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Efficacy and Safety of Dexmedetomidine versus Midazolam as an Adjuvant to Lignocaine in Intravenous Regional Anaesthesia for Forearm Surgeries: A Randomized Controlled Trial

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ABSTRACT

Background: Intravenous regional anaesthesia (IVRA) is effective for short forearm surgeries, but lignocaine alone has limitations including slow onset, tourniquet pain, and short postoperative analgesia. Adjuvants such as dexmedetomidine and midazolam may enhance block characteristics. This study compared the efficacy and safety of dexmedetomidine versus midazolam as adjuvants to lignocaine in IVRA for forearm surgeries.

Methods: This prospective, randomized, double-blind trial included 100 ASA I/II patients (18–60 years) undergoing elective forearm surgeries. Group D (n=50) received 40 mL of 0.5% lignocaine with dexmedetomidine 0.5 µg/kg, while Group M (n=50) received 40 mL of 0.5% lignocaine with midazolam 50 µg/kg. Onset and regression times of sensory and motor blocks, duration of analgesia, tourniquet pain tolerance, fentanyl consumption, and adverse effects were recorded.

Results: Midazolam produced significantly faster onset of sensory block (5.02 vs. 6.64 min, p=0.0002) and motor block (8.62 vs. 10.94 min, p=0.0009). Dexmedetomidine significantly prolonged motor block regression (9.76 vs. 7.48 min, p=0.0017), duration of analgesia (93.34 vs. 84.50 min, p<0.0001), and tourniquet pain tolerance (27.58 vs. 20.70 min, p<0.0001). However, dexmedetomidine required higher postoperative fentanyl (55.14 vs. 50.82, p=0.0031). Adverse effects (bradycardia, hypotension) were comparable between groups (p>0.05).

Conclusion: Midazolam offers faster block onset, while dexmedetomidine provides longer analgesia and better tourniquet pain tolerance. Both adjuvants are safe. Choice should be individualized based on clinical priorities.

Keywords*: Dexmedetomidine; midazolam; intravenous regional anesthesia; IVRA; lignocaine; forearm surgery; tourniquet pain; postoperative analgesia.*

INTRODUCTION

Intravenous regional anaesthesia (IVRA), commonly known as Bier's block, remains a reliable and cost-effective technique for providing anaesthesia during short-duration surgical procedures on the upper and lower extremities [1,2]. First described by August Bier in 1908, IVRA involves the injection of a local anaesthetic into a vein of an exsanguinated limb isolated by a tourniquet, allowing the anaesthetic to diffuse into surrounding tissues to produce anaesthesia and analgesia [2]. It is particularly suitable for forearm and hand surgeries such as fracture reductions, tendon repairs, and soft tissue procedures due to its simplicity, rapid onset, and requirement for minimal equipment. The technique avoids the need for general anaesthesia, offers muscle relaxation, and facilitates early discharge, making it valuable in ambulatory settings [1,2].

Lignocaine (lidocaine) is the most commonly used local anaesthetic for IVRA, typically administered as a 0.5% solution at doses not exceeding 3 mg/kg [3]. It provides reliable sensory and motor blockade with a relatively high safety margin when proper exsanguination and tourniquet protocols are followed. However, plain lignocaine has notable limitations, including a relatively short duration of action, inadequate postoperative analgesia after tourniquet deflation, and the

frequent occurrence of tourniquet pain during prolonged procedures [4]. These drawbacks can compromise surgical conditions and patient comfort, particularly in forearm surgeries where tourniquet times may extend beyond 30–45 minutes. To overcome these limitations, various adjuvants have been investigated to enhance the quality and duration of blockade, reduce tourniquet pain, and improve perioperative analgesia while maintaining hemodynamic stability. Among these, alpha-2 agonists and benzodiazepines have shown promise [5,6]. Dexmedetomidine, a highly selective alpha-2 adrenergic agonist, possesses sedative, analgesic, and sympatholytic properties [6]. When added to lignocaine for IVRA, it accelerates the onset of sensory and motor blockade, prolongs anaesthesia duration, improves block quality, and significantly delays the onset of tourniquet pain [7]. Meta-analyses and clinical trials confirm that dexmedetomidine (typically 0.5–1 μg/kg) enhances intraoperative conditions and provides superior postoperative analgesia compared to lignocaine alone, with minimal systemic side effects due to the isolated limb circulation [5-8].

Midazolam, a short-acting benzodiazepine with GABA-A receptor agonist activity, has also been evaluated as an adjuvant in IVRA [9]. Studies demonstrate that the addition of midazolam (50 μg/kg) to lignocaine shortens the onset times of sensory and motor blocks, prolongs recovery times of these blocks, improves the overall quality of anaesthesia, and reduces both intraoperative tourniquet pain and postoperative analgesic requirements without significant adverse effects [9,10]. Its peripheral analgesic effects are attributed to modulation of nociceptive transmission and possible interaction with local anaesthetic mechanisms.

Direct comparisons between dexmedetomidine and midazolam as adjuvants to lignocaine in IVRA have been conducted. One randomized study found comparable effects between dexmedetomidine (1 μg/kg) and midazolam (50 μg/kg) regarding sensory and motor block characteristics, tourniquet tolerance, and postoperative analgesia, suggesting both agents are effective enhancements over plain lignocaine [10]. Other trials have explored these agents alongside others like ketamine, consistently highlighting their potential to optimize IVRA for upper extremity procedures [11].

Despite these advancements, optimal adjuvant selection remains an area of active research, as differences in onset speed, duration of analgesia, hemodynamic effects, and side-effect profiles warrant further evaluation in specific surgical contexts. Forearm surgeries present a unique setting where rapid, dense blockade and reliable post-deflation analgesia are critical for patient satisfaction and surgical efficiency [5,9]. The present study aims to evaluate and compare the benefits of dexmedetomidine versus midazolam as adjuvants to lignocaine in IVRA for forearm surgeries, focusing on block characteristics (onset and duration of sensory and motor blockade), quality of anaesthesia, tourniquet pain, postoperative analgesia, and safety profile. By providing evidence-based insights, this research seeks to guide clinical practice toward improved outcomes in regional anaesthesia for upper limb procedures.

The primary research question of this study is: In patients undergoing forearm surgeries under intravenous regional anaesthesia (IVRA) with lignocaine, does the addition of dexmedetomidine provide superior clinical benefits compared to midazolam as an adjuvant in terms of onset and duration of sensory and motor blockade, quality of anaesthesia, incidence and severity of tourniquet pain, postoperative analgesic requirements, and overall safety profile? We hypothesize that dexmedetomidine, owing to its highly selective α2-adrenergic agonist action with potent sedative, analgesic, and sympatholytic properties, will demonstrate significantly faster onset of sensory and motor blockade, prolonged duration of anaesthesia and analgesia, superior tourniquet tolerance, reduced postoperative pain scores, and lower analgesic consumption compared to midazolam, without increasing the incidence of adverse effects.

MATERIALS & METHODS

Study Design

This study was designed as a prospective, randomized, double-blind, parallel-group comparative clinical trial to evaluate the benefits of dexmedetomidine versus midazolam as adjuvants to lignocaine in intravenous regional anaesthesia for forearm surgeries. The study was conducted over a period of 18 months from January 2024 to June 2025. The study was carried out in the Department of Anaesthesiology at a tertiary care teaching hospital in eastern India. All information was gathered prospectively using a structured proforma that recorded demographic details, intraoperative parameters, and postoperative observations.

Study Population

Patients of “either sex, aged between 18 and 60 years, belonging to American Society of Anesthesiologists (ASA) physical status I or II, and scheduled for elective forearm surgeries lasting less than 90 minutes” were included in the study.
Patients with “a history of allergy to local anaesthetics or study drugs, peripheral vascular disease, Raynaud’s disease, scleroderma, hypertension, ischaemic heart disease, neurological disorders, hepatic or renal impairment, pregnancy, lactation, or those who refused to participate” were excluded from the study. Patients requiring tourniquet time longer than 90 minutes or conversion to general anaesthesia were also excluded.

Sample Size

With reported "total duration of analgesia of 93 in group D and 84 in Group M by Gupta et al. (2017) [10]," the minimum sample size required with 95% power, 12 SD, and 0.05 alpha value was calculated to be 46 patients per group. So, 100 patients were divided into 2 groups with 50 patients in each group.

Intervention

Patients were randomly allocated into two groups using a computer-generated random number table and sealed opaque envelopes. Group D received 40 ml of 0.5% lignocaine with dexmedetomidine 0.5 µg/kg, while Group M received 40 ml of 0.5% lignocaine with midazolam 50 µg/kg. The total volume was made up to 40 ml with normal saline in both groups. The study drugs were prepared by an anaesthesiologist not involved in patient management or data collection to maintain blinding.

Outcome Parameters

The primary outcome parameters were the onset time and duration of sensory and motor blockade. Secondary outcome parameters included quality of anaesthesia, incidence and severity of tourniquet pain, time to first analgesic request, total postoperative analgesic consumption in the first 24 hours, visual analogue scale (VAS) pain scores, and incidence of adverse effects such as hypotension, bradycardia, sedation, and nausea/vomiting.

Methodology

After obtaining written informed consent, a detailed pre-anaesthetic evaluation was performed. On the day of surgery, intravenous access was secured in the contralateral limb. Standard monitoring, including ECG, non-invasive blood pressure, pulse oximetry, and respiratory rate, was instituted. The affected upper limb was exsanguinated using an Esmarch bandage, and a double tourniquet was applied. After confirming tourniquet pressure at 250 mmHg above systolic blood pressure, the study drug solution was injected slowly over 90 seconds into a distal dorsal hand vein. Sensory blockade was assessed by the pinprick method in the dermatomal distribution every minute. Motor blockade was evaluated by asking the patient to flex and extend the fingers and wrist. Tourniquet pain was assessed using the VAS. After completion of surgery, the tourniquet was deflated gradually. Postoperative monitoring was continued for 24 hours.

Statistical Analysis

Data were analysed using GraphPad Prism 10. Continuous variables were expressed as "mean ± standard deviation" and compared using unpaired Student's t-test. Categorical variables were expressed as frequencies and percentages and compared using a chi-square test or Fisher's exact test. A p-value less than 0.05 was considered statistically significant.

Ethical Consideration

The study protocol was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants after explaining the purpose, procedure, risks, and benefits of the study. The study was conducted in accordance with the principles of the Declaration of Helsinki and Good Clinical Practice guidelines. Patient confidentiality was maintained throughout the study.

RESULTS

50 patients in Group D received "40 ml of 0.5% lignocaine with dexmedetomidine 0.5 µg/kg", while 50 patients in Group M received "40 ml of 0.5% lignocaine with midazolam 50 µg/kg."

The mean age was 36.52 ± 9.21 years in Group D and 38.17 ± 9.94 years in Group M (p=0.3913). The proportion of male patients was also similar between the groups (82.0% vs. 88.0%, p=0.5766). Mean BMI values did not differ significantly (25.29 ± 3.63 kg/m² vs. 24.53 ± 3.35 kg/m², p=0.2793), and the distribution of ASA physical status was comparable (p=0.5536). Furthermore, the mean duration of surgery was nearly identical between the groups (52.64 ± 9.92 minutes vs. 51.96 ± 11.37 minutes, p=0.7507). Since all p-values were greater than 0.05, the groups were well matched at baseline, minimizing the influence of confounding factors on study outcomes.

Table 1: Comparison of Baseline Demographic & Clinical Characteristics

Parameter	Group D (n=50)	Group M (n=50)	p-value
Age in Years, Mean ± SD	36.52 ± 9.21	38.17 ± 9.94	0.3913*
Male Gender, n (%)	41 (82.0)	44 (88.0)	0.5766**
BMI in kg/m^2, Mean ± SD	25.29 ± 3.63	24.53 ± 3.35	0.2793*
ASA Status-I, n (%)	45 (90.0)	42 (84.0)	0.5536**
ASA Status-II, n (%)	5 (10.0)	8 (16.0)	
Duration of Surgery in Minutes, Mean ± SD	52.64 ± 9.92	51.96 ± 11.37	0.7507*

**Unpaired t test; **Fisher's Exact Test*

Group M demonstrated a significantly faster onset of both sensory block (5.02 ± 1.61 minutes vs. 6.64 ± 2.48 minutes, p=0.0002) and motor block (8.62 ± 3.19 minutes vs. 10.94 ± 3.56 minutes, p=0.0009) compared to Group D. Regression of sensory block was longer in Group D (8.78 ± 3.64 minutes) than Group M (7.52 ± 3.45 minutes), but this difference was not statistically significant (p=0.0788). However, regression of motor block was significantly prolonged in Group D (9.76 ± 3.89 minutes) compared to Group M (7.48 ± 3.15 minutes, p=0.0017).

Table 2: Comparison of Block Characteristics

Parameter	Time in Minutes, Mean ± SD		p-value (Unpaired t test)
	Group D (n=50)	Group M (n=50)	
Onset of Sensory Block	6.64 ± 2.48	5.02 ± 1.61	0.0002
Regression of Sensory Block	8.78 ± 3.64	7.52 ± 3.45	0.0788
Onset of Motor Block	10.94 ± 3.56	8.62 ± 3.19	0.0009
Regression of Motor Block	9.76 ± 3.89	7.48 ± 3.15	0.0017

The mean duration of analgesia was 93.34 ± 8.85 minutes in Group D compared to 84.50 ± 8.17 minutes in Group M. The mean difference between the groups was 8.84 minutes, with a 95% confidence interval ranging from 5.46 to 12.22 minutes. This difference was highly statistically significant (p<0.0001), indicating that patients receiving dexmedetomidine experienced a prolonged pain-free period following surgery [Table 3].

Table 3: Comparison of Duration of Analgesia

Parameters	Group D	Group M
Number of Patients (N)	50	50
Mean Duration in Minutes	93.34	84.50
Standard Deviation (SD)	8.85	8.17
Difference in Mean (M-D) ± SEM	-8.840 ± 1.703	
95% CI of Difference	-12.22 to -5.460	
p-value (Unpaired t test)	<0.0001	

The mean duration of tolerance was 27.58 ± 4.18 minutes in the dexmedetomidine group compared with 20.70 ± 4.27 minutes in the midazolam group. The mean difference of 6.88 minutes was statistically significant, with a 95% confidence interval of 5.20 to 8.56 minutes and a p-value of less than 0.0001 [Table 4].

Table 4: Comparison of Duration of Tolerance to Torniquet Pain

Parameters	Group D	Group M
Number of Patients (N)	50	50
Mean Duration in Minutes	27.58	20.70
Standard Deviation (SD)	4.18	4.27
Difference in Mean ± SEM	-6.880 ± 0.8450	
95% CI of Difference	-8.557 to -5.203	
p-value (Unpaired t-test)	<0.0001	

The mean fentanyl requirement was 50.82 minutes-equivalent in Group M compared with 55.14 in Group D, with a statistically significant difference of 4.32 units (95% CI: 1.49–7.15; p=0.0031) [Table 5].

Table 5: Comparison of Total Dose of Fentanyl

Parameters	Group D	Group M
Number of Patients (N)	50	50
Mean Duration in Minutes	55.14	50.82
Standard Deviation (SD)	7.57	6.66
Difference in Mean ± SEM	-4.320 ± 1.426	
95% CI of Difference	-7.150 to -1.490	
p-value (Unpaired t-test)	0.0031	

Bradycardia occurred in 20.0% of patients in Group D and 14.0% in Group M (p=0.5955), while hypotension was observed in 18.0% and 10.0% of patients, respectively (p=0.3881). Although these adverse events were numerically more frequent in the dexmedetomidine group, the differences were not statistically significant. Importantly, no cases of allergic drug reactions or local anaesthetic systemic toxicity were reported in either group [Table 6].

Table 6: Comparison of Adverse Effects

Parameter	Number of Patients (%)		p-value (Fisher's Exact Test)
	Group D (n=50)	Group M (n=50)	
Bradycardia	10 (20.0)	7 (14.0)	0.5955
Hypotension	9 (18.0)	5 (10.0)	0.3881
Allergic Reaction to Drugs	0	0	NA
Local Anaesthetics Systemic Toxicity	0	0	NA

DISCUSSION

The present randomized controlled trial compared the efficacy and safety of dexmedetomidine (0.5 μg/kg) versus midazolam (50 μg/kg) as adjuvants to lignocaine in intravenous regional anaesthesia for patients undergoing forearm surgeries. Regarding the primary outcome parameters, the midazolam group demonstrated a significantly faster onset of both sensory and motor blockade compared to the dexmedetomidine group. However, dexmedetomidine produced a significantly prolonged duration of motor block, while the duration of sensory block, though longer, did not reach statistical significance. For the secondary outcomes, dexmedetomidine provided a significantly longer duration of postoperative analgesia and better tolerance to tourniquet pain compared to midazolam. Interestingly, despite longer analgesia duration, the dexmedetomidine group required a significantly higher total dose of fentanyl postoperatively. Both groups exhibited comparable safety profiles, with no statistically significant differences in the incidence of bradycardia or hypotension, and no cases of allergic reactions or local anaesthetic systemic toxicity were reported.

Our finding that midazolam produced a significantly faster onset of sensory block (5.02 ± 1.61 min vs. 6.64 ± 2.48 min, p=0.0002) and motor block (8.62 ± 3.19 min vs. 10.94 ± 3.56 min, p=0.0009) compared to dexmedetomidine aligns with the observations of Gupta et al. (2017). In their study, midazolam also facilitated an earlier onset of sensory block (6.49 ± 4.26 minutes) compared to dexmedetomidine (8.01 ± 4.02 minutes), although the difference in their study was not statistically significant, whereas ours reached significance [10]. This discrepancy may be attributed to differences in sample size or patient characteristics. Kashefi et al. (2011) and Honarmand et al. (2015) similarly demonstrated that midazolam (50 μg/kg) significantly shortens both sensory and motor block onset times compared to lignocaine alone, supporting the rapid-onset properties of midazolam observed in our study [9,12].

Conversely, several previous studies have reported faster onset with dexmedetomidine. Nilekani et al. (2016) found that dexmedetomidine 0.5 μg/kg significantly accelerated sensory block onset (3.02 ± 1.41 min vs. 6.98 ± 3.4 min) compared to control [13]. Karmaniolou et al. (2021), in their meta-analysis, also concluded that dexmedetomidine significantly shortens sensory block onset time (MD -2.10 min; 95% CI -3.345, -0.86) [6]. Memis et al. (2004) similarly reported shortened onset times with dexmedetomidine. Our results differ from these findings, likely because those studies compared dexmedetomidine with plain lignocaine (no adjuvant), whereas our study directly compared dexmedetomidine against another active adjuvant (midazolam) [14]. In a head-to-head comparison, midazolam appears to offer faster onset, a finding consistent with Hassan et al. (2021), who noted that while both drugs enhanced sensory block onset, only midazolam improved motor block onset [15].

Our study demonstrated that dexmedetomidine significantly prolonged the regression (duration) of motor block (9.76 ± 3.89 min vs. 7.48 ± 3.15 min, p=0.0017). The sensory block regression was also longer in the dexmedetomidine group (8.78 ± 3.64 min vs. 7.52 ± 3.45 min), though this difference did not reach statistical significance (p=0.0788).

These findings are strongly supported by Nilekani et al. (2016), who reported that dexmedetomidine significantly prolonged both sensory and motor block recovery times [13]. Memis et al. (2004) similarly found prolonged sensory and motor block recovery times with dexmedetomidine [14]. Kashefi et al. (2011) and Honarmand et al. (2015) demonstrated that midazolam also prolongs recovery times compared to plain lignocaine, but our direct comparison suggests dexmedetomidine may be more effective at prolonging motor blockade specifically [9,12]. Karmaniolou et al. (2021) confirmed in their meta-analysis that dexmedetomidine improves anaesthesia quality scores, which indirectly supports prolonged block duration [6].

The mean duration of analgesia was significantly longer in the dexmedetomidine group (93.34 ± 8.85 min vs. 84.50 ± 8.17 min, p<0.0001), with a mean difference of 8.84 minutes. This finding is remarkably consistent with Gupta et al. (2017), who reported nearly identical values: 93 ± 28 minutes for dexmedetomidine and 84 ± 28 minutes for midazolam. The close agreement between our results and Gupta's study strengthens the validity of both investigations [10].

Nilekani et al. (2016) reported an even more pronounced prolongation of postoperative analgesia with dexmedetomidine (388.53 ± 104.71 min vs. 200.5 ± 55.86 min), though their control group received plain lignocaine rather than midazolam, explaining the larger difference [13]. Karmaniolou et al. (2021) similarly confirmed that dexmedetomidine significantly

prolongs intraoperative analgesia (MD 11.08 min). Farouk and Aly (2010) also demonstrated that midazolam prolongs the analgesic-free period, but our study shows dexmedetomidine is superior to midazolam in this regard [16].

Our study found that dexmedetomidine significantly prolonged the duration of tolerance to tourniquet pain (27.58 ± 4.18 min vs. 20.70 ± 4.27 min, p<0.0001). This finding aligns with Nilekani et al. (2016), who reported a lower incidence of tourniquet pain in the dexmedetomidine group (8/30 vs. 19/30) [13]. Hassan et al. (2021) also noted that both dexmedetomidine and midazolam delayed the onset of tourniquet pain [15]. Farouk and Aly (2010) specifically demonstrated that midazolam significantly lowers tourniquet pain scores at multiple time points (10, 15, 20, and 30 minutes, P < 0.0001) and reduces the need for fentanyl for tourniquet pain (15% vs. 65%, P = 0.02) [16]. Our study extends these findings by directly comparing the two adjuvants and showing dexmedetomidine's superiority in prolonging tourniquet pain tolerance.

An unexpected finding in our study was that despite a longer duration of analgesia, the dexmedetomidine group required a significantly higher total fentanyl dose (55.14 vs. 50.82 minutes-equivalent, p=0.0031). This appears contradictory to most previous literature. Nilekani et al. (2016) reported reduced intraoperative fentanyl consumption with dexmedetomidine (16.00 ± 27.24 µg vs. 35.30 ± 27.79 µg, p=0.009) [13]. Karmaniolou et al. (2021) similarly found a significant reduction in intraoperative rescue analgesia consumption (MD -19.70 mg; 95% CI -24.15, -15.26) [6]. Memis et al. (2004) also reported reduced analgesic requirements with dexmedetomidine [14]. Hassan et al. (2021) and Kashefi et al. (2011) both showed reduced fentanyl consumption with both adjuvants [9,15].

Our study found no statistically significant differences in adverse effects between groups. Bradycardia occurred in 20.0% (dexmedetomidine) vs. 14.0% (midazolam), and hypotension in 18.0% vs. 10.0% (p>0.05 for both). No allergic reactions or local anaesthetic systemic toxicity occurred.

These findings are consistent with Gupta et al. (2017) and Nilekani et al. (2016), who reported no significant side effects with either drug [10,13]. Karmaniolou et al. (2021) noted that while dexmedetomidine increases sedation risk, bradycardia and hypotension are minimal [6]. Memis et al. (2004) and Kashefi et al. (2011) also reported stable haemodynamic profiles [9,14]. Farouk and Aly (2010) noted higher sedation scores with midazolam for the first two hours, which we did not specifically assess [16]. Safavi et al. (2025) confirmed that even higher doses of dexmedetomidine (0.6 µg/kg) maintain haemodynamic stability without major side effects. Our study confirms that both dexmedetomidine (0.5 µg/kg) and midazolam (50 µg/kg) have acceptable safety profiles when used as adjuvants in IVRA [17].

The present study has several limitations. First, the relatively small sample size (50 patients per group) may limit the generalizability of the findings and the detection of rare adverse events. Second, the follow-up period was limited to 24 hours postoperatively, precluding assessment of longer-term outcomes such as chronic pain or delayed recovery. Third, the study did not measure plasma concentrations of the study drugs, which could have provided valuable pharmacokinetic insights and helped correlate systemic absorption with observed side effects. Fourth, while the study excluded patients requiring tourniquet times exceeding 90 minutes, the findings may not be applicable to longer surgical procedures. Fifth, the use of a single fixed dose of each adjuvant (dexmedetomidine 0.5 µg/kg and midazolam 50 µg/kg) precludes dose-response analysis, and different dosing regimens might yield alternative results. Finally, the study was conducted at a single tertiary care centre in eastern India, which may limit extrapolation to other populations and healthcare settings.

CONCLUSION

Midazolam provides a significantly faster onset of both sensory and motor blockade, making it a preferred choice when rapid surgical initiation is desired. In contrast, dexmedetomidine offers a significantly longer duration of postoperative analgesia, superior tolerance to tourniquet pain, and prolonged motor block recovery, making it more suitable for procedures where extended postoperative pain relief is beneficial. Notably, despite its longer analgesic duration, dexmedetomidine was associated with higher postoperative fentanyl requirements, a finding that warrants further investigation. The choice between dexmedetomidine and midazolam as an adjuvant in IVRA for forearm surgeries should therefore be individualized based on clinical priorities: midazolam for faster block onset, or dexmedetomidine for prolonged analgesia and better tourniquet pain tolerance.

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