

Comparison of Dexmedetomidine and Dexamethasone as Adjuvants to Ropivacaine in Ultrasound-Guided Peripheral Nerve Blocks

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Received: 01-10-2025 / Revised: 15-11-2025 / Accepted: 21-12-2025

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Conflict of interest: Nil

Abstract

Background: Peripheral nerve blocks are widely used to provide surgical anaesthesia and postoperative analgesia. Ropivacaine provides effective and relatively long-lasting regional anaesthesia; however, its duration following a single-shot block remains limited. Dexmedetomidine and dexamethasone have been investigated as adjuvants to prolong block duration and postoperative analgesia. This study compared their efficacy and safety as adjuvants to ropivacaine in ultrasound-guided peripheral nerve blocks.

Methods: This prospective randomized comparative study included 90 patients undergoing surgery under ultrasound-guided peripheral nerve block. Patients were randomly allocated into two groups of 45 each. Group DEX received ropivacaine with dexmedetomidine, while Group DXM received ropivacaine with dexamethasone. Sensory and motor block characteristics, duration of postoperative analgesia, rescue analgesic requirement, haemodynamic parameters, and adverse events were assessed.

Results: Sensory and motor block onset was significantly faster with dexmedetomidine than dexamethasone (8.24 ± 1.67 vs 10.02 ± 1.89 min and 10.36 ± 2.04 vs 12.11 ± 2.23 min, respectively; $p < 0.001$). Dexmedetomidine significantly prolonged sensory (758.42 ± 86.35 vs 681.27 ± 79.64 min) and motor blockade (642.18 ± 75.21 vs 584.73 ± 70.56 min; both $p < 0.001$). Postoperative analgesia was longer (812.36 ± 91.47 vs 721.64 ± 84.29 min; $p < 0.001$), and rescue analgesic consumption was lower with dexmedetomidine. Bradycardia, hypotension, and sedation occurred more frequently with dexmedetomidine, although no serious complications were observed.

Conclusion: Dexmedetomidine provided faster block onset, prolonged sensory and motor blockade, and superior postoperative analgesia compared with dexamethasone, although with greater haemodynamic and sedative effects.

Keywords: Dexmedetomidine; Dexamethasone; Ropivacaine; Peripheral Nerve Block; Ultrasound Guidance; Postoperative Analgesia.

DOI: 10.25258/ijcpr.18.1.300

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Introduction

Effective perioperative pain management is an important component of modern anaesthetic practice, as inadequate postoperative analgesia may contribute to increased sympathetic activity, delayed mobilization, impaired recovery, prolonged hospitalization, and reduced patient satisfaction. Although opioids are effective analgesics, their use is associated with adverse effects such as nausea, vomiting, sedation, pruritus, urinary retention, and respiratory depression. Consequently, regional anaesthesia and multimodal opioid-sparing strategies have gained increasing importance.[1] Peripheral nerve blocks provide targeted surgical anaesthesia and postoperative analgesia by depositing local anaesthetic near an individual nerve or nerve plexus, thereby temporarily

interrupting sensory and motor conduction.[2] Among upper-limb regional techniques, the supraclavicular brachial plexus block provides dense and reliable anaesthesia for surgeries involving the arm, elbow, forearm, and hand. The technique was first described by Kulenkampff in 1911.[3] Traditional landmark-guided approaches were associated with complications such as vascular puncture, pneumothorax, and nerve injury.[4] The introduction of ultrasound guidance has improved block precision by permitting direct visualization of neural structures, blood vessels, needle advancement, and local anaesthetic spread, thereby increasing safety and reliability. Despite technical advances, the duration of analgesia following a single-shot peripheral nerve block

remains limited by the pharmacological duration of the local anaesthetic. [5] Once the block wears off, patients may develop breakthrough pain requiring systemic analgesics. Prolonging postoperative analgesia without increasing adverse effects therefore remains an important objective in regional anaesthesia.[6] Ropivacaine is a long-acting amide local anaesthetic commonly used for peripheral nerve blocks because it provides effective sensory blockade with relatively less motor blockade and has a more favourable cardiovascular and central nervous system safety profile than bupivacaine. However ropivacaine alone may not provide sufficiently prolonged postoperative analgesia.

Addition of pharmacological adjuvants to local anaesthetic solutions is therefore frequently employed to improve block quality, accelerate onset, prolong sensory and motor blockade, and reduce rescue analgesic requirements.[7] Dexmedetomidine is a highly selective α_2 -adrenergic receptor agonist with analgesic, sedative, anxiolytic, and sympatholytic properties. Its peripheral actions include inhibition of nerve action potentials, modulation of hyperpolarization-activated cation currents, reduced norepinephrine release, and local vasoconstriction.

When added to local anaesthetics, dexmedetomidine may accelerate block onset and prolong sensory and motor blockade as well as postoperative analgesia. However, dose-dependent bradycardia, hypotension, and sedation may occur.[8]

Dexamethasone is a long-acting corticosteroid that has also been widely evaluated as a peripheral nerve block adjuvant. Its proposed mechanisms include suppression of inflammatory mediators, inhibition of nociceptive C-fibre transmission, modulation of potassium channels, and local vasoconstriction.[9] Dexamethasone has been shown to prolong sensory blockade and postoperative analgesia, generally without the sedative or sympatholytic effects associated with dexmedetomidine.

Comparative evidence regarding these two adjuvants remains inconsistent. Gao et al. reported that dexmedetomidine provided longer sensory blockade and better postoperative analgesia than dexamethasone when combined with ropivacaine.[10] Conversely observed comparable prolongation of block duration with dexmedetomidine and dexamethasone in ultrasound-guided axillary brachial plexus block. Such variability may be related to differences in block technique, dose, local anaesthetic concentration, and surgical setting.[11] Hence, the present study is undertaken to compare dexmedetomidine and dexamethasone as adjuvants

to ropivacaine in ultrasound-guided peripheral nerve blocks.

Materials and Methods

This prospective, randomized, comparative study was conducted in the Department of Anaesthesiology at a tertiary care teaching hospital

Study Population: A total of 90 adult patients scheduled to undergo elective upper-limb surgery under ultrasound-guided peripheral nerve block were enrolled in the study. Patients fulfilling the eligibility criteria were randomly allocated into two groups of 45 patients each.

- Group DEX (n = 45): Patients received ropivacaine with dexmedetomidine.
- Group DXM (n = 45): Patients received ropivacaine with dexamethasone.

Inclusion Criteria: Patients aged 18–65 years, of either sex, belonging to American Society of Anesthesiologists (ASA) physical status I or II, and scheduled for elective upper-limb surgery under ultrasound-guided peripheral nerve block were included. Only patients who provided written informed consent and were able to understand the postoperative pain assessment method were enrolled.

Exclusion Criteria: Patients who refused participation, had known hypersensitivity to any study drug, infection at the proposed injection site, coagulopathy or ongoing anticoagulant therapy, pre-existing neurological deficit involving the operative limb, significant cardiac, hepatic, renal, or respiratory disease, uncontrolled diabetes mellitus or hypertension, pregnancy, history of chronic opioid use, or inability to understand the pain scoring system were excluded.

Randomization and Blinding: Patients were randomly allocated to either group using a computer-generated randomization sequence. Group allocation was concealed using sequentially numbered, opaque, sealed envelopes. The study drug solution was prepared by an anaesthesiologist who was not involved in subsequent assessment of block characteristics or postoperative outcomes. The investigator responsible for recording observations remained unaware of the group allocation.

Pre-Anaesthetic Assessment: All patients underwent a detailed pre-anaesthetic evaluation before surgery. Demographic characteristics, relevant medical history, physical examination findings, and routine laboratory investigations were recorded. Patients were instructed regarding the visual analogue scale (VAS) for postoperative pain assessment, where a score of 0 represented no pain and 10 represented the worst imaginable pain.

Patients were kept fasting according to standard institutional guidelines. On arrival in the operating room, an intravenous line was secured and standard monitoring, including non-invasive blood pressure, electrocardiography, and peripheral oxygen saturation (SpO₂), was instituted. Baseline heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and SpO₂ were recorded.

Study Drug Preparation: Patients in Group DEX received ropivacaine with dexmedetomidine as the adjuvant, whereas patients in Group DXM received ropivacaine with dexamethasone as the adjuvant.

The concentration and total volume of ropivacaine were kept identical in both groups, and the final volume of the study solution was standardized by adding normal saline where required. The study solutions were prepared under strict aseptic precautions immediately before administration. Patients received 30 mL of 0.5% ropivacaine (150 mg) with dexmedetomidine 1 µg/kg perineurally for the ultrasound-guided peripheral nerve block.

Ultrasound-Guided Peripheral Nerve Block Technique: The peripheral nerve block was performed under strict aseptic precautions with the patient appropriately positioned according to the selected block. A high-frequency linear ultrasound transducer was used to identify the target neural structures and surrounding anatomical landmarks.

After skin preparation and infiltration with local anaesthetic, an appropriate block needle was advanced under real-time ultrasound guidance using an in-plane technique. After confirming correct needle-tip position and negative aspiration for blood, the prepared study solution was administered incrementally. The spread of local anaesthetic around the target neural structures was continuously visualized ultrasonographically.

Intermittent aspiration was performed during injection to minimize the possibility of inadvertent intravascular administration.

Assessment of Sensory Block: Sensory blockade was assessed at regular intervals after completion of the injection using the pinprick method in the dermatomal distribution of the nerves supplying the operative area.

Sensory blockade was graded as:

- 0 = Normal sensation;
- 1 = Reduced sensation to pinprick;
- 2 = Complete loss of sensation to pinprick.

The onset of sensory block was defined as the time interval between completion of study drug administration and development of complete sensory blockade.

The duration of sensory block was defined as the time from the onset of complete sensory blockade until return of normal sensation in the blocked nerve distribution.

Assessment of Motor Block: Motor blockade was assessed using an appropriate modified motor block scale based on movements supplied by the nerves involved in the block.

Motor block was graded as:

- 0 = Normal motor function;
- 1 = Reduced motor strength with preserved movement;
- 2 = Complete motor blockade.

The onset of motor block was defined as the interval between completion of drug administration and development of complete motor blockade. The duration of motor block was defined as the interval from complete motor blockade until recovery of normal motor function.

Intraoperative Monitoring: Heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, SpO₂, and respiratory rate were monitored throughout the intraoperative period. Haemodynamic parameters were recorded at baseline, after administration of the block, at predetermined intervals during surgery, and at the end of the procedure.

Any requirement for supplementary analgesia, sedation, or conversion to general anaesthesia was documented.

Postoperative Pain Assessment: Postoperative pain was assessed using the VAS score (0–10) at predetermined intervals after surgery. The duration of postoperative analgesia was calculated from the completion/onset of successful blockade until the patient first reported clinically significant pain requiring rescue analgesia.

The time to first rescue analgesic was recorded for each patient. Rescue analgesia was administered according to the institutional postoperative analgesia protocol when the predefined VAS threshold was reached. The total rescue analgesic requirement during the first 24 postoperative hours was also recorded.

Haemodynamic Parameters: Heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and SpO₂ were recorded at baseline and at predetermined intraoperative and postoperative intervals. Clinically significant bradycardia and hypotension were identified and treated according to standard institutional protocols.

Assessment of Adverse Events: All patients were observed for block-related and drug-related adverse events throughout the perioperative period.

Particular attention was paid to bradycardia, hypotension, excessive sedation, nausea, vomiting, respiratory depression, hypoxaemia, vascular puncture, neurological symptoms, local anaesthetic systemic toxicity, and other complications. Bradycardia was treated with intravenous atropine when clinically indicated, whereas hypotension was managed with intravenous fluids and vasopressor therapy as required. Any episode suggestive of local anaesthetic systemic toxicity was managed according to established institutional protocols.

Outcome Measures: The primary outcome of the study was the duration of postoperative analgesia following ultrasound-guided peripheral nerve block. The secondary outcomes included onset of sensory block, onset of motor block, duration of sensory block, duration of motor block, time to first rescue analgesic requirement, total postoperative rescue analgesic consumption, postoperative VAS scores, intraoperative and postoperative haemodynamic parameters, and incidence of adverse events.

Statistical Analysis: Data were entered into a Microsoft Excel spreadsheet and subsequently analysed using an statistical software package 27. Continuous variables were expressed as mean $\pm$ standard deviation (SD) for normally distributed data and as median with interquartile range (IQR) for non-normally distributed data. Categorical variables were expressed as frequency and percentage.

Normality of continuous data was assessed using the Shapiro–Wilk test. Normally distributed continuous variables between the two groups were compared using the independent Student's t-test, whereas non-normally distributed variables were compared using the Mann–Whitney U test. Categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate. Repeated haemodynamic and pain measurements were analysed using an appropriate repeated-measures analysis. A two-tailed p-value <0.05 was considered statistically significant.

Results

A total of 90 patients undergoing ultrasound-guided peripheral nerve block were included in the analysis, with 45 patients each in the dexmedetomidine group (Group DEX) and dexamethasone group (Group DXM).

The two groups were comparable with respect to baseline demographic and clinical characteristics. The mean age was 39.62 ± 12.18 years in Group DEX and 40.84 ± 11.73 years in Group DXM ($p=0.629$). There were no statistically significant differences in sex distribution, body mass index (BMI), ASA physical status, or duration of surgery between the groups (all $p>0.05$), indicating adequate baseline comparability (Table 1, Figure 1).

Table 1: Baseline demographic and clinical characteristics

Characteristic	Group DEX (n=45)	Group DXM (n=45)	p-value
Age (years), mean $\pm$ SD	39.62 ± 12.18	40.84 ± 11.73	0.629
Male, n (%)	28 (62.2)	27 (60.0)	0.829
Female, n (%)	17 (37.8)	18 (40.0)	
BMI (kg/m^2), mean $\pm$ SD	24.38 ± 2.91	24.71 ± 3.06	0.601
ASA I, n (%)	29 (64.4)	27 (60.0)	0.665
ASA II, n (%)	16 (35.6)	18 (40.0)	
Duration of surgery (min), mean $\pm$ SD	92.47 ± 19.82	94.16 ± 20.31	0.690

Values are presented as mean $\pm$ SD or n (%).

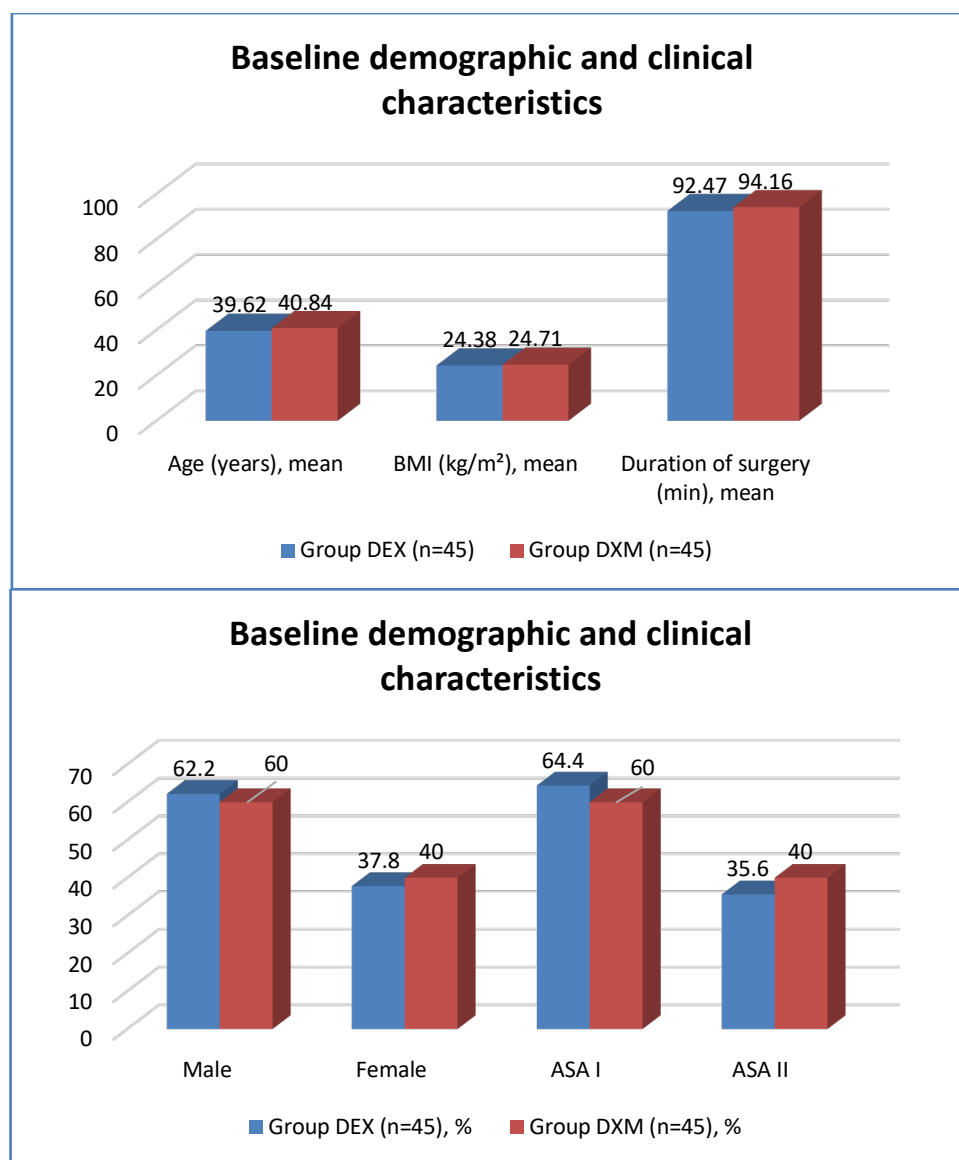


Figure 1: Baseline demographic and clinical characteristics

Characteristics of Sensory and Motor Blockade:

The onset of both sensory and motor blockade was significantly earlier in Group DEX. Mean sensory block onset was 8.24 ± 1.67 minutes in Group DEX compared with 10.02 ± 1.89 minutes in Group DXM ($p < 0.001$). Similarly, motor blockade developed at 10.36 ± 2.04 minutes and 12.11 ± 2.23 minutes, respectively ($p < 0.001$). The duration of

sensory blockade was significantly longer with dexmedetomidine (758.42 ± 86.35 minutes) than with dexamethasone (681.27 ± 79.64 minutes; $p < 0.001$).

Motor blockade was also prolonged in Group DEX (642.18 ± 75.21 minutes) compared with Group DXM (584.73 ± 70.56 minutes; $p < 0.001$) (Table 2, Figure 2).

Table 2: Comparison of sensory and motor block characteristics

Block characteristic	Group DEX (n=45)	Group DXM (n=45)	p-value
Sensory block onset (min)	8.24 ± 1.67	10.02 ± 1.89	<0.001
Motor block onset (min)	10.36 ± 2.04	12.11 ± 2.23	<0.001
Duration of sensory block (min)	758.42 ± 86.35	681.27 ± 79.64	<0.001
Duration of motor block (min)	642.18 ± 75.21	584.73 ± 70.56	<0.001

Values are expressed as mean $\pm$ SD; independent Student's t-test was used.

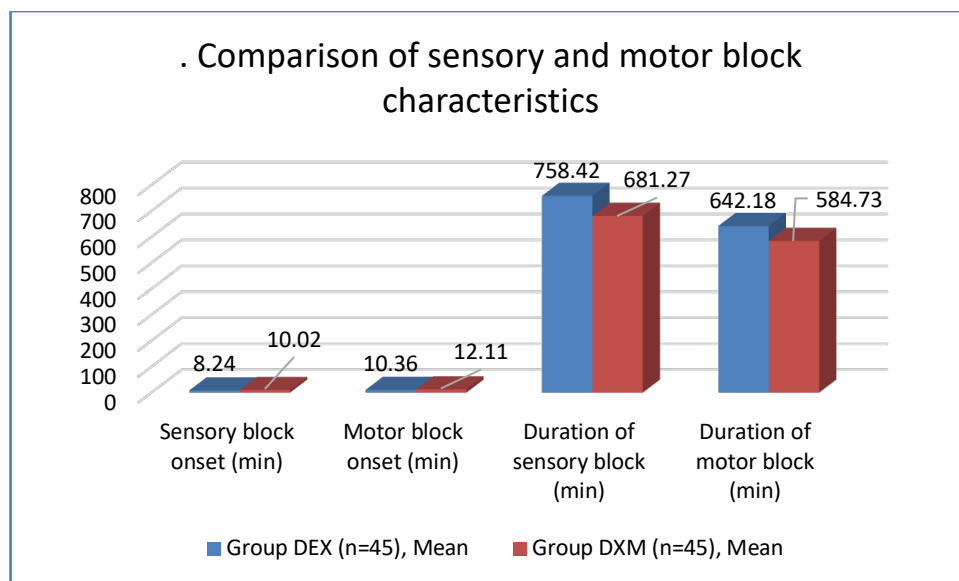


Figure 2: Comparison of sensory and motor block characteristic

The duration of postoperative analgesia was significantly greater in Group DEX than in Group DXM.

Patients receiving dexmedetomidine remained pain-free for 812.36 ± 91.47 minutes, compared with 721.64 ± 84.29 minutes among patients receiving dexamethasone ($p < 0.001$). Accordingly, the time to first rescue analgesic administration was

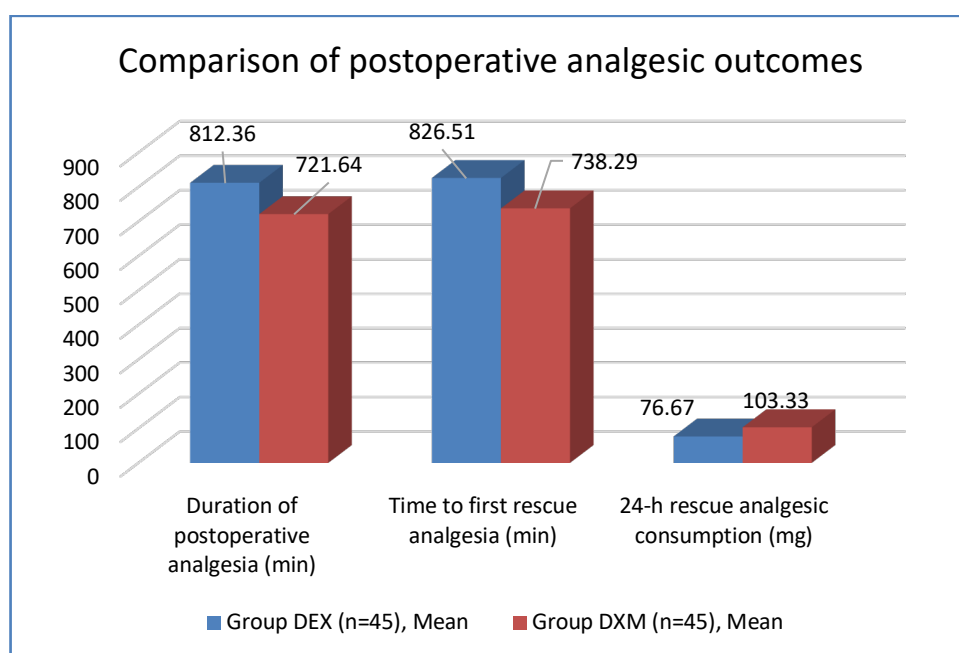
significantly longer in Group DEX (826.51 ± 94.36 minutes) than in Group DXM (738.29 ± 87.62 minutes; $p < 0.001$).

The cumulative rescue analgesic requirement during the first 24 postoperative hours was also lower in Group DEX (76.67 ± 30.52 mg) compared with Group DXM (103.33 ± 34.81 mg; $p < 0.001$) (Table 3, Figure3).

Table 3: Comparison of postoperative analgesic outcomes

Outcome	Group DEX (n=45)	Group DXM (n=45)	p-value
Duration of postoperative analgesia (min)	812.36 ± 91.47	721.64 ± 84.29	<0.001
Time to first rescue analgesia (min)	826.51 ± 94.36	738.29 ± 87.62	<0.001
24-h rescue analgesic consumption (mg)	76.67 ± 30.52	103.33 ± 34.81	<0.001
Patients requiring ≥ 2 rescue doses, n (%)	10 (22.2)	21 (46.7)	0.014

Values are presented as mean $\pm$ SD or n (%).



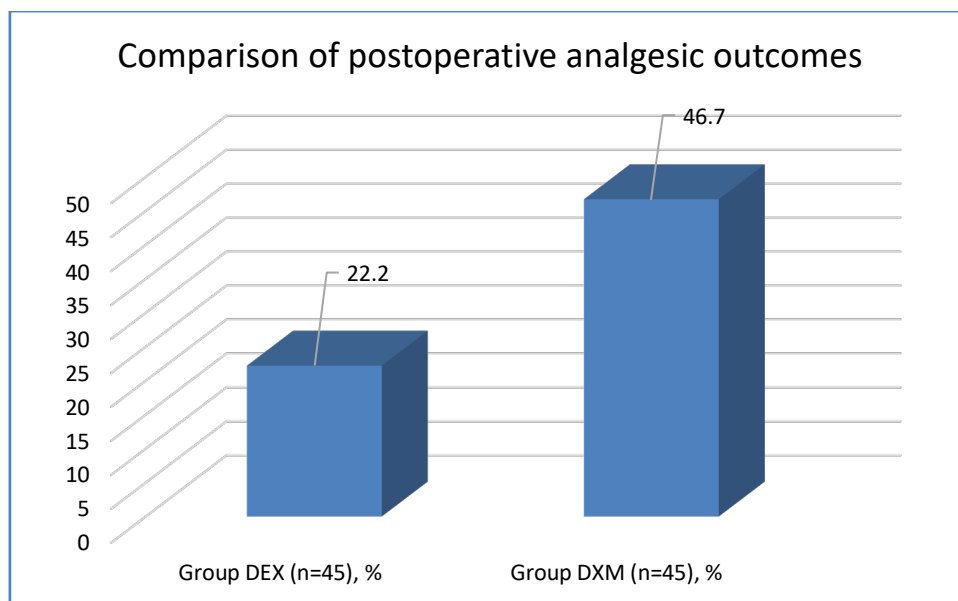


Figure 3: Comparison of postoperative analgesic outcomes

Postoperative VAS scores were low in both groups during the early postoperative period. No significant between-group difference was observed at 2 or 4 hours. However, pain scores were significantly lower in Group DEX at 6, 8, and 12 hours. The greatest difference was observed at 8

hours, when the mean VAS score was 1.71 ± 0.79 in Group DEX compared with 2.42 ± 0.92 in Group DXM ($p < 0.001$).

By 24 hours, pain scores were comparable between the groups ($p = 0.340$) (Table 4, Figure 4).

Table 4. Comparison of postoperative VAS scores

Postoperative time	Group DEX (n=45)	Group DXM (n=45)	p-value
2 h	0.42 ± 0.50	0.51 ± 0.51	0.399
4 h	0.76 ± 0.61	0.93 ± 0.65	0.203
6 h	1.11 ± 0.68	1.49 ± 0.73	0.012
8 h	1.71 ± 0.79	2.42 ± 0.92	<0.001
12 h	2.13 ± 0.81	2.56 ± 0.89	0.019
24 h	2.24 ± 0.83	2.40 ± 0.75	0.340

Values are expressed as mean $\pm$ SD.

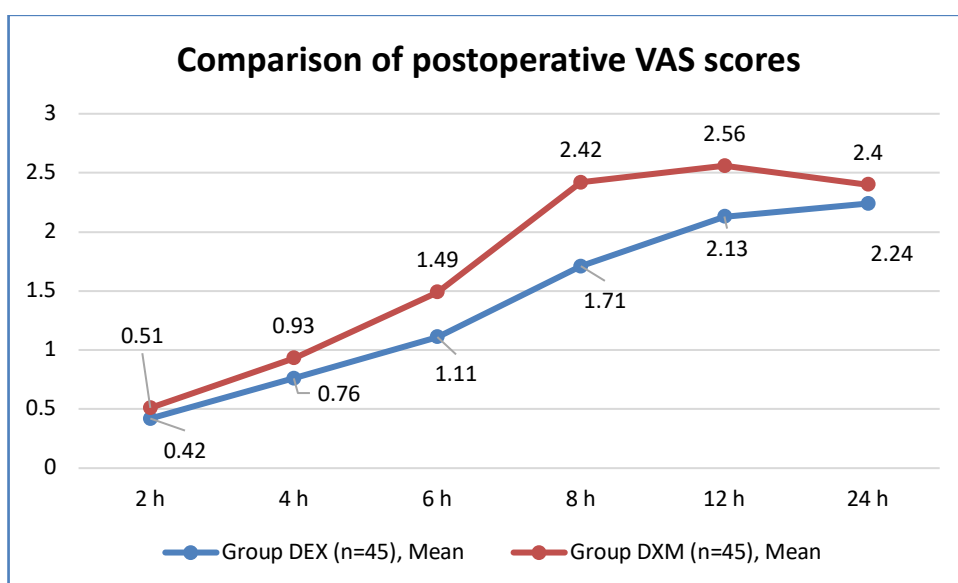


Figure 4: Comparison of postoperative VAS scores

Baseline haemodynamic parameters were comparable between the two groups. A modest reduction in heart rate and mean arterial pressure was observed following administration of dexmedetomidine. Mean heart rate at 30 minutes was significantly lower in Group DEX ($72.42 \pm$

7.18 beats/min) compared with Group DXM (77.31 ± 7.64 beats/min; $p=0.002$). Mean arterial pressure was also lower in Group DEX at 30 and 60 minutes. Nevertheless, the haemodynamic changes were generally clinically acceptable and did not result in major complications (Table 5).

Table 5: Comparison of perioperative haemodynamic parameters

Parameter	Group DEX (n=45)	Group DXM (n=45)	p-value
Baseline HR (beats/min)	79.82 ± 8.26	80.47 ± 8.13	0.707
HR at 30 min (beats/min)	72.42 ± 7.18	77.31 ± 7.64	0.002
HR at 60 min (beats/min)	70.84 ± 7.03	75.18 ± 7.27	0.005
Baseline MAP (mmHg)	92.64 ± 8.37	93.11 ± 8.06	0.786
MAP at 30 min (mmHg)	85.29 ± 7.52	89.02 ± 7.81	0.023
MAP at 60 min (mmHg)	84.82 ± 7.14	88.36 ± 7.49	0.024
SpO ₂ (%)	98.31 ± 0.82	98.38 ± 0.78	0.680

HR, heart rate; MAP, mean arterial pressure; SpO₂, peripheral oxygen saturation.

The overall incidence of adverse events was low in both groups. Bradycardia occurred more frequently in Group DEX (8.9%) than in Group DXM (2.2%), although the difference was not statistically significant ($p=0.361$). Hypotension occurred in 6.7% and 2.2% of patients, respectively. Sedation

was more frequent with dexmedetomidine (13.3% vs 2.2%), whereas postoperative nausea and vomiting were comparable between groups. No patient developed respiratory depression, local anaesthetic systemic toxicity, persistent neurological deficit, or pneumothorax (Table 6).

Table 6: Comparison of adverse events

Adverse event	Group DEX (n=45), n (%)	Group DXM (n=45), n (%)	p-value
Bradycardia	4 (8.9)	1 (2.2)	0.361
Hypotension	3 (6.7)	1 (2.2)	0.616
Sedation	6 (13.3)	1 (2.2)	0.110
Nausea/vomiting	2 (4.4)	3 (6.7)	>0.999

Discussion

The present study compared dexmedetomidine and dexamethasone as adjuvants to ropivacaine for ultrasound-guided peripheral nerve block in 90 patients, with 45 patients in each group. Dexmedetomidine was associated with earlier onset of sensory and motor blockade, longer duration of sensory and motor block, prolonged postoperative analgesia, delayed requirement for rescue analgesia, and lower 24-hour analgesic consumption.

However, it also produced greater reductions in heart rate and mean arterial pressure and was associated with more frequent bradycardia and sedation. The demographic and baseline clinical characteristics of the two groups were comparable, thereby reducing the likelihood of confounding. Sensory block onset was significantly faster with dexmedetomidine than dexamethasone (8.24 ± 1.67 vs 10.02 ± 1.89 min; $p<0.001$), as was motor block onset (10.36 ± 2.04 vs 12.11 ± 2.23 min; $p<0.001$). Singh et al. [12] reported a similar trend, with sensory onset times of 13.5 ± 4.1 and 15.6 ± 3.6 minutes and motor onset times of 17.0 ± 4.1 and 18.5 ± 3.7 minutes for dexmedetomidine and dexamethasone, respectively. In contrast, Lee et al. [13] found no significant difference in block onset

between the two adjuvants, possibly because of differences in block technique, drug doses, and local anaesthetic volume. The duration of sensory blockade in our study was significantly longer with dexmedetomidine (758.42 ± 86.35 min) than dexamethasone (681.27 ± 79.64 min; $p<0.001$). Motor block duration was also longer (642.18 ± 75.21 vs 584.73 ± 70.56 min; $p<0.001$). Mokkarala and Badugu similarly reported prolonged sensory and motor blockade with dexmedetomidine. Manzoor and Ara observed motor block durations of 853.5 ± 115 minutes with dexmedetomidine and 628.6 ± 135 minutes with dexamethasone, supporting the same direction of effect. Postoperative analgesia was significantly prolonged with dexmedetomidine in the present study (812.36 ± 91.47 vs 721.64 ± 84.29 min; $p<0.001$), while time to first rescue analgesia was also delayed (826.51 ± 94.36 vs 738.29 ± 87.62 min; $p<0.001$). Mokkarala and Badugu reported analgesic durations of 1142.47 ± 28.32 and 1045.95 ± 78.55 minutes, respectively. Similarly, Manzoor and Ara reported complete analgesia lasting 1374.2 ± 185.4 minutes with dexmedetomidine compared with 1084.2 ± 156.5 minutes with dexamethasone. Singh et al. [12] also observed longer analgesia with dexmedetomidine than dexamethasone. Rescue analgesic consumption was lower with

dexmedetomidine in our study (76.67 ± 30.52 vs 103.33 ± 34.81 mg; $p < 0.001$), and postoperative VAS scores were significantly lower at 6, 8, and 12 hours. These findings were consistent with reports of reduced postoperative analgesic requirement with dexmedetomidine. However, published evidence is not uniform. Lee et al. [13] found both adjuvants equally effective, while Albrecht et al. [14] in a meta-analysis of 49 trials involving 3019 patients, reported that dexamethasone prolonged analgesia by approximately 148 minutes compared with dexmedetomidine.

Other network meta-analyses have also ranked dexamethasone higher for prolongation of block duration, highlighting methodological heterogeneity across studies. Regarding safety, bradycardia occurred in 8.9% of dexmedetomidine patients compared with 2.2% in the dexamethasone group, while sedation occurred in 13.3% and 2.2%, respectively. These findings were consistent with the sympatholytic and sedative profile of dexmedetomidine.

Overall, dexmedetomidine provided superior block characteristics and postoperative analgesia in the present study, although this advantage was accompanied by greater haemodynamic and sedative effects. Differences among published studies may reflect variations in adjuvant dose, local anaesthetic concentration, anatomical block, and outcome definitions.

Conclusion

Both dexmedetomidine and dexamethasone were effective adjuvants to ropivacaine for ultrasound-guided peripheral nerve blocks. Dexmedetomidine provided faster onset, longer sensory and motor blockade, prolonged postoperative analgesia, and reduced rescue analgesic requirement compared with dexamethasone. However, it was associated with greater haemodynamic changes and sedation. Thus, dexmedetomidine appeared more effective for prolonging block duration, while dexamethasone offered a more haemodynamically stable alternative.

Limitations

The study was conducted at a single centre with a relatively small sample size, which may limit the generalizability of the findings. Postoperative pain assessment was subjective and could have been influenced by individual pain perception. Long-term neurological outcomes and potential delayed adverse effects of perineural adjuvants were not evaluated. Larger multicentre studies with longer follow-up are required to confirm these findings.

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