Section: Emergency Medicine

Original Research Article

A PROSPECTIVE COMPARATIVE STUDY, TO ASSESS THE SENSORY AND MOTOR BLOCKADE ONSET AND DURATION OF ACTION OF INTRATHECAL ROPIVACAINE AND DEXMEDETOMIDINE AS AN ADJUVANT TO ROPIVACAINE IN OPEN INGUINAL HERNIA SURGERIES

D.Balaji[1], R.Arun Shankar[2], S.C.Ganesh Prabhu[3]

[1]Senior Resident, Department of Emergency Medicine, Government Medical College and Hospital, Krishnagiri, Tamilnadu, India.
[2]Associate Professor, Department of Anaesthesiology, Velammal Medical College Hospital and Research Institute, Madurai, Tamilnadu, India.
[3]Professor, Department of Anaesthesiology, Velammal Medical College Hospital, Madurai, Tamilnadu, India.

Received *: 07/09/2026*
Received in revised form *: 02/10/2026*
Accepted *: 15/10/2026*

Corresponding Author:
Dr. D.Balaji,
Senior Resident, Department of Emergency Medicine, Government Medical College and Hospital, Krishnagiri, Tamilnadu, India.
Email: durai.balaji27@gmail.com

DOI: 10.70034/ijmedph.2026.4.191

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (4); 1248-1253

ABSTRACT

Background: Open inguinal hernia surgery commonly uses spinal anaesthesia. Ropivacaine provides effective sensory and motor block, but block duration may be limited. Dexmedetomidine may improve block quality and analgesia. This study compared intrathecal ropivacaine alone with ropivacaine plus dexmedetomidine in patients.

Materials and Methods: This prospective randomised comparative study included 100 patients at Velammal Medical College Hospital and Research Institute from December 2022 to November 2023. Group R received 3 ml of 0.75% hyperbaric ropivacaine with 0.1 ml normal saline, while Group RD received 3 ml ropivacaine with 10 mcg dexmedetomidine. Spinal anaesthesia was given at L3–L4 using a 25-gauge Quincke needle. Sensory and motor block, heart rate, blood pressure, mean arterial pressure, rescue analgesia, and adverse effects were recorded.

Results: Age and weight were comparable with mean age 45.32 ± 4.43 versus 44.46 ± 4.23 years (P>0.05), and weight 53.52 ± 5.38 versus 53.42 ± 5.32 kg (P>0.05). Sensory onset was 4.12 ± 0.15 versus 3.30 ± 0.10 (P<0.005), and motor onset 5.10 ± 0.32 versus 4.20 ± 0.45 (P<0.001). Motor and sensory durations were 180.40 ± 24.3 versus 253.45 ± 24.2 and 196.40 ± 30.6 versus 275.34 ± 45.2 (P>0.05). Rescue analgesia was 205.35 ± 12.4 versus 295.35 ± 32.1 (P<0.001). MAP and heart rate showed P>0.05 through 120 minutes, P<0.001 at 150 minutes, and P<0.05 at 180 minutes. Side effects were P>0.05.

Conclusion: Dexmedetomidine improved block onset and duration and delayed rescue analgesia, while haemodynamics remained generally stable without significant differences in adverse effects.

Keywords: Ropivacaine; Dexmedetomidine; Spinal anaesthesia; Inguinal hernia; Sensory block; Motor block.

INTRODUCTION

Open inguinal hernia repair is a commonly performed lower abdominal surgery in which a safe and predictable anesthetic technique is important for satisfactory surgical conditions and recovery. Spinal anesthesia is widely used for procedures below the umbilicus because it provides effective sensory and motor blockade with good intraoperative analgesia. The technique also has advantages over general anesthesia in selected patients.1,2 An ideal spinal anesthetic should provide a rapid onset, adequate sensory and motor blockade, predictable recovery, effective postoperative analgesia, stable

hemodynamics, and minimal adverse effects. Thus, the aim is not only to achieve adequate surgical anesthesia but also to maintain a suitable balance between block onset, duration, motor recovery, analgesia, and safety. Ropivacaine has been used for spinal anesthesia and has been evaluated as a local anesthetic for lower limb surgery, including in regimens involving dexmedetomidine as an adjunct.[1,2]

Ropivacaine is a long-acting amide local anesthetic that produces sensory and motor blockade by inhibiting sodium-channel-mediated nerve conduction. Its use in spinal anesthesia has been studied clinically, particularly when a shorter-acting alternative to bupivacaine is desired.[2] However, the duration of ropivacaine spinal anesthesia may be limited, creating interest in strategies that can improve the duration of analgesia and blockade without unnecessarily delaying motor recovery.[2] Adjuvants have therefore been investigated with ropivacaine-based regional anesthesia, including dexmedetomidine in abdominal wall blocks for inguinal hernia surgery.[3,4] Although these studies were conducted with peripheral regional blocks rather than intrathecal anesthesia, they provide additional clinical evidence for the use of dexmedetomidine with ropivacaine in this surgical setting.[3,4]

Intrathecal adjuvants are used to improve the quality and duration of spinal anesthesia. Several agents, including opioids and α2-adrenergic agonists, have been investigated for this purpose. Among the non-opioid agents, dexmedetomidine has received particular attention because of its ability to prolong spinal analgesia and modify sensory and motor block characteristics.[5,6] Dexmedetomidine is a selective α2-adrenergic receptor agonist with analgesic and sympatholytic properties. Its action at the spinal level contributes to modulation of nociceptive transmission, and its use as an adjuvant to local anesthetics has been associated with prolonged spinal analgesia and blockade.[6-8] A meta-analysis found that intrathecal dexmedetomidine, when combined with local anesthetics including ropivacaine or bupivacaine, was associated with earlier sensory block and longer sensory blockade and analgesic duration compared with clonidine.[7] Similarly, a systematic review and meta-analysis found longer sensory and motor block duration and a prolonged pain-free period with dexmedetomidine.[9] However, the findings also showed substantial heterogeneity between studies, related to differences in local anesthetics, doses, surgical procedures, and outcome assessment.[9]

The hemodynamic effects of dexmedetomidine are also clinically important because of its sympatholytic action. Although concerns regarding hypotension and bradycardia warrant appropriate monitoring, the available meta-analytic evidence did not demonstrate a significant increase in these events compared with fentanyl.[9] A randomized trial of intrathecal dexmedetomidine with hyperbaric bupivacaine also reported prolonged sensory and motor blockade and improved postoperative analgesia, supporting its role as a spinal adjuvant.[6] More broadly, dexmedetomidine has been described as an adjuvant that can enhance the duration of spinal analgesia when combined with local anesthetics.[8]

The relevance of this question is particularly important in open inguinal hernia surgery, where adequate surgical anesthesia, effective postoperative analgesia, timely motor recovery, and hemodynamic stability are all desirable. Importantly, a randomized study involving patients showed that the route of dexmedetomidine administration influenced spinal anesthesia quality and hemodynamic responses, providing direct evidence for the relevance of intrathecal dexmedetomidine in this surgical population.[10] However, evidence evaluating intrathecal ropivacaine alone versus ropivacaine combined with intrathecal dexmedetomidine in open inguinal hernia surgery remains limited.

Aim

This study aimed to compare the clinical effects of intrathecal ropivacaine alone versus ropivacaine combined with dexmedetomidine.

MATERIALS AND METHODS

This prospective, randomised comparative study was conducted in 100 patients attending Velammal Medical College Hospital and Research Institute (VMCH&RI), MOT Complex, from December 2022 to November 2023. Ethical approval was obtained from the Institutional Ethics Committee (Ref. No. IECNO-VMCIEC/73/2022), and written informed consent was obtained from the participants before study initiation.

Inclusion and Exclusion criteria

This study included male patients aged 18–60 years, weighing 40–85 kg, with ASA physical status I and II, scheduled for elective open inguinal hernia surgery under spinal anesthesia. Patients with ASA physical status III or higher, age above 60 years, allergy to local anesthetics, hypertension, hepatic or renal impairment, cardiac disease, coagulopathy, use of β-blockers or long-term analgesic therapy, and those receiving drugs known to interact with the study medications were excluded from the study.

Materials

The materials used in the study included a 25-gauge Quincke spinal needle, 0.75% hyperbaric ropivacaine, dexmedetomidine, normal saline, intravenous fluids, oxygen, vasopressors, atropine, ondansetron, and equipment required for monitoring heart rate, blood pressure, and mean arterial pressure.

Methods

One hundred patients were randomised into Group R (n=50) (3 ml of 0.75% hyperbaric ropivacaine with 0.1 ml normal saline) and Group RD (n=50) (3 ml of 0.75% hyperbaric ropivacaine combined with 10 mcg (0.1 ml) dexmedetomidine). After aseptic precautions, spinal anesthesia was administered

intrathecally at the L3–L4 interspace using a 25-gauge Quincke spinal needle. After administration of the study drugs, the patients were immediately placed in the supine position.

Sensory block was assessed using a pinprick test with a 22-gauge hypodermic needle. The onset of sensory block was recorded when complete loss of pinprick sensation at the T10 dermatome was achieved. The duration of sensory block was recorded from the onset until the return of pinprick sensation to the T7–T8 sensory level, provided that the block was adequate for the duration of surgery. Motor block was assessed using the Bromage scale. The onset of motor block was recorded when Bromage grade 3 was achieved, and the duration was recorded until complete recovery of motor function.

Heart rate, blood pressure, and mean arterial pressure were recorded every 3 minutes after spinal anesthesia and periodically throughout the surgery. Hypotension, defined as a decrease in blood pressure or mean arterial pressure of more than 20% from baseline or MAP <60 mmHg, was treated with intravenous fluids, oxygen, and vasopressors as required. Bradycardia, defined as heart rate below 60 beats/minute, was treated with atropine 0.6 mg IV. The interval between subarachnoid block administration and the first request for supplemental analgesia was recorded as the time to rescue analgesia. Adverse effects such as nausea and vomiting were treated with ondansetron 4 mg IV as required.

Statistical Analysis

Data were presented as mean, standard deviation, frequency and percentage. Continuous variables were compared using the independent sample t-test. Categorical variables were compared using the Pearson chi-square test. Significance was defined by P values less than 0.05 using a two-tailed test. Data analysis was performed using IBM-SPSS version 20.0 (IBM-SPSS Statistics Inc., Chicago, IL).

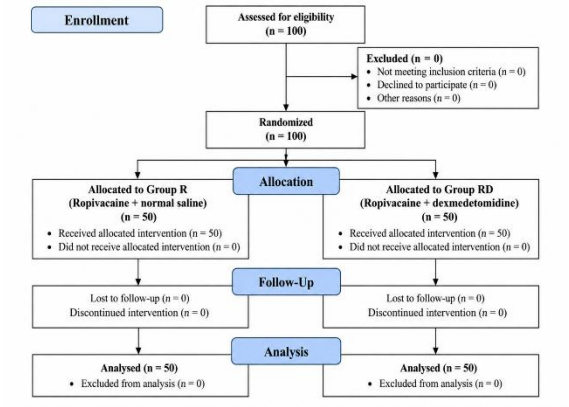


Figure 1: CONSORT Flowchart

RESULTS

Most patients were in the 50-60-year age group, accounting for 40% in Group R and 36% in Group RD. This was followed by the 40-50-year group (32% and 30%) and the 30-40-year group (28% and 34%), respectively. With respect to weight, the 55-60 kg category was the most common, with 34% of patients in Group R and 28% in Group RD. The remaining patients were distributed across the 60-65 kg (16% and 22%), 50-55 kg (18% and 20%), 45-50 kg (18% and 12%), and 40-45 kg (14% and 8%) categories. T8 was the sensory level in 80% of patients in Group R and 60% in Group RD, while T7 was observed in 20% and 40% (P>0.05). Most patients were ASA I with 86% in Group R and 90% in Group RD, whereas ASA II in 14% and 10% (P>0.05). [Table 1]

Table 1: Comparison of age, weight, sensory level, and ASA grade between Groups

		N (%)		P value
		Group R	Group RD	
Age (years)	30-40	14 (28%)	17 (34%)	-
	40-50	16 (32%)	15 (30%)	
	50-60	20 (40%)	18 (36%)	
Weight (kg)	40-45	7 (14%)	4 (8%)	-
	45-50	9 (18%)	6 (12%)	
	50-55	9 (18%)	10 (20%)	
	55-60	17 (34%)	14 (28%)	
	60-65	8 (16%)	11 (22%)	
Sensory level	T7	10 (20%)	20 (40%)	>0.05
	T8	40 (80%)	30 (60%)	
ASA grade	I	43 (86%)	45 (90%)	>0.05
	II	7 (14%)	5 (10%)	

The mean age was 45.32 ± 4.43 years in Group R and 44.46 ± 4.23 years in Group RD (P>0.05). Mean weight was also close between the groups, at 53.52 ± 5.38 kg and 53.42 ± 5.32 kg, respectively (P>0.05). Sensory block onset was 4.12 ± 0.15 in Group R compared with 3.30 ± 0.10 in Group RD (P<0.005). Similarly, motor block onset was 5.10 ± 0.32 and 4.20 ± 0.45, respectively (P<0.001). The duration of motor block until Bromage 0 was 180.40 ± 24.3 in Group R and 253.45 ± 24.2 in Group RD. Sensory block duration was 196.40 ± 30.6 and 275.34 ± 45.2, respectively, with the reported difference being non-significant (P>0.05). The time to rescue analgesia was 205.35 ± 12.4 in Group R and 295.35 ± 32.1 in Group RD (P<0.001). [Table 2]

Table 2: Comparison of age, weight, onset and duration of sensory and motor blockade, and time to rescue analgesia between Groups

		Mean ± SD		P Value
		Group R	Group RD	
Age (in years)		45.32 ± 4.43	44.46 ± 4.23	>0.05
Weight (Kg)		53.52 ± 5.38	53.42 ± 5.32	>0.05
Onset of	Sensory block	4.12 ± 0.15	3.30 ± 0.10	<0.005
	Motor block	5.10 ± 0.32	4.20 ± 0.45	<0.001
Duration of	Motor block (Bromage 0)	180.40 ± 24.3	253.45 ± 24.2	>0.05
	Sensory block	196.40 ± 30.6	275.34 ± 45.2	>0.05
Rescue analgesia (time of requirement)		205.35 ± 12.4	295.35 ± 32.1	<0.001

MAP at baseline was 90.57 ± 8.99 mmHg in Group R and 83.53 ± 8.48 mmHg in Group RD (P>0.05). At 3, 15, 30, 45, 60, 90, and 120 min, the values were 87.34 ± 7.45 and 80.12 ± 7.38; 80.11 ± 6.56 and 72.23 ± 6.48; 73.53 ± 4.56 and 71.53 ± 7.12; 75.57 ± 7.78 and 69.12 ± 4.65; 78.54 ± 6.32 and 61.18 ± 6.56; 75.57 ± 4.56 and 67.13 ± 8.67; and 75.57 ± 4.78 and 66.32 ± 7.67 mmHg in Group R and Group RD, respectively (P>0.05). At 150 min, MAP was 80.57 ± 5.90 and 70.12 ± 5.67 mmHg (P<0.001), while at 180 min it was 80.57 ± 4.56 and 76.24 ± 3.56 mmHg (P<0.05). [Table 3]

Table 3: Comparison of mean arterial pressure at different time points after spinal anaesthesia between Groups

		Mean arterial pressure (mmHg) (Mean ± SD)		P value
		Group R	Group RD	
Time after spinal anaesthesia	Baseline (0 min)	90.57 ± 8.99	83.53 ± 8.48	>0.05
	3 min	87.34 ± 7.45	80.12 ± 7.38	>0.05
	15 min	80.11 ± 6.56	72.23 ± 6.48	>0.05
	30 min	73.53 ± 4.56	71.53 ± 7.12	>0.05
	45 min	75.57 ± 7.78	69.12 ± 4.65	>0.05
	60 min	78.54 ± 6.32	61.18 ± 6.56	>0.05
	90 min	75.57 ± 4.56	67.13 ± 8.67	>0.05
	120 min	75.57 ± 4.78	66.32 ± 7.67	>0.05
	150 min	80.57 ± 5.90	70.12 ± 5.67	<0.001
	180 min	80.57 ± 4.56	76.24 ± 3.56	<0.05

Heart rate at baseline was 90.57 ± 8.99 beats/min in Group R and 87.34 ± 8.48 beats/min in Group RD (P>0.05). At 3, 15, 30, 45, 60, 90, and 120 min, the values were 80.57 ± 843 and 84.44 ± 6.75; 75.51 ± 8.45 and 73.56 ± 6.55; 78.67 ± 8.99 and 63.54 ± 3.56; 74.34 ± 8.67 and 63.56 ± 6.76; 76.11 ± 8.43 and 65.34 ± 9.06; 76.54 ± 7.65 and 64.23 ± 5.67; and 73.37 ± 7.56 and 68.34 ± 4.56 beats/min, respectively (P>0.05). At 150 min, the values were 70.59 ± 6.78 and 65.12 ± 8.69 beats/min (P<0.001), and at 180 min, 75.17 ± 4.56 and 67.45 ± 8.45 beats/min (P<0.05). [Table 4]

Table 4: Comparison of heart rate at different time points after spinal anesthesia between Groups

		Heart Rate (beats/min) (Mean ± SD)		P value
		Group R	Group RD	
Time after spinal anaesthesia	Baseline (0 min)	90.57 ± 8.99	87.34 ± 8.48	>0.05
	3 min	80.57 ± 843[a]	84.44 ± 6.75	>0.05
	15 min	75.51 ± 8.45	73.56 ± 6.55	>0.05
	30 min	78.67 ± 8.99	63.54 ± 3.56	>0.05
	45 min	74.34 ± 8.67	63.56 ± 6.76	>0.05
	60 min	76.11 ± 8.43	65.34 ± 9.06	>0.05
	90 min	76.54 ± 7.65	64.23 ± 5.67	>0.05
	120 min	73.37 ± 7.56	68.34 ± 4.56	>0.05
	150 min	70.59 ± 6.78	65.12 ± 8.69	<0.001
	180 min	75.17 ± 4.56	67.45 ± 8.45	<0.05

Hypotension reported in none of patients in Group R and 4 patients (8%) in Group RD. Bradycardia was not seen in Group R but occurred in 1 patient (2%) in Group RD. Nausea and vomiting were absent in Group R and occurred in 2 patients (4%) in Group RD. The reported difference in side effects was not significant (P>0.05). [Table 5]

Table 5: Comparison of postoperative side effects between Groups

		N (%)		P value
		Group R	Group RD	
Side effect	Hypotension (MAP < 60)	0	4 (8%)	>0.05
	Bradycardia (HR < 50 beats/min)	0	1 (2%)	
	Nausea and vomiting	0	2 (4%)	

DISCUSSION

In our study, the maximum sensory level was T8 in 80% of Group R and 60% of Group RD, while T7 was seen in 20% and 40%, respectively (P>0.05). Dolma et al. also reported similar sensory spread, with a median level of T6 [IQR T6-T8] in the dexmedetomidine group and T8 [IQR T6-T8] in the ropivacaine group (P=0.344).[11] In our study, sensory block started earlier with dexmedetomidine, at 3.30 ± 0.10 versus 4.12 ± 0.15 min (P<0.005). Singh et al. reported 5.42 ± 1.48 versus 12.52 ± 3.22 min (P<0.0001), while Sharath et al. reported 2.69 ± 0.53 versus 3.31 ± 0.63 min (P<0.001).12,13 However, Dolma et al. reported 9.94 ± 6.7 versus 7.60 ± 4.7 min (P=0.123), while Shashikala et al. reported 1.673 ± 0.567 versus 1.763 ± 0.420 min (P=0.783).[11,14] These differences may be related to variations in drug dose, baricity, concentration, and the method used to assess block onset.

In our study, both groups were similar at baseline. The mean age was 45.32 ± 4.43 years in Group R and 44.46 ± 4.23 years in Group RD (P>0.05), while the mean weight was 53.52 ± 5.38 and 53.42 ± 5.32 kg, respectively (P>0.05). ASA I patients accounted for 86% and 90%, while ASA II patients accounted for 14% and 10%, respectively. Sharath et al. reported lower-limb surgery reported mean ages of 39.69 ± 11.54 years in the dexmedetomidine group and 40.26 ± 12.30 years in the ropivacaine group (P=0.842).13

In our study, motor block also started earlier with dexmedetomidine, at 4.20 ± 0.45 versus 5.10 ± 0.32 min (P<0.001). Shashikala et al. reported 1.59 ± 0.59 versus 2.07 ± 0.20 min (P<0.001), while Sharath et al. reported 4.54 ± 0.95 versus 5.37 ± 0.88 min (P<0.001).[13,14] Thus, the earlier motor onset in our study was similar to these findings.

In our study, motor block lasted 180.40 ± 24.3 min in Group R and 253.45 ± 24.2 min in Group RD. Zhang et al. reported 232.70 ± 93.29 min with ropivacaine alone and 353.60 ± 137.28, 350.57 ± 118.01, and 404.67 ± 112.83 min with 5, 7.5, and 10 µg dexmedetomidine, respectively (P<0.01).[15] Shashikala et al. reported 319.57 ± 64.752 versus 183.93 ± 35.252 min (P<0.001), while Singh et al. reported regression to L4 at 351.28 ± 31.54 versus 167.00 ± 14.21 min (P<0.001).[12,14] The longer motor block seen with dexmedetomidine in these studies was in the same direction as our findings.

In our study, sensory block lasted 196.40 ± 30.6 min with ropivacaine alone and 275.34 ± 45.2 min with dexmedetomidine (P>0.05). Dolma et al. reported 202.90 ± 50.2 versus 157.33 ± 31.6 min (P<0.001).[11] Mohanty et al. reported regression to S2 at 330.60 ± 22.56 versus 186.00 ± 18.87 min (P<0.001), while Zhang et al. also reported longer sensory regression with 5, 7.5, and 10 µg dexmedetomidine (P<0.001).[15,16] These findings were in the same direction as the longer sensory block observed in our study.

In our study, rescue analgesia was required at 205.35 ± 12.4 min in Group R and 295.35 ± 32.1 min in Group RD (P<0.001). Dolma et al. reported 265.16 ± 71.4 versus 203.67 ± 35.6 min (P<0.001).[11] Shashikala et al. reported 356.67 ± 63.022 versus 197.67 ± 37.605 min (P<0.001), while Mohanty et al. reported 348.00 ± 23.02 versus 207.60 ± 17.23 min (P<0.001).[14,16] These findings support the longer postoperative analgesia seen after adding dexmedetomidine in our study.

In our study, hypotension occurred in 0 versus 4 patients (8%) (P>0.05). Zhang et al. reported 60.0% versus 56.7%, 66.7%, and 60.0% with 5, 7.5, and 10 µg dexmedetomidine (P=0.938), while Mohanty et al. reported 1 versus 2 patients. Bradycardia occurred in 0 versus 1 patient (2%).[15,16] Dolma et al. reported 9.7% versus 6.6% (P=1.000), Zhang et al. reported 13.3%, 30.0%, and 16.7% versus 16.7% (P=0.373), and Mohanty et al. reported 1 versus 2 patients. Nausea and vomiting occurred in 0 versus 2 patients (4%) (P>0.05).[11,15,16] Dolma et al. reported 9.7% versus 3.3% (P=0.612); Zhang et al. reported nausea in 30.0%, 26.7%, and 30.0% versus 40.0% (P=0.053), and vomiting in 16.7%, 20.0%, and 16.7% versus 13.3% (P=0.233). Mohanty et al. reported nausea in 2 patients in each group and no vomiting.[11,15,16] Thus, our study showed faster sensory and motor block onset, longer blockade, and a later need for rescue analgesia with dexmedetomidine, while hypotension, bradycardia, and nausea or vomiting remained uncommon.

CONCLUSION

This study showed dexmedetomidine addition to intrathecal ropivacaine improved the quality of spinal anaesthesia in patients undergoing open inguinal hernia surgery. It produced a faster onset of both sensory and motor block and prolonged the duration of blockade. The need for rescue analgesia was also delayed. Haemodynamic changes were generally limited, with only minor episodes of hypotension and bradycardia, while nausea and vomiting were also reported in a small number of patients. Overall, dexmedetomidine was a useful adjuvant to intrathecal ropivacaine, providing faster block onset, longer-lasting anaesthesia, and prolonged postoperative analgesia without significant adverse effects.

REFERENCES

1. Sharma UD, Prateek, Tak H. Effect of addition of dexamethasone to ropivacaine on post-operative analgesia in ultrasonography-guided transversus abdominis plane block for inguinal hernia repair: A prospective, double-blind, randomised controlled trial. Indian J Anaesth 2018; 62:371–5. https://doi.org/10.4103/ija.IJA_605_17.
2. Lee SC, Kim TH, Choi SR, Park SY. No difference between spinal anesthesia with hyperbaric ropivacaine and intravenous dexmedetomidine sedation with and without intrathecal fentanyl: A randomized noninferiority trial. Pain Res Manag 2022; 2022:3395783. https://doi.org/10.1155/2022/3395783.

3. Vaghela SS, Chaurasiya MK, Prakash R, Khan MP. Ultrasound-guided quadratus lumborum block versus transversus abdominis plane block for laparoscopic inguinal hernia repair and appendicectomy using ropivacaine with dexmedetomidine. Cureus 2023;15: e33450. https://doi.org/10.7759/cureus.33450.
4. Koutalas I, Orfanou C, Theodoraki K. EP202 The effect of adding dexmedetomidine to the local anesthetic solution for ultrasonography-guided TAP block in inguinal hernia repair. A randomized controlled study. ePoster session 6 – Station 4, BMJ Publishing Group Ltd; 2023, p. A150.2-A150. https://doi.org/10.1136/rapm-2023-ESRA.263.
5. Singhal A, Taksande K. Role of adjuvants in enhancing the efficacy and duration of anesthesia blocks: A comprehensive review. Cureus 2024;16: e69880. https://doi.org/10.7759/cureus.69880.
6. Tawadros SIG, El Mageed AMSA, El Taher WAEMM, Salama MGM, Ansary Amin AMA. Dexmedetomidine vs. fentanyl as adjuvants to hyperbaric bupivacaine for unilateral spinal anesthesia in lower limb orthopedic surgeries: a randomized trial. Anaesth Pain Intensive Care 2023; 27:456–63. https://doi.org/10.35975/apic.v27i4.2191.
7. Zhang C, Li C, Pirrone M, Sun L, Mi W. Comparison of dexmedetomidine and clonidine as adjuvants to local anesthetics for intrathecal anesthesia: A meta-analysis of randomized controlled trials: Comparison between dexmedetomidine and clonidine. J Clin Pharmacol 2016; 56:827–34. https://doi.org/10.1002/jcph.666.
8. Rao S, Rajan N. Dexmedetomidine as an adjunct for regional anesthetic nerve blocks. Curr Pain Headache Rep 2021; 25:8. https://doi.org/10.1007/s11916-020-00926-z.
9. Sun S, Wang J, Bao N, Chen Y, Wang J. Comparison of dexmedetomidine and fentanyl as local anesthetic adjuvants in spinal anesthesia: a systematic review and meta-analysis of randomized controlled trials. Drug Des Devel Ther 2017; 11:3413–24. https://doi.org/10.2147/DDDT.S146092.
10. Khosravi F, Sadeghi N, Jarineshin H. The effect of dexmedetomidine on spinal anesthesia quality and hemodynamic changes in patients undergoing inguinal hernia repair surgery: intravenous versus intrathecal. Eur J Clin Pharmacol 2020; 76:923–8. https://doi.org/10.1007/s00228-020-02870-8.
11. Dolma L, Salhotra R, Rautela RS, Banerjee A. Isobaric ropivacaine with or without dexmedetomidine for surgery of neck femur fracture under subarachnoid block. J Anaesthesiol Clin Pharmacol 2018; 34:518–23. https://doi.org/10.4103/joacp.JOACP_226_18.
12. Singh AK, Kumar A, Kumar A, Prasad BK, Tiwary PK, Kumar R. A comparison of intrathecal dexmedetomidine and neostigmine as adjuvant to ropivacaine for lower limb surgeries: A double-blind randomized controlled study. Anesth Essays Res 2017; 11:987–92. https://doi.org/10.4103/aer.AER_62_17.
13. Sharath K, Vaka MD, Akshay C. Effect of intrathecal dexmedetomidine as an adjuvant to 0.75% hyperbaric ropivacaine on analgesia and block characteristics in patients undergoing elective lower-limb surgery under subarachnoid block. J Contemp Clin Pract 2026; 12:1025–36. https://doi.org/10.61336/jccp/26-8-124.
14. Shashikala TK, Sagar SS, Ramaliswamy P, Hudgi VV. Comparing effects of intrathecal adjuvants fentanyl and dexmedetomidine with hyperbaric ropivacaine in patients undergoing elective infraumbilical surgeries: A prospective, double-blind, clinical study. Anesth Essays Res 2019; 13:654–62. https://doi.org/10.4103/aer.AER_183_18.
15. Zhang Q, Xia L-Y, Liang W-D, Rao D-Y, Zhu P-P, Huang K-N, et al. Intrathecal dexmedetomidine combined with ropivacaine in cesarean section: A prospective randomized double-blind controlled study. Front Med (Lausanne) 2022; 9:922611. https://doi.org/10.3389/fmed.2022.922611.
16. Mohanty RR, Sahoo D, Sahu N, Baksi R. Dexmedetomidine as adjuvant to ropivacaine in subarachnoid block for postoperative analgesia in lower limb surgeries. J Cardiovasc Dis Res 2023; 14:850–6. https://www.jcdronline.org/index.php/JCDR/article/view/3622/3516.