 13.14 ± 3.96 versus 15.80 ± 4.86 minutes. [4] Kaur et al. reported that adding epidural dexmedetomidine to ropivacaine prolonged sensory blockade from 375.20 ± 15.97 to 535.18 ± 19.85 minutes. [5] More recently, Sabu et al. found that dexmedetomidine 0.5 µg/kg added to low-concentration epidural bupivacaine significantly reduced postoperative VAS scores and rescue-analgesic requirements (p=0.003). [6]

These findings support dexmedetomidine as an effective epidural adjuvant, but the balance between enhanced analgesia and α2-mediated cardiovascular effects remains dose dependent. Against this background, the objective of the present study was to compare the efficacy of postoperative pain relief and the associated hemodynamic effects of continuous epidural infusion of 0.125% bupivacaine with two different doses of dexmedetomidine (1 µg/mL and 2 µg/mL) as an adjuvant in patients undergoing surgeries below the level of the umbilicus.

Materials and Methods

This was a single center, hospital-based, randomized, double blinded, prospective study conducted in the Department of Anesthesia, ACS Medical College and Hospital, Chennai, Tamil Nadu, India over a period of two years. The study received approval from the Institutional Human Ethics Committee (IHEC) (reference number 817/2023/IEC/ACSMCH dated 10/04/2023). Each participant was provided with a Participant Information Sheet (PIS) translated into their local language. The information was also explained verbally to ensure clear understanding and voluntary agreement. Written informed consent was obtained prior to enrolling participants in the study. The study included conscious and cooperative patients aged >18 years of either sex who were classified as American Society of Anesthesiologists (ASA) physical status I–III and were undergoing surgical procedures below the level of the umbilicus. Patients with a history of hypersensitivity to any study drug, hematological or haemostatic disorders, chronic pain syndrome, morbid obesity with a body mass index >40 kg/m², psychiatric illness, or inability to cooperate were excluded.

The sample size was estimated based on the study by Kurhekar et al.,(7) which evaluated different doses of dexmedetomidine for continuous epidural postoperative analgesia. Assuming an increase in the proportion of patients achieving complete analgesia from 76% to 99%, a two-sided significance level of 5% (α = 0.05), 80% statistical power (β = 0.20), and equal allocation, the minimum required sample size was approximately 32 patients per group. Accordingly, for the three study groups, the final sample size was 96 patients (32 patients in each group); enrolled using nonprobability sampling technique – convenience sampling. Each participant was personally interviewed using a semi-structured proforma, through which demographic characteristics, including age and sex, anthropometric measurements such as height and weight, clinical diagnosis, type of surgical procedure, ASA physical status, and MPC assessment were recorded. Participants were allocated to three study groups: the control group received 0.125% bupivacaine alone, Group 1 received 0.125% bupivacaine with dexmedetomidine 1 µg/mL, and Group 2 received 0.125% bupivacaine with dexmedetomidine 2 µg/mL through continuous epidural infusion. Following completion of surgery and transfer to the postoperative care unit, baseline clinical parameters, including the Visual Analogue Scale (VAS) score, heart rate, blood pressure, mean arterial pressure, and peripheral oxygen saturation (SpO_2), were recorded before initiation of the study-drug infusion. Patients in whom intraoperative activation of the epidural catheter was required and those whose surgical duration exceeded 3.5 hours were excluded to minimize potential bias. Subsequently, VAS score and hemodynamic parameters were monitored and recorded at activation of the study-drug infusion and at 30 minutes, 60 minutes, 2 hours, 6 hours, 12

hours, 18 hours, and 24 hours after initiation of the infusion.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics for Windows, version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean and standard deviation, whereas categorical variables were presented as frequencies and percentages. Comparisons among the three study groups were performed using appropriate parametric or non-parametric tests, including analysis of variance (ANOVA) or the Kruskal–Wallis test for continuous variables and the chi-square test for categorical variables. Post hoc pairwise comparisons were performed when indicated. A two-sided p value <0.05 was considered statistically significant.

Results

The mean age was 52.31 ± 10.29 years in the control group, 52.78 ± 9.50 years in Group 1, and 49.97 ± 7.81 years in Group 2, with no significant difference among the groups (p=0.432). Females constituted 56.3% of the overall study population, and gender distribution was also comparable (p=0.881). Most participants were classified as ASA II (78.1%), followed by ASA I (20.8%), with no significant difference in ASA classification among the groups (p=0.425). TAH with BSO was the most frequently performed procedure (31.3%), followed by bilateral inguinal hernioplasty (20.8%), and the distribution of surgical procedures was similar across the three groups (p=0.982). VAS scores differed significantly among the three groups at all assessed time points.

At baseline, the mean VAS score was 2.59 ± 0.49 in the control group, 2.06 ± 0.72 in Group 1, and 1.66 ± 0.48 in Group 2 (p<0.001). At 6 hours, the corresponding scores were 2.84 ± 0.77, 1.91 ± 0.64, and 1.66 ± 0.48, respectively, while at 24 hours they were 3.16 ± 0.72, 1.91 ± 0.64, and 1.66 ± 0.48, respectively (p<0.001 for both comparisons). Pairwise comparisons showed significantly lower VAS scores in both Group 1 and Group 2 compared with the control group throughout the observation period, with Group 2 consistently demonstrating the lowest pain scores.

SBP differed significantly among the three groups at all assessed time points (p<0.001). At baseline, mean SBP was 140.47 ± 10.73 mmHg in the control group and 121.88 ± 10.91 mmHg in both Groups 1 and 2. Group 2 subsequently showed consistently lower SBP values, decreasing to 89.47 ± 8.21 mmHg at 24 hours, compared with 133.13 ± 6.57 mmHg in the control group and 121.88 ± 10.91 mmHg in Group 1. Pairwise comparisons between the control and Group 2 were significant at all time points, whereas differences between the control and Group 1 were not significant at 30 minutes (p=0.088) and 12 hours (p=0.144). Baseline and activation DBP values were comparable among the groups (p=0.940); however, significant differences emerged from 30 minutes onward (p<0.001). At 24 hours, mean DBP was 80.94 ± 6.41, 78.44 ± 9.54, and 59.69 ± 2.88 mmHg in the control, Group 1, and Group 2, respectively, with Group 2 remaining significantly lower than the control throughout the post-activation period. MAP showed significant overall differences among the three groups at every assessed time point (p<0.001). At baseline, MAP was 78.25 ± 7.23 mmHg in the control group, 80.88 ± 5.60 mmHg in Group 1, and 72.16 ± 7.95 mmHg in Group 2. At 6 hours, the corresponding values were 78.09 ± 7.13, 79.72 ± 5.83, and 69.50 ± 6.61 mmHg. Pairwise differences varied according to the time point, with Group 2 differing significantly from the control at several intervals, although not at 12 hours (p=0.755) or 24 hours (p=0.890). Pulse rate also differed significantly among the groups throughout follow-up (p<0.001), with Group 2 consistently demonstrating the lowest values. At baseline, mean pulse rates were 77.19 ± 11.28, 76.78 ± 9.29, and 61.53 ± 6.10 beats/min in the control, Group 1, and Group 2, respectively; at 24 hours, the respective values were 69.81 ± 4.59, 75.00 ± 9.33, and 60.84 ± 5.35 beats/min. Group 2 differed significantly from the control at all time points, whereas Group 1 did not differ significantly at baseline, activation, 30 minutes, or 6 hours. SpO_2 remained high in all three groups throughout the 24-hour observation period, despite statistically significant overall differences among the groups. At baseline, mean SpO_2 was 98.38 ± 0.55% in the control group, 98.81 ± 0.39% in Group 1, and 98.47 ± 0.51% in Group 2 (p=0.001). Similar values were maintained at 24 hours at 98.31 ± 0.59%, 98.81 ± 0.39%, and 98.47 ± 0.51%, respectively (p=0.001). Pairwise analysis demonstrated significant differences between the control group and Group 1 at most time points, whereas no significant differences were observed between the control group and Group 2 throughout follow-up, with p values ranging from 0.150 to 0.868.

Table 1: Baseline characteristics, ASA classification, and type of surgery across the three groups (N=96)

	Control (n=32)	Group 1 (n=32)	Group 2 (n=32)	Total (N=96)	p value
Age, years, mean (SD)	52.31 (10.29)	52.78 (9.50)	49.97 (7.81)	51.69 (9.24)	0.432
Gender, n (%)					
Male	13 (40.6%)	14 (43.8%)	15 (46.9%)	42 (43.8%)	0.881
Female	19 (59.4%)	18 (56.3%)	17 (53.1%)	54 (56.3%)	
ASA classification, n (%)					
ASA I	5 (15.6%)	6 (18.8%)	9 (28.1%)	20 (20.8%)	0.425
ASA II	27 (84.4%)	26 (81.3%)	22 (68.8%)	75 (78.1%)	
ASA III	0 (0.0%)	0 (0.0%)	1 (3.1%)	1 (1.0%)	
Type of surgery, n (%)					
B/l Inguinal hernioplasty	7 (21.9%)	6 (18.8%)	7 (21.9%)	20 (20.8%)	0.982
Incisional hernioplasty	1 (3.1%)	1 (3.1%)	3 (9.4%)	5 (5.2%)	
Left femur fracture	0 (0.0%)	1 (3.1%)	0 (0.0%)	1 (1.0%)	
Left hemiarthoplasty	1 (3.1%)	1 (3.1%)	3 (9.4%)	5 (5.2%)	
Left tibia fibula fracture ORIF	0 (0.0%)	0 (0.0%)	1 (3.1%)	1 (1.0%)	
Left TKR	1 (3.1%)	1 (3.1%)	1 (3.1%)	3 (3.1%)	
Left tri malleolar fracture	1 (3.1%)	1 (3.1%)	1 (3.1%)	3 (3.1%)	
Recurrent incisional hernioplasty	1 (3.1%)	1 (3.1%)	0 (0.0%)	2 (2.1%)	
Right femur orif plating	1 (3.1%)	1 (3.1%)	0 (0.0%)	2 (2.1%)	
Right hemiarthoplasty	1 (3.1%)	1 (3.1%)	1 (3.1%)	3 (3.1%)	
Right hernioplasty	0 (0.0%)	0 (0.0%)	1 (3.1%)	1 (1.0%)	
Right para ovarian cystectomy	1 (3.1%)	1 (3.1%)	0 (0.0%)	2 (2.1%)	
Right tibia compound fracture	0 (0.0%)	1 (3.1%)	0 (0.0%)	1 (1.0%)	
Right tibia compound fracture ORIF	0 (0.0%)	0 (0.0%)	1 (3.1%)	1 (1.0%)	
Right TKR	1 (3.1%)	1 (3.1%)	3 (9.4%)	5 (5.2%)	
Right trimalleolar fracture	1 (3.1%)	1 (3.1%)	0 (0.0%)	2 (2.1%)	
TAH with BSO	12 (37.5%)	10 (31.3%)	8 (25.0%)	30 (31.3%)	
Umbilical hernioplasty	1 (3.1%)	1 (3.1%)	0 (0.0%)	2 (2.1%)	
Vaginal hysterectomy	1 (3.1%)	1 (3.1%)	0 (0.0%)	2 (2.1%)	
VH with PFR	1 (3.1%)	2 (6.3%)	2 (6.3%)	5 (5.2%)	

Table 2: Visual Analogue Scale (VAS) scores from baseline to 24 hours with overall and pairwise comparisons (N=96)

Time point	Control, mean (SD)	Group 1, mean (SD)	Group 2, mean (SD)	Overall p	Control vs Group 1 p	Control vs Group 2 p
VAS						
Baseline	2.59 (0.49)	2.06 (0.72)	1.66 (0.48)	0.000*	0.001*	0.000*
Activation	2.56 (0.50)	2.06 (0.72)	1.66 (0.48)	0.000*	0.002*	0.000*
30 min	3.00 (0.00)	2.06 (0.72)	1.66 (0.48)	0.000*	0.000*	0.000*
60 min	2.50 (0.51)	1.56 (0.50)	1.66 (0.48)	0.000*	0.000*	0.000*
2 Hours	2.84 (0.81)	1.91 (0.59)	1.66 (0.48)	0.000*	0.000*	0.000*
6 Hours	2.84 (0.77)	1.91 (0.64)	1.66 (0.48)	0.000*	0.000*	0.000*
12 Hours	3.13 (0.66)	2.06 (0.72)	1.66 (0.48)	0.000*	0.000*	0.000*
18 Hours	3.03 (0.69)	1.91 (0.59)	1.66 (0.48)	0.000*	0.000*	0.000*
24 Hours	3.16 (0.72)	1.91 (0.64)	1.66 (0.48)	0.000*	0.000*	0.000*

Table 3: Systolic and diastolic blood pressure from baseline to 24 hours with overall and pairwise comparisons (N=96)

Time point	Control, mean (SD)	Group 1, mean (SD)	Group 2, mean (SD)	Overall p	Control vs Group 1 p	Control vs Group 2 p
Panel A. Systolic blood pressure (SBP)						
Baseline	140.47 (10.73)	121.88 (10.91)	121.88 (10.91)	0.000*	0.001*	0.000*
Activation	140.47 (10.73)	121.88 (10.91)	121.88 (10.91)	0.000*	0.002*	0.000*
30 min	115.13 (9.33)	120.84 (11.99)	96.56 (10.66)	0.000*	0.088	0.000*
60 min	112.69 (5.97)	120.84 (11.99)	91.19 (9.72)	0.000*	0.003*	0.000*
2 Hours	115.13 (9.33)	121.88 (10.91)	90.59 (9.07)	0.000*	0.019*	0.000*
6 Hours	122.97 (10.02)	116.25 (8.71)	91.19 (9.72)	0.000*	0.016*	0.000*
12 Hours	126.88 (11.41)	121.88 (10.91)	90.56 (9.09)	0.000*	0.144	0.000*
18 Hours	131.53 (8.65)	122.41 (10.45)	89.78 (8.97)	0.000*	0.001*	0.000*
24 Hours	133.13 (6.57)	121.88 (10.91)	89.47 (8.21)	0.000*	0.000*	0.000*
Panel B. Diastolic blood pressure (DBP)						
Baseline	79.16 (9.13)	78.44 (9.54)	78.44 (9.54)	0.940	0.950	0.950
Activation	79.16 (9.13)	78.44 (9.54)	78.44 (9.54)	0.940	0.950	1.000
30 min	70.75 (9.09)	76.25 (4.92)	60.00 (0.00)	0.000*	0.01*	0.000*
60 min	73.09 (6.99)	76.25 (4.92)	59.75 (2.68)	0.000*	0.043*	0.000*
2 Hours	70.75 (9.09)	78.44 (9.54)	60.00 (2.59)	0.000*	0.000*	0.000*
6 Hours	75.59 (8.46)	75.00 (5.96)	60.00 (2.59)	0.000*	0.921	0.000*
12 Hours	78.94 (6.84)	78.44 (9.54)	60.19 (2.35)	0.000*	0.955	0.000*
18 Hours	78.22 (7.92)	77.03 (4.37)	59.72 (3.29)	0.000*	0.670	0.000*
24 Hours	80.94 (6.41)	78.44 (9.54)	59.69 (2.88)	0.000*	0.314	0.000*

Table 4: Mean arterial pressure and pulse rate from baseline to 24 hours with overall and pairwise comparisons (N=96)

Time point	Control, mean (SD)	Group 1, mean (SD)	Group 2, mean (SD)	Overall p	Control vs Group 1 p	Control vs Group 2 p
Panel A. Mean arterial pressure (MAP)						
Baseline	78.25 (7.23)	80.88 (5.60)	72.16 (7.95)	0.000*	0.295	0.002*
Activation	78.09 (7.13)	80.88 (5.60)	72.16 (7.95)	0.000*	0.252	0.003*
30 min	71.47 (5.38)	79.13 (6.39)	67.44 (2.97)	0.000*	0.000*	0.006*
60 min	73.19 (4.25)	78.03 (5.88)	69.47 (5.95)	0.000*	0.002*	0.020*
2 Hours	75.03 (5.57)	80.03 (5.92)	68.91 (6.02)	0.000*	0.003*	0.000*
6 Hours	78.09 (7.13)	79.72 (5.83)	69.50 (6.61)	0.000*	0.583	0.000*
12 Hours	71.47 (5.38)	79.16 (5.67)	70.34 (7.61)	0.000*	0.000*	0.755
18 Hours	78.09 (7.13)	78.22 (5.69)	71.38 (7.74)	0.000*	0.997	0.001*
24 Hours	71.47 (5.38)	79.78 (5.67)	70.75 (7.49)	0.000*	0.000*	0.890
Panel B. Pulse rate (PR)						
Baseline	77.19 (11.28)	76.78 (9.29)	61.53 (6.10)	0.000*	0.983	0.000*
Activation	74.81 (7.61)	76.78 (9.29)	61.53 (6.10)	0.000*	0.571	0.000*
30 min	72.19 (6.76)	74.59 (9.43)	60.63 (5.64)	0.000*	0.403	0.000*
60 min	67.59 (4.93)	74.00 (8.22)	59.25 (5.59)	0.000*	0.000*	0.000*
2 Hours	69.81 (4.59)	76.94 (9.49)	60.84 (5.61)	0.000*	0.000*	0.000*
6 Hours	72.16 (5.32)	71.84 (7.33)	59.16 (5.67)	0.000*	0.978	0.000*
12 Hours	72.19 (6.76)	77.66 (8.94)	58.88 (5.33)	0.000*	0.008*	0.000*
18 Hours	67.59 (4.93)	72.91 (7.35)	60.22 (5.53)	0.000*	0.002*	0.000*
24 Hours	69.81 (4.59)	75.00 (9.33)	60.84 (5.35)	0.000*	0.008*	0.000*

Table 5: SPO2 from baseline to 24 hours with overall and pairwise comparisons (N=96)

Time point	Control, mean (SD)	Group 1, mean (SD)	Group 2, mean (SD)	Overall p	Control vs Group 1 p	Control vs Group 2 p
SPO2						
Baseline	98.38 (0.55)	98.81 (0.39)	98.47 (0.51)	0.001*	0.002	0.725
Activation	98.31 (0.59)	98.81 (0.39)	98.47 (0.51)	0.001*	0.001*	0.434
30 min	98.38 (0.49)	98.81 (0.39)	98.47 (0.51)	0.001*	0.001*	0.703
60 min	98.22 (0.66)	98.81 (0.39)	98.47 (0.51)	0.000*	0.001*	0.150
2 Hours	98.41 (0.49)	98.81 (0.39)	98.47 (0.51)	0.002*	0.002*	0.856
6 Hours	98.25 (0.67)	98.81 (0.39)	98.47 (0.51)	0.000*	0.001*	0.239
12 Hours	98.41 (0.56)	98.81 (0.39)	98.47 (0.51)	0.003*	0.004*	0.868
18 Hours	98.31 (0.59)	98.81 (0.39)	98.47 (0.51)	0.001*	0.001*	0.434
24 Hours	98.31 (0.59)	98.81 (0.39)	98.47 (0.51)	0.001*	0.001*	0.434

Discussion

The present study evaluated continuous epidural 0.125% bupivacaine alone and in combination with dexmedetomidine at concentrations of 1 µg/mL and 2 µg/mL for postoperative analgesia following infraumbilical surgery. The three groups were broadly comparable demographically: the mean age was 52.31 ± 10.29 years in the control group, 52.78 ± 9.50 years in Group 1, and 49.97 ± 7.81 years in Group 2 (p=0.432), while sex distribution and ASA physical status were also comparable. Overall, 78.1% of participants were ASA II and 20.8% were ASA I. The distribution of surgical procedures was similar among the groups (p=0.982), with total abdominal hysterectomy with bilateral salpingo-oophorectomy accounting for 31.3% and bilateral inguinal hernioplasty for 20.8%.

This broad balance permitted comparison of the postoperative analgesic and physiological profiles of the three epidural regimens across a heterogeneous infraumbilical surgical population. [8-10] A major finding was the sustained reduction in pain scores when dexmedetomidine was added to epidural bupivacaine. At 6 hours, mean VAS scores were 2.84 ± 0.77 in the control group, 1.91 ± 0.64 in Group 1, and 1.66 ± 0.48 in Group 2; at 24 hours, the corresponding scores were 3.16 ± 0.72, 1.91 ± 0.64, and 1.66 ± 0.48, respectively (p<0.001). Hetta et al. randomized 64 patients undergoing major abdominal cancer surgery to epidural 0.1% bupivacaine alone or bupivacaine with dexmedetomidine 0.5 µg/mL. [11] Addition of dexmedetomidine reduced mean VAS scores at rest from 2.38 ± 0.08 to 1.60 ± 0.08 and during movement from 3.25 ± 0.07 to 2.17 ± 0.07, while cumulative 48-hour morphine consumption decreased from 23.23 ± 8.37 mg to 10.40 ± 5.16 mg (p<0.001). [11] The consistently lower pain scores in Group 2 suggested a dose-related enhancement of postoperative analgesia. Kurhekar et al. compared continuous epidural dexmedetomidine 0.5 µg/kg/24 h with 1 µg/kg/24 h, each administered with 0.1% ropivacaine at 5 mL/h for 48 hours, in 72 patients undergoing lower-limb surgery. [7] The 1 µg/kg/24 h regimen produced significantly lower pain intensity and a lower requirement for rescue analgesia than the lower-dose regimen. Afandy et al. similarly reported that adding dexmedetomidine to epidural 0.125% bupivacaine shortened the onset of analgesia from 16.8 to 9.23 minutes and prolonged its duration from 102.8 to 169.13 minutes, with both differences reaching p<0.001. [12] Nazarian et al. demonstrated dose-dependent spinal antinociception mediated predominantly through α2A-adrenoceptors, accompanied by suppression of dorsal-horn neuronal activation, providing a biological basis for the potentiation of neuraxial local-anesthetic analgesia by dexmedetomidine. [13]

The hemodynamic profile showed a substantially greater effect with the higher dexmedetomidine concentration. In Group 2, SBP decreased from 121.88 ± 10.91 mmHg at baseline to 89.47 ± 8.21 mmHg at 24 hours, compared with 133.13 ± 6.57 mmHg in the control group and 121.88 ± 10.91 mmHg in Group 1 at 24 hours.

DBP was comparable among the groups at baseline; however, from 30 minutes onward, Group 2 values remained close to 60 mmHg and were significantly below those of the control group. MAP showed a similar pattern; at 6 hours it was 78.09 ± 7.13 mmHg in the control group, 79.72 ± 5.83 mmHg in Group 1, and 69.50 ± 6.61 mmHg in Group 2. Pulse rate also remained lowest in Group 2, reaching 60.84 ± 5.35 beats/min at 24 hours compared with 69.81 ± 4.59 beats/min in the control group and 75.00 ± 9.33 beats/min in Group 1. Since SBP, MAP, pulse rate, and VAS differed to varying degrees before activation of the study infusion, the subsequent absolute values are best interpreted in relation to their respective baseline measurements; nevertheless, the sustained post-activation reduction in Group 2 was pharmacologically consistent with a stronger α2-mediated sympatholytic response. Clinical dose-response supports this hemodynamic pattern. Kurhekar et al. reported that the higher continuous epidural dose of dexmedetomidine was associated with greater hypotension while simultaneously

producing better analgesia than the lower dose. [7] Wan et al. demonstrated a dose-dependent effect of epidural dexmedetomidine on both local-anesthetic requirements and heart rate: dexmedetomidine doses of 0.75 and 1.0 µg/kg significantly reduced heart rate, whereas values with 0.5 µg/kg remained comparatively close to those of the control group. [14] Their findings indicated that increasing the epidural dexmedetomidine dose enhanced the local-anesthetic effect but simultaneously intensified cardiovascular effects. The marked reductions in SBP, DBP, MAP, and pulse rate observed in Group 2 of the present study therefore fit a recognizable dose-related sympatholytic pattern.

Group 1 demonstrated an intermediate clinical profile, with substantially lower VAS scores than the control group but less persistent reduction in blood pressure than Group 2. Chiruvella et al. studied 80 women undergoing total abdominal hysterectomy and found that epidural dexmedetomidine 1 µg/kg combined with 0.125% levobupivacaine produced an earlier onset and longer duration of analgesia than epidural clonidine.[15] Only 42.5% of patients receiving dexmedetomidine required rescue analgesia compared with 70% in the clonidine group, while cardiorespiratory parameters remained stable. These observations support the finding that a lower dexmedetomidine regimen may retain substantial analgesic efficacy while exerting a less pronounced hemodynamic effect than a higher concentration.

Oxygenation remained clinically preserved throughout the present study. Mean SpO_2 remained approximately 98% in all three groups; at 24 hours, values were 98.31 ± 0.59% in the control group, 98.81 ± 0.39% in Group 1, and 98.47 ± 0.51% in Group 2. Although statistically significant overall differences were present, the absolute differences were small, and pairwise comparisons between Group 2 and the control group remained nonsignificant throughout follow-up. Soliman and Melika similarly documented comparable oxygen saturation during epidural bupivacaine-based regimens, with baseline SpO_2 values of 97.27 ± 1.39%, 97.53 ± 1.41%, and 97.10 ± 1.30% across their study groups (p=0.770). [16] Thus, in the present study, the dose-associated cardiovascular changes observed with dexmedetomidine occurred without a clinically relevant deterioration in peripheral oxygen saturation.

The present study had several limitations. It was conducted at a single tertiary-care center with a relatively modest sample size of 96 patients, which may limit the generalizability of the findings. The study included a heterogeneous range of infraumbilical surgical procedures, with potentially different postoperative pain characteristics. Outcomes were assessed only up to 24 hours after initiation of epidural analgesia, precluding evaluation of longer-term analgesic efficacy and delayed adverse effects.

Furthermore, the reported analysis primarily focused on VAS scores, blood pressure, pulse rate, and SpO_2, while detailed assessment of rescue analgesic consumption, sedation, motor blockade, patient satisfaction, and the incidence of specific adverse events such as bradycardia and hypotension was limited.

Conclusion

The addition of dexmedetomidine to continuous epidural infusion of 0.125% bupivacaine provided superior postoperative analgesia compared with bupivacaine alone in patients undergoing infraumbilical surgeries.

Both dexmedetomidine groups demonstrated significantly lower VAS scores throughout the 24-hour observation period, with the 2 µg/mL concentration producing the lowest pain scores. However, the higher dexmedetomidine concentration was also associated with more pronounced reductions in systolic and diastolic blood pressure, mean arterial pressure, and pulse rate, indicating a greater hemodynamic effect.

In contrast, the 1 µg/mL concentration provided effective analgesia with comparatively less marked hemodynamic alteration. Peripheral oxygen saturation remained well maintained in all groups. These findings suggest that epidural dexmedetomidine is an effective adjuvant to bupivacaine for postoperative pain control, with the lower concentration offering a more favorable balance between analgesic efficacy and hemodynamic stability.

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