Original Article

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Anaesthesia Section

Efficacy of Two Doses of Dexmedetomidine as an Adjuvant to Intrathecal Hyperbaric Ropivacaine in Lower Limb Surgeries: A Randomised Controlled Trial

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ABSTRACT

Introduction: Regional anaesthesia techniques are widely used for lower limb surgeries, offering superior postoperative pain relief and faster recovery. Dexmedetomidine, a highly selective α2-adrenergic agonist, has emerged as a promising adjuvant to local anaesthetics in intrathecal anaesthesia, providing prolonged analgesia without significant adverse effects.

Aim: To compare the efficacy of two different doses of dexmedetomidine (5 mcg versus 10 mcg) as an adjuvant to intrathecal hyperbaric ropivacaine in patients undergoing lower limb surgeries.

Materials and Methods: This double-blinded, randomised controlled trial was conducted at the Department of Anaesthesia and Critical Care, Pradyumna Bal Memorial Hospital (PBMH), Kalinga Institute of Medical Science, Bhubaneswar, Odisha, India, from May 2023 to September 2025. The study included 76 American Society of Anaesthesiologists (ASA) physical status I-II patients aged 18-60 years undergoing elective lower limb surgeries. Patients were randomly allocated into two groups. Group A (n=38) received 3 mL of 0.75% hyperbaric ropivacaine with 5 mcg dexmedetomidine, and Group B (n=38) received 3 mL of 0.75% hyperbaric ropivacaine with 10 mcg dexmedetomidine intrathecally. Primary outcomes included postoperative Visual Analogue Scale (VAS) scores and duration of analgesia. Secondary outcomes encompassed haemodynamic parameters, onset of sensory and motor blocks, sedation scores, and adverse effects. Data analysis was performed using IBM Statistical Package for Social Sciences (SPSS) software version 25.0 with the Mann-Whitney U test for continuous variables and the Chi-square test for categorical variables. A p-value <0.05 was considered statistically significant.

Results: Both groups were comparable regarding demographic variables, including age (group A: 41.0±10.5 years vs group B: 38.5±11.2 years, p-value=0.28), gender distribution, Body Mass Index (BMI), and ASA physical status. Group B demonstrated significantly lower VAS scores at 2, 4, 6, and 8 hours postoperatively compared to Group A (p-value <0.05). At 2 hours, mean VAS was 1.5±1.2 in group B versus 3.2±1.5 in group A (p-value=0.01). The requirement for rescue analgesia was significantly delayed in group B, with 71.05% of group A patients requiring tramadol at 6 hours versus only 2.63% in group B (p-value <0.001). Group B achieved faster sensory block onset to T8 level (mean time: 4.5±0.8 minutes) compared to group A (mean time: 5.8±1.2 minutes, p-value=0.01). Haemodynamic parameters remained stable in both groups with no significant differences. No cases of postoperative nausea and vomiting were observed in either group.

Conclusion: Intrathecal dexmedetomidine 10 mcg provides superior postoperative analgesia, faster sensory block onset, and reduced rescue analgesic requirements compared to the 5 mcg dose, while maintaining a comparable haemodynamic safety profile. This higher dose represents an optimal balance between enhanced analgesic efficacy and safety in lower limb surgeries.

Keywords: Local Anaesthetics, Postoperative pain, Sensory block

INTRODUCTION

Subarachnoid Block (SAB) remains one of the most widely employed regional anaesthesia techniques for lower limb surgeries, offering rapid onset, profound sensory blockade, and minimal systemic pharmacological effects [1]. While SAB provides superior postoperative pain relief compared to systemic analgesics and promotes early recovery of mobility and bowel function, [2] its limited duration often poses clinical challenges. Inadequate pain control can significantly impede functional recovery, delaying early physiotherapy and rehabilitation protocols essential for optimal patient outcomes [3]. The quest for prolonging the duration of regional anaesthesia has led to extensive research into various pharmacological adjuvants. Opioids, traditionally added to local anaesthetics for neuraxial blocks, are frequently limited by significant adverse effects, including urinary retention, respiratory depression, haemodynamic instability, pruritus, and severe nausea and vomiting [4,5]. This has prompted investigation into alternative adjuvants, including epinephrine, neostigmine, magnesium, midazolam, ketamine, and clonidine, each offering distinct advantages and limitations [6,7].

Alpha-2 (α2) adrenergic receptor agonists have emerged as particularly promising adjuvants due to their unique pharmacological profile encompassing sedative, analgesic, perioperative sympatholytic, anaesthetic-sparing, and Haemodynamic stabilisation properties [8,9]. Dexmedetomidine, a highly selective α2-adrenergic agonist introduced in clinical practice, has evolved as a versatile agent for various perioperative and critical care applications. Its favourable safety profile, characterised by the absence of respiratory depression, makes it an attractive and relatively safer adjunct for diverse clinical applications [10,11]. Ropivacaine, introduced in 1996, represents a newer generation amide local anaesthetic with a superior safety profile compared to bupivacaine [12]. Its high protein-binding capacity and favourable pharmacokinetic properties, combined with reduced cardiac and neurological toxicity, have

established it as a preferred agent for intrathecal anaesthesia [13]. The specific gravity of local anaesthetic solutions plays a crucial role in determining drug spread within the cerebrospinal fluid, thereby influencing the extent of motor and sensory blockade [14].

Despite the growing interest in dexmedetomidine as a neuraxial adjuvant, limited literature exists comparing different doses of intrathecal dexmedetomidine with ropivacaine in intrathecal anaesthesia [15]. The optimal dose that provides adequate duration of blockade and analgesia while minimising adverse effects remains a subject of ongoing investigation. The present study was designed to compare the efficacy and safety of two different doses of intrathecal dexmedetomidine (5 mcg versus 10 mcg) as adjuvants to hyperbaric ropivacaine in patients undergoing lower limb surgeries. The primary objective of the current study was to compare the efficacy of two different doses of dexmedetomidine as an adjuvant to intrathecal ropivacaine in lower limb surgeries. The secondary objective was to estimate the incidence of hypotension and bradycardia and to determine the duration of onset of sensory block up to the level of T8.

MATERIALS AND METHODS

This double-blinded, randomised controlled trial was conducted at the Department of Anaesthesia and Critical Care, PBMH, KIMS, Bhubaneswar, Odisha, India, from May 2023 to September 2025. The study was approved by the Institutional Ethics Committee (Reference No: KIIT/KIMS/IEC/1214/2023) and registered with Clinical Trial Registry of India (Registration Number: CTRI/2023/05/052743). Written informed consent was obtained from all participants before enrollment.

Sample size calculation: Based on previous literature by Farokhmehr L et al., (2019), [15] which reported mean VAS scores at 12 hours of 3.40±0.50 in the 5 mcg group and 2.93±0.37 in the 10 mcg group, and considering 5% level of significance, 95% confidence interval, and 95% power, the sample size was calculated using the formula:

$n=2(Z\alpha + Z\beta)^2 \sigma^2 / (\mu 1 - \mu 2)^2$.

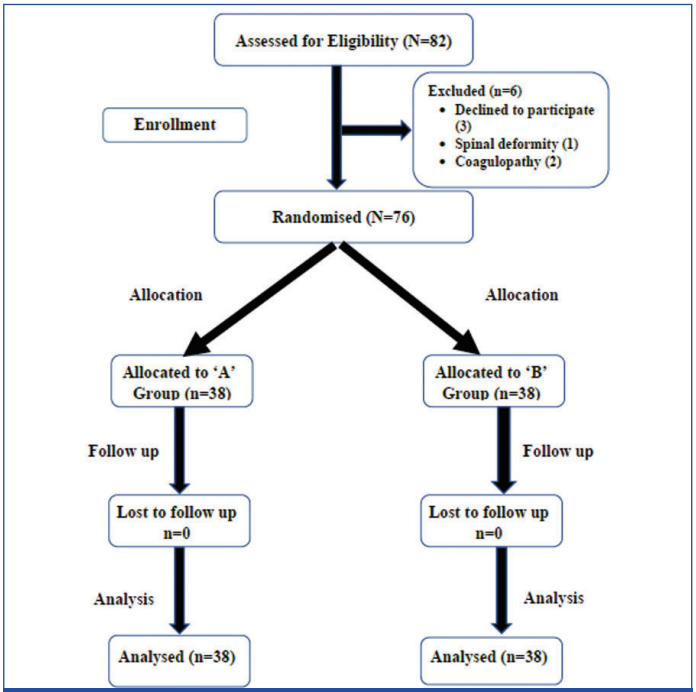
Accounting for 20% attrition, the minimum calculated sample size was 38 patients per group, totalling 76 participants.

Inclusion criteria: The study included patients aged 18-60 years with ASA physical status I or II who were scheduled for elective lower limb orthopedic surgeries under spinal anaesthesia and were willing to provide informed consent.

Exclusion criteria: Patients were excluded if they refused to participate, had a history of allergy to study drugs (ropivacaine, dexmedetomidine, tramadol, or ondansetron), were pregnant or lactating, had spinal deformity or anatomical abnormalities, had sinus bradycardia (heart rate <50 beats/minute), had coagulopathy or bleeding disorders, had infection at the injection site, had any contraindications to intrathecal anaesthesia, had neurological disorders, had severe hepatic or renal impairment, or had a history of psychiatric disorders.

A total of 82 patients were assessed for eligibility. Six patients were excluded (3 declined to participate, 2 had coagulopathy, and 1 had spinal deformity). The remaining 76 patients were randomised into two groups of 38 patients each. All 76 patients completed the study with no dropouts or protocol violations.

Computer-generated randomisation tables were used for group allocation [Table/Fig-1]. Randomisation sequence was generated by a biostatistician not involved in patient care. Patients were randomly assigned using the opaque sealed envelope technique, which was opened by the anaesthesiologist preparing the study drug just before the procedure. A double-blind approach was maintained where neither the patients nor the principal investigator conducting assessments knew the medication allocation. Study medications were prepared by an anaesthesiologist not involved in data collection or analysis to minimise bias. The medication syringes were identical in appearance and labelled only with the patient study number.



[Table/Fig-1]: CONSORT (Consolidated Standards of Reporting Trials) flow diagram for enrollment, group allocation, follow-up, and analysis.

Study Procedure

The choice of drug doses was based on previous studies demonstrating the safety and efficacy of dexmedetomidine in the range of 3-10 mcg for intrathecal use [15].

- **Group A (control group, n=38):** 3 mL of 0.75% hyperbaric ropivacaine with 5 mcg dexmedetomidine (diluted to a total volume of 3.1 mL with normal saline)
- **Group B (n=38):** 3 mL of 0.75% hyperbaric ropivacaine with 10 mcg dexmedetomidine (total volume 3.1 mL)

The primary outcomes of the study were the assessment of postoperative pain intensity, measured using the Visual Analogue Scale (VAS) at 2, 4, 6, 8, 10, and 12-hour intervals, and the determination of the duration of analgesia, defined as the time from the onset of the sensory block to the first patient request for analgesia when the VAS score exceeded 4. The Secondary were the onset time to achieve a sensory block at the T8 level and the onset time for a complete motor block, indicated by a modified Bromage score of 3. Furthermore, the study monitored Haemodynamic stability by recording the incidence of hypotension and bradycardia, assessed sedation levels using the Ramsay Sedation Scale, and documented the prevalence of adverse effects such as Postoperative Nausea and Vomiting (PONV), pruritus, respiratory depression, and urinary retention.

All patients received preloading with 10 mL/kg Ringer's lactate solution via an 18-gauge intravenous cannula. Standard ASA monitors, including non invasive blood pressure, electrocardiogram, and pulse oximetry, were attached. Under strict aseptic conditions, spinal anaesthesia was performed at L3-L4 or L2-L3 intervertebral space in sitting position using a 25-gauge Quincke's needle. After confirming free flow of cerebrospinal fluid, the study medication was injected intrathecally.

Assessments: Sensory block was assessed bilaterally using the pinprick test with a 23-gauge needle at 2-minute intervals until T8 dermatome level was achieved, then at 5-minute intervals until stabilisation [15]. Motor block was evaluated using the modified Bromage scale:

- 0=no motor block (able to raise extended leg);
- 1=inability to raise the extended leg, but able to flex the knee;

- 2=inability to flex the knee but able to flex the ankle;
- 3=complete motor block with inability to move leg or foot. Surgery commenced only after achieving sensory block to the T8 dermatome and a modified Bromage score of 3.

Haemodynamic parameters including Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), Heart Rate (HR), and Oxygen Saturation (SpO_2) were recorded at baseline (preoperatively), then intraoperatively at 15 minutes, 30 minutes, 60 minutes, 90 minutes, 120 minutes, 150 minutes, and 180 minutes after intrathecal injection. Postoperatively, measurements were taken first at 2 hours, then at 4, 6, 8, 10, and 12 hours.

Sedation was assessed using the Ramsay Sedation Scale:

- 1=anxious and agitated;
- 2=cooperative, oriented, and tranquil;
- 3=responds to commands only;
- 4=brisk response to light glabellar tap or loud auditory stimulus;
- 5=sluggish response to light glabellar tap or loud auditory stimulus;
- 6=no response.

Postoperative pain was assessed using the Visual Analogue Scale (VAS), where 0 represents no pain and 10 represents worst imaginable pain. VAS scores were recorded at 2, 4, 6, 8, 10, and 12 hours postoperatively.

Management Protocols: Rescue analgesia with tramadol 1.5 mg/kg i.v. was administered when VAS≥4. Hypotension (SBP decrease >20% from baseline or <90 mmHg) was treated with ephedrine 6 mg i.v. bolus. Bradycardia (HR decrease >20% from baseline or <50 bpm) was managed with atropine 0.6 mg i.v. PONV was treated with ondansetron 8 mg i.v. Respiratory depression (RR<10/min or SpO_2<92%) was managed with supplemental oxygen.

STATISTICAL ANALYSIS

Data analysis was performed using IBM SPSS version 25.0. Continuous variables were expressed as mean±SD or median-Interquartile Range (IQR) after Shapiro-Wilk normality testing. Between group comparisons used an Independent t-test or the Mann-Whitney U test for continuous variables and a Chi-square or Fisher's exact test for categorical variables. A p-value <0.05 was considered significant.

RESULTS

Demographic Characteristics

Both groups were demographically comparable with no significant differences in age, gender, height, weight, BMI, ASA physical status, or surgery duration [Table/Fig-2]. The mean age was 41.0±10.5 years in group A versus 38.5±11.2 years in group B (p-value=0.28). Male patients comprised 47.4% and 52.6% of groups A and B, respectively (p-value=0.64). Mean BMI and surgery duration were similar between groups (p-value >0.05).

Haemodynamic Parameters

Intraoperative and postoperative haemodynamic parameters remained stable and comparable between groups throughout the study period [Table/Fig-3]. The SBP, DBP, MAP, and HR showed no statistically significant differences at any time point (all p-values>0.05). Baseline mean SBP was 128.5±12.3 mmHg in group A and 126.8±11.9 mmHg in group B (p-value=0.54). At 15 minutes after intrathecal injection, mean SBP was 118.2±10.5 mmHg in group A and 116.5±11.2 mmHg in group B (p-value=0.48). Oxygen saturation remained consistently between 98-100% in both groups with no episodes of desaturation. No patient in either group required intervention for hypotension or bradycardia.

Variables	Group A (5 µg) (n=38)	Group B (10 µg) (n=38)	t- value	p-value
Age (years), (Mean±SD)	41.0±10.5	38.5±11.2	1.09	0.28
Gender (M/F), n (%)	18 (47.4) / 20 (52.6)	20 (52.6) / 18 (47.4)	–	0.64
Height (cm), (Mean±SD)	165.2±8.4	166.8±7.9	0.87	0.39
Weight (kg), (Mean±SD)	67.5±9.2	68.1±8.7	0.31	0.76
BMI (kg/m^2), (Mean±SD)	24.6±1.8	24.3±1.6	0.81	0.42
ASA I/II, n (%)	26 (68.4) / 12 (31.6)	27 (71.1) / 11 (28.9)	–	0.79
Surgery duration (min), (Mean±SD)	85.5±8.2	84.2±7.8	0.73	0.47

[Table/Fig-2]: Baseline demographic and clinical characteristics of patients. Chi-square test; All other comparisons using an Independent t-test

Parameters	Time point	Group A (5 µg) (Mean±SD)	Group B (10 µg) Mean±SD	t-value	p-value
SBP (mmHg)	Baseline	128.5±12.3	126.8±11.9	0.62	0.54
	15 min	118.2±10.5	116.5±11.2	0.71	0.48
	30 min	120.4±9.8	118.8±10.3	0.71	0.50
	2 h	122.6±10.1	121.3±9.7	0.58	0.57
	6 h	125.2±11.4	124.1±10.9	0.44	0.66
	12 h	127.8±12.1	126.5±11.6	0.49	0.62
DBP (mmHg)	Baseline	78.3±8.5	77.2±8.1	0.59	0.56
	15 min	72.4±7.2	71.1±7.8	0.77	0.45
	30 min	73.8±6.9	72.5±7.3	0.82	0.42
	2 h	75.2±7.4	74.3±7.1	0.55	0.59
	6 h	76.8±8.2	75.9±7.9	0.50	0.62
	12 h	78.1±8.4	77.4±8.2	0.38	0.71
MAP (mmHg)	Baseline	95.0±9.2	93.7±8.8	0.66	0.51
	15 min	87.7±8.1	86.2±8.4	0.81	0.42
	30 min	89.3±7.8	87.9±8.0	0.79	0.43
	2 h	91.0±8.3	89.9±7.9	0.61	0.54
	6 h	92.9±9.3	91.9±8.9	0.50	0.62
	12 h	94.7±9.6	93.8±9.2	0.43	0.67
Heart Rate (bpm)	Baseline	82.5±10.2	80.8±9.8	0.76	0.45
	15 min	76.3±8.5	74.2±9.1	1.07	0.29
	30 min	77.8±8.9	75.6±8.6	1.12	0.27
	2 h	78.5±9.2	77.1±8.8	0.69	0.50
	6 h	80.2±9.8	79.3±9.4	0.42	0.68
	12 h	81.8±10.1	80.5±9.7	0.58	0.57
SpO_2 (%)	Baseline	99.1±0.6	99.0±0.7	0.70	0.49
	15 min	98.8±0.8	98.7±0.9	0.53	0.60
	30 min	98.9±0.7	98.8±0.8	0.60	0.55
	2 h	99.0±0.6	98.9±0.7	0.70	0.49
	6 h	99.1±0.6	99.0±0.6	0.75	0.46
	12 h	99.2±0.5	99.1±0.6	0.82	0.41

[Table/Fig-3]: Comparison of haemodynamic parameters between groups. SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; bpm: beats per minute. Statistical analysis: Independent t-test. All haemodynamic parameters remained stable with no significant differences between groups at any time point (p-value >0.05).

Block Characteristics

Group B demonstrated significantly faster onset of sensory block to T8 level compared to group A, as shown in [Table/Fig-4]. The mean time to achieve T8 sensory block was 4.5±0.8 minutes in group B versus 5.8±1.2 minutes in group A (p-value=0.01). At 4 minutes, 68.4% of group B patients achieved T8 block compared to 36.8% in group A (p-value=0.01). By 6 minutes, 97.4% of group B versus 78.9% of group A had achieved an adequate sensory level (p-value=0.03).

Variables	Group A (5 µg) (n=38)	Group B (10 µg) (n=38)	t-value	p-value
Onset to T8 sensory block (min), (Mean±SD)	5.8±1.2	4.5±0.8	5.77	0.01*
Patients achieving T8 at 4 min, n (%)	14 (36.8)	26 (68.4)	–	0.01*
Patients achieving T8 at 6 min, n (%)	30 (78.9)	37 (97.4)	–	0.03*
Onset to complete motor block (min), (Mean±SD)	5.6±1.1	5.3±0.9	1.27	0.21
Peak sensory level (T6), n (%)	32 (84.2)	33 (86.8)	–	0.85
Time to peak sensory level (min), (Mean±SD)	12.5±2.8	11.2±2.4	2.21	0.04*
Duration of sensory block (min), (Mean±SD)	285±35	395±42	12.56	<0.001*
Duration of motor block (min), (Mean±SD)	245±32	325±38	10.12	<0.001*

[Table/Fig-4]: Characteristics of sensory and motor block.
Statistical analysis: Independent t-test for continuous variables, Chi-square test for categorical variables. *p-value <0.05 is considered statistically significant. Group B demonstrated significantly faster onset and longer duration of sensory block compared to group A.

Motor block onset was comparable between groups. The mean time to achieve complete motor block (modified Bromage score 3) was 5.6±1.1 minutes in group A and 5.3±0.9 minutes in group B (p-value=0.21). All patients in both groups achieved complete motor block by 8 minutes. The peak sensory level achieved was T6 in most patients, with no difference between groups (p-value=0.85).

Group B demonstrated significantly superior postoperative analgesia with lower VAS scores at all measured time points [Table/Fig-5]. At 2 hours, mean VAS was 1.5±1.2 in group B versus 3.2±1.5 in group A (p-value=0.01). At 4 hours, mean VAS was 2.1±1.4 in group B versus 4.3±1.6 in group A (p-value <0.001). At 6 hours, mean VAS was 2.8±1.5 in group B versus 5.2±1.7 in group A (p-value <0.001). At 8 hours, mean VAS was 3.5±1.6 in group B versus 4.8±1.8 in group A (p-value <0.001). This significant difference persisted until 8 hours postoperatively, with group B consistently showing approximately 1.3-2.4 points lower VAS scores compared to group A.

Time point	Group A (5 µg) (Mean±SD)	Group B (10 µg) (Mean±SD)	t-value	p-value
2 hours	3.2±1.5	1.5±1.2	5.56	0.01*
4 hours	4.3±1.6	2.1±1.4	6.48	<0.001*
6 hours	5.2±1.7	2.8±1.5	6.72	<0.001*
8 hours	4.8±1.8	3.5±1.6	3.44	<0.001*
10 hours	4.2±1.7	3.8±1.5	1.09	0.28
12 hours	3.9±1.6	3.6±1.4	0.87	0.39

[Table/Fig-5]: Postoperative Visual Analogue Scale (VAS) scores.
VAS: Visual Analogue Scale (0-10, where 0 = no pain and 10 = worst imaginable pain). Statistical analysis: Independent t-test. *p<0.05 considered statistically significant. Group B demonstrated significantly lower pain scores at 2, 4, 6, and 8 hours postoperatively.

Rescue Analgesia Requirements

The requirement for rescue analgesia differed dramatically between groups [Table/Fig-6]. At 6 hours postoperatively, 27 patients (71.05%) of group A required tramadol compared to only 1 patient (2.63%) in group B (p-value <0.001). Peak tramadol requirement in group B occurred at 8 hours, 25 patients (65.79), while group A had already required rescue analgesia much earlier.

Sedation

Sedation scores assessed using the Ramsay Sedation Scale were comparable between groups throughout the intraoperative and immediate postoperative period [Table/Fig-7]. Both groups achieved mild to moderate sedation (Ramsay score 2-3) without oversedation or respiratory depression. At 15 minutes after intrathecal injection, the mean Ramsay score was 2.4±0.6 in group A and 2.6±0.7 in group B (p-value=0.19). No patient in either group had a Ramsay score >4 at any time point. All patients remained easily arousable and cooperative throughout the study period.

Variables	Group A (5 µg) (n=38)	Group B (10 µg) (n=38)	t-value	p-value
Duration of analgesia (hours), (Mean±SD)	5.3±1.2	8.2±1.5	9.45	<0.001*
Patients requiring rescue analgesia at 6 h, n (%)	27 (71.05)	1 (2.63)	–	<0.001*
Patients requiring rescue analgesia at 8 h, n (%)	34 (89.47)	25 (65.79)	–	0.01*
Patients requiring rescue analgesia at 12 h, n (%)	36 (94.74)	31 (81.58)	–	0.08
Total tramadol consumed in 12 hours (mg), Mean±SD	135.5±22.4	98.2±18.7	8.13	<0.001*
Number of tramadol doses, Mean±SD	2.1±0.6	1.5±0.5	4.92	<0.001*

[Table/Fig-6]: Rescue analgesia requirements.
Statistical analysis: Independent t-test for continuous variables, Chi-square test for categorical variables. *p<0.05 considered statistically significant. Group B demonstrated significantly delayed requirement for rescue analgesia and reduced total opioid consumption compared to Group A.

Time point	Group A (5 µg) (Mean±SD)	Group B (10 µg) (Mean±SD)	p-value
15 minutes	2.4±0.6	2.6±0.7	0.19
30 minutes	2.3±0.5	2.5±0.6	0.13
1 hour	2.2±0.5	2.3±0.6	0.43
2 hours	2.1±0.4	2.2±0.5	0.35
4 hours	1.8±0.4	1.9±0.4	0.29
6 hours	1.7±0.5	1.8±0.4	0.36

[Table/Fig-7]: Ramsay sedation scale scores.
Ramsay Sedation Scale: 1 = anxious/agitated; 2 = cooperative/tranquil; 3 = responds to commands; 4 = brisk response to stimulation; 5 = sluggish response; 6 = no response. Statistical analysis: Mann-Whitney U test. No significant differences in sedation levels were observed between groups. Both groups maintained appropriate sedation (scores 2-3) without oversedation.

Adverse Effects

The incidence of adverse effects was comparable between both groups [Table/Fig-8]. No cases of postoperative nausea and vomiting, respiratory depression, or significant bradycardia requiring intervention were observed in either group at any time point during the 12-hour observation period. Although 2 patients (5.26%) in group A and 3 patients (7.89%) in group B experienced mild pruritus, which resolved spontaneously without requiring treatment (p-value=0.64). No episodes of hypotension requiring vasopressor support occurred in either group. However, 3 patients (7.89%) in group A and 2 patients (5.26%) in group B reported urinary retention, which further required catheterisation (p-value=0.64). No patient developed any neurological complications, headache, or signs of infection at the injection site.

Adverse event	Group A (5 µg) n (%)	Group B (10 µg) n (%)	p-value
Pruritus (mild)	2 (5.26)	3 (7.89)	0.64
Urinary retention	3 (7.89)	2 (5.26)	0.64
Shivering	1 (2.63)	0 (0)	0.31
Total adverse events	6 (15.79)	5 (13.16)	0.74

[Table/Fig-8]: Incidence of adverse events.
Statistical analysis: Fisher's exact test for categorical variables. No significant differences in the incidence of adverse events were observed between groups. All adverse events were mild and self-limiting or easily managed. The excellent safety profile was comparable between both doses of dexmedetomidine.

DISCUSSION

Haemodynamic parameters, including SBP, DBP, MAP, and HR remained stable in both groups throughout the study period, with no significant differences at any time point. Oxygen saturation was consistently maintained between 98-100% in both groups with no episodes of desaturation. Sedation scores indicated

mild to moderate sedation (Ramsay 2-3) without oversedation or respiratory depression. The incidence of adverse effects was low and comparable between groups, with only mild pruritus and urinary retention observed in a small number of patients. The current findings are consistent with previous studies demonstrating dose-dependent analgesic effects of intrathecal dexmedetomidine. Kanazi JE et al., reported that low-dose dexmedetomidine (3 mcg) added to bupivacaine spinal block significantly prolonged the duration of sensory and motor block [10]. Al-Mustafa MM et al., demonstrated that dexmedetomidine (5 mcg) added to spinal bupivacaine for urological procedures resulted in prolonged sensory and motor block duration with stable haemodynamics [11]. The present study extends these findings by directly comparing two clinically relevant doses (5 mcg versus 10 mcg) with ropivacaine as the local anaesthetic. The 55% increase in analgesic duration observed in our 10 mcg group aligns with findings from Mahendru V et al., who compared intrathecal dexmedetomidine, clonidine, and fentanyl as adjuvants to hyperbaric bupivacaine [12]. They reported that dexmedetomidine provided the longest duration of analgesia among the three adjuvants. Similarly, Kathuria S et al., demonstrated that dexmedetomidine as an adjuvant to ropivacaine in supraclavicular brachial plexus block provided superior analgesia compared to control groups [13]. The enhanced analgesic effect of the higher dose can be attributed to increased α2-receptor activation in the spinal cord, producing neuronal hyperpolarisation and inhibition of nociceptive transmission [16,17]. The use of hyperbaric ropivacaine in the current study may explain the greater analgesic advantage compared with isobaric formulations used elsewhere, as baricity influences the spread of local anaesthetic within the cerebrospinal fluid [18,19].

The present study demonstrated that the 10 mcg group achieved significantly faster sensory block onset to T8 level (4.5±0.8 minutes versus 5.8±1.2 minutes), with 68.4% of patients in the 10 mcg group achieving T8 block at 4 minutes compared to 36.8% in the 5 mcg group. These findings are consistent with Al-Mustafa MM et al., who reported faster onset of sensory block with dexmedetomidine as an adjuvant [11]. The mechanism underlying this enhanced onset may involve facilitation of local anaesthetic action through α2A-adrenoceptor activation, as demonstrated by Yoshitomi T et al., [20]. Motor block onset was comparable between groups (5.3±0.9 minutes versus 5.6±1.1 minutes), suggesting that the dose increment primarily affects sensory rather than motor pathways. However, the duration of motor block was significantly longer in the 10 mcg group (325±38 minutes versus 245±32 minutes). This preferential sensory enhancement is clinically beneficial as it ensures adequate analgesia without excessive motor impairment, facilitating earlier mobilisation [8]. Singh AK et al., similarly reported that intrathecal dexmedetomidine provided dose-dependent prolongation of sensory block with less pronounced effects on motor block duration [8].

Haemodynamic parameters remained stable in both groups throughout current study, corroborating the findings of Farokhmehr L et al., and Gupta R et al., that intrathecal dexmedetomidine up to 10 mcg does not increase the risk of hypotension or bradycardia [15,21]. The present study found no episodes requiring intervention for hypotension or bradycardia in either group. This haemodynamic stability can be attributed to minimal systemic absorption of intrathecally administered dexmedetomidine and its predominantly spinal site of action [22,23]. Afonso J and Reis F reviewed the role of dexmedetomidine in anaesthesia and intensive care, noting that while systemic administration produces dose-dependent sympatholytic effects, intrathecal administration at doses up to 10 mcg maintains haemodynamic stability due to limited systemic exposure [22]. Similarly, Abdallah FW et al., in their systematic review and meta-analysis reported that dexmedetomidine as an adjuvant to spinal anaesthesia did not significantly increase the incidence of clinically significant hypotension or bradycardia [23].

No respiratory depression occurred in the present study, and all patients maintained oxygen saturation of 98-100%, confirming the respiratory safety of dexmedetomidine compared with opioid adjuvants [12,14,24]. Belleville JP et al., demonstrated in their study of intravenous dexmedetomidine in humans that, unlike opioids, dexmedetomidine does not cause respiratory depression even at higher doses [24]. Esmaoğlu A et al., similarly reported no episodes of respiratory depression with intrathecal dexmedetomidine in their study of transurethral endoscopic surgery [14].

Sedation scores in the present study (Ramsay 2-3) indicated calm, cooperative patients throughout the observation period, consistent with the "cooperative sedation" profile described by Bajwa SJ et al., and Nelson LE et al., [25,26]. This unique sedation pattern, where patients are easily arousable and cooperative, is attributed to dexmedetomidine's action on endogenous sleep-promoting pathways, specifically through α2-adrenoceptor activation in the locus coeruleus [26]. No patient in either group had a Ramsay score exceeding 4, indicating the absence of deep sedation or oversedation.

Both groups in the current study were free from postoperative nausea and vomiting, reflecting dexmedetomidine's opioid-sparing and antiemetic properties [14,27,28]. Gan TJ et al., in their consensus guidelines for management of postoperative nausea and vomiting, noted that reducing opioid consumption through multimodal analgesia, including the use of adjuvants like dexmedetomidine, significantly decreases PONV incidence [27]. Kim JE et al., specifically demonstrated that intrathecal dexmedetomidine reduced the incidence of PONV in elderly patients undergoing transurethral prostatectomy [28]. Other adverse effects in the present study, such as pruritus (5.26% in group A and 7.89% in group B) and urinary retention (7.89% in group A and 5.26% in group B), were infrequent and mild. The low incidence of pruritus contrasts sharply with the high rates (30-100%) reported with neuraxial opioids [29]. No neurological complications, headache, or infection occurred in either group, supporting the safety profile of intrathecal dexmedetomidine at both doses studied.

The findings of this study have significant clinical implications for perioperative pain management in lower limb surgeries. The 10 mcg dose of intrathecal dexmedetomidine provides notable benefits in terms of extended analgesia and decreased opioid use, supporting current emphasis on opioid-sparing multimodal pain relief strategies [30]. Memtsoudis SG et al., [30] demonstrated that multimodal pain management approaches are linked to improved perioperative outcomes and reduced resource use utilisation. The delayed requirement for rescue analgesia in the 10 mcg group (mean duration 8.2 hours) allows for better postoperative comfort and may facilitate earlier mobilisation and rehabilitation, key factors in enhanced recovery after surgery protocols. The absence of respiratory depression and minimal sedation make this regimen particularly suitable for ambulatory and same-day discharge procedures.

Future research should explore several important aspects. Long-term follow-up studies are needed to assess the impact of intrathecal dexmedetomidine on chronic pain development, functional recovery, and patient satisfaction beyond the immediate postoperative period. Dose-finding studies investigating doses between 10 mcg and 15 mcg could help establish the optimal dose that maximises analgesia while maintaining the excellent safety profile observed in our study. Studies in special populations, including elderly patients, those with significant co-morbidities (ASA III-IV), and paediatric patients, would help extend the applicability of these findings. Additionally, comparative studies evaluating intrathecal dexmedetomidine with other neuraxial adjuvants in combination with different local anaesthetics could provide valuable information for optimising regional anaesthesia protocols.

Limitation(s)

The present single-centre study's generalisability may be limited to similar clinical settings. The 12-hour follow-up period, while adequate for assessing immediate postoperative outcomes, does not capture longer-term effects. The study population was restricted to healthy adults (ASA III), limiting applicability to high-risk patients or extremes of age.

CONCLUSION(S)

The present study demonstrates that 10 mcg intrathecal dexmedetomidine as an adjuvant to hyperbaric ropivacaine provides superior postoperative analgesia, faster sensory block onset, and reduced rescue analgesic requirements compared to 5 mcg, while maintaining comparable haemodynamic and respiratory safety. For patients undergoing lower limb surgeries under spinal anaesthesia, adding 10 mcg of dexmedetomidine to hyperbaric ropivacaine provides significantly better and longer-lasting pain relief than a 5 mcg dose, while being equally safe. This higher dose is the more effective choice for optimising postoperative analgesia and reducing opioid consumption.

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