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ORIGINAL RESEARCH **AMBULTORY ANESTHESIA**

The impact of different doses of intrathecal dexmedetomidine used as adjuvant to hyperbaric prilocaine in short ambulatory procedures under spinal anesthesia: a randomized controlled study

Eslam Gamal Biekhet [1], Hesham Elazzazi [2], Wael Hussein [3], Mohamed Zedan [4], Bassant Mohamed Abdelhamid [5]

Author affiliations:

1. Eslam Gamal Biekhet, Department of Anesthesiology, Surgical ICU & Pain Management, Armed Forces College of Medicine, Cairo, Egypt: E-mail: Eslambiekhet@gmail.com
2. Hesham Elazzazi, Department of Anesthesiology, Surgical ICU & Pain Management, Armed Forces College of Medicine / Faculty of Medicine, Ain-Shams University, Cairo, Egypt: E-mail: heshamelazzazi@hotmail.com
3. Wael Hussein, Department of Anesthesiology, Surgical ICU & Pain Management, Armed Forces College of Medicine, Cairo, Egypt: E-mail: w.abdelmoez@gmail.com
4. Mohamed Zedan, Department of Anesthesiology, Surgical ICU & Pain Management, Armed Forces College of Medicine, Cairo, Egypt: E-mail: mohammedzedan999@gmail.com
5. Bassant Mohamed Abdelhamid, Professor of Anesthesiology, Surgical ICU & Pain Management, Armed Forces College of Medicine / Kasr-Alainy Faculty of Medicine, Cairo University, Giza, Cairo, Egypt: E-mail: bassantmohamed197@yahoo.com; {ORCID: 0000-0001-8938-4175}

Correspondence: Bassant Mohamed Abdelhamid, **E-mail:** bassantmohamed197@yahoo.com; **Phone:** +201224254012

ABSTRACT

Objective: This study aimed to determine the most effective dose of dexmedetomidine as an adjuvant to prilocaine in spinal anesthesia.

Methods: Sixty-nine adult patients (21 to 65 y) scheduled for elective surgeries under spinal anesthesia were included in the study. Patients received spinal anesthesia with 3 mL of prilocaine and 0.5 mL dexmedetomidine of dose according to randomization of 5,10 and15 µg (D5, D10 and D15 respectively). Time of the first request of analgesia was set as a primary outcome.

Results: Time of the first request of rescue opioid was significantly shorter in D5 group (8 ± 6 h) compared to D15group (21 ± 4 h) ($P < 0.018$). 24 h of postoperative Nalbuphine consumption was higher in D5 group (4.67 ± 0.59 mg) compared to D15 group (2.5 ± 0.71 mg) ($P = 0.012$). The onset of sensory and motor blocks was significantly earlier in group D15 and D10 compared to group D5. Group D15 showed a significantly prolonged duration of sensory and motor blockade than Groups D10 and D5. The duration of sensory and motor blockade was significantly prolonged in group D10 compared with group D5 ($P < 0.001$).

Conclusion: 10 and 15 µg dexmedetomidine as an adjuvant to prilocaine in spinal anesthesia shortened the onset of both sensory and motor block, prolonged the duration of sensory block, motor block, and the time to first analgesic.

Clinical trial registration: The study was registered on Pan African Clinical Trial Registry (www.pactr.org) (ID: PACTR202204558879194-April 2022).

Keywords: Intrathecal Dexmedetomidine; Hyperbaric prilocaine; Ambulatory Surgery; Spinal Anesthesia

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1. INTRODUCTION

The growing trend of ambulatory surgery necessitates the administration of anesthetics with rapid onset and rapid recovery for protective reflexes, mobility, and micturition, as well as adequate postoperative pain management. The primary concern while administering spinal anesthesia (SA) is the potential for extended motor block and urine retention, which could result in delays in the patient's discharge from the hospital.[1]

Prilocaine is an amide local anesthetic with a high safety profile, fast onset, intermediate potency and duration of action. When compared to plain prilocaine, the hyperbaric preparation has been proven to have a noticeably faster onset, recovery, and time to first void.[2] However, the short duration is linked to a shorter analgesic duration, a higher need for postoperative analgesics, and delayed hospital discharge.[3]

Dexmedetomidine, a highly selective α2-adrenergic receptor agonist, can cause central hypnosis, sedation, and anxiolysis with hyperpolarization of nerve tissue, produce analgesia, and enhance regional anesthesia by changing the trans-membrane ionic conductivity of the locus ceruleus in the brainstem.[4]

Previous research has investigated the analgesic and hemodynamic effects of dexmedetomidine as an adjuvant to SA, revealing that it shortened the onset, prolonged the duration of sensory and motor blocks, and increased the analgesic durations.[5, 6, 7] But there have been no studies investigating the analgesic effect of intrathecal dexmedetomidine with prilocaine.

We hypothesised that the addition of intrathecal dexmedetomidine to hyperbaric prilocaine would induce deferential SA that may prolong the duration of the sensory block without a significant effect on the motor block, which would offer early mobilization, a short hospital stay, and no need of postoperative analgesia.

We aimed to determine the most effective dose of dexmedetomidine, out of 15 µg, 10 µg, or 5 µg, to prolong the postoperative analgesia without altering the motor block duration.

2. METHODOLOGY

A randomized, controlled, double-blinded trial was conducted in Almaza Military Hospital after the approval of the Institutional Review Board, faculty of Medicine, Armed Forces College of Medicine (IRB number 76-2021). The study was registered with the Pan African Clinical Trial Registry (www.pactr.org) (ID: PACTR202204558879194-April 2022). Written informed consent was obtained from all patients.

To determine the sample size the software www.openepi.com was used. Setting the time of first request for analgesia as a primary outcome and considering a previous study,[5] assuming 80% power, 0.05 level of significance, mean 1 for D5 = 1.90 ± 0.48, mean 2 for D10 =1.57 ± 2.8 with cases to control ratio, 2:1 The sample size was 69 participants (23 in each group). And after considering the drop-out rate of 10%, the final sample size was 78 participants (26 in each group).

Adult patients aged 21 to 65 y, of both genders, ASA I-II, scheduled for elective surgeries under SA, were included in the study. Patients with impaired mental status, body mass index <18 or > 35 kg/m^2, patients with coagulation disorders, patients with histories of allergic reactions to local anesthetics or dexmedetomidine, patients suffering from severe cardiac, respiratory, hepatic, renal, or neuropsychiatric disorders, or patients with histories of chronic use or abuse of sedatives, narcotics, alcohol, or other drug abuse were excluded from the study.

One day before the procedure, each patient had a comprehensive pre-anesthesia examination that included standard preoperative investigations. Prior to surgery, patients were instructed to fast for 6–8 h. The 10-point numerical rating scale (NRS), with 0 denoting no pain and 10 the worst pain possible, was explained to and understood by each patient.

Patients were connected to monitoring of non-invasive blood pressure, heart rate (HR), electrocardiogram, and pulse oximetry as soon as they arrived in the operating room. An intravenous line was established to preload the patient with Ringer lactate solution at a rate of 10 mL/kg before the initiation of the spinal procedure.

Using computer-generated random numbers kept in sealed envelopes, patients were randomly allocated into one of the three groups.

- Group D5: received SA with 0.5 mL of 5 µg dexmedetomidine and 3 mL (60 mg) of prilocaine (total 3.5 mL).
- Group D10: received SA with 0.5 mL of 10 µg dexmedetomidine and 3 mL (60 mg) of prilocaine (total 3.5 mL).
- Group D15: received SA with 0.5 mL of 15 µg dexmedetomidine and 3 mL (60 mg) of prilocaine (total 3.5 mL).

The group-specific drug solutions were prepared by only the researchers and handed in a closed envelope to another anesthesiologist, who was not involved in the study, to inject intrathecally. The patient's group assignment was concealed from the attending

Table 1: Basic characteristics of the studied groups.

Parameter	D5 (n = 23)	D10 (n = 23)	D15 (n = 23)	P value
Age (y)	31.52 ± 15.98	34.65 ± 17.9	34.78 ± 16.22	0.656
Weight (kg)	73.35 ± 9.97	73.43 ± 12.65	78.65 ± 8.79	0.159
Height (cm)	172.78 ± 7.55	171.78 ± 6.24	175.87 ± 6.3	0.107
BMI (Kg/m^2)	24.62 ± 3.47	24.93 ± 4.57	25.52 ± 3.41	0.335
Surgery time (min)	34.35 ± 6.62	37.39 ± 10.54	40.87 ± 9.49*	0.041
Type of Surgery:				
Anal surgery	13 (56.52)	12 (52.17)	7 (30.43)	0.486
Varicocele	2 (8.7)	5 (21.74)	7 (30.43)	
Hydrocele	2 (8.7)	0 (0)	2 (8.7)	
Ureteroscope	4 (17.39)	3 (13.04)	4 (17.39)	
Cystoscope	2 (8.7)	3 (13.04)	3 (13.04)	
Position				
Supine	3 (13.04)	5 (21.74)	9 (39.13)	0.147
Lithotomy	20 (86.96)	18 (78.26)	14 (60.87)	

*Data presented as mean ± SD). or number (percentage). * Denotes statistically significant from group D5 as P < 0.05. # Denotes Statistically significant from group D10 as P < 0.05.*

anesthesiologist, the patient, and the post-operative data collector. In this trial, dexmedetomidine hydrochloride (Precedex®, Pfizer, Germany) and hyperbaric prilocaine 2% (Takipril®, Sintetica, Germany) were administered to every patient.

2.1. Spinal anesthesia technique

Using a 25-gauge needle and 2 mL of 2% lignocaine, local infiltration was performed on a seated patient under perfect aseptic conditions. Subsequently, utilizing a midline approach and a 25-gauge Quincke spinal needle (Spinocan, B-Braun Melsungen AG, Melsungen, Germany), SA was administered at the L4–L5 level with 3.5 mL of a prepared local anesthetic. The patients were shifted into supine positions with their heads up. A short bevel (25G) needle was used for the pin prick test to evaluate the sensory block, and the Extended Modified Bromage Scale was used for the motor assessment.[8]

Ringer's lactate solution at a rate of 4 mL/kg/h was used for intraoperative fluid maintenance. Hypotension, which is defined as a drop in mean arterial blood pressure (MAP) of more than 20% of the baseline reading, was treated with the administration of 5 mg increments of ephedrine to keep MAP above 70 mmHg. Bradycardia, defined as HR < 50 beats/min, was treated with 0.5 mg of atropine, repeated if necessary.

Following the procedure, the patients were transferred to the post-anesthesia care unit (PACU), and SpO_2, HR, and MAP were monitored for two hours. After that, they were moved to the ward. All patients received paracetamol 1 gm infusion every 6 h. IV nalbuphine 5 mg boluses were given as additional rescue analgesia if the patient reported a Numeric Pain Rating Scale score ≥ 4. A maximum of 60 mg of nalbuphine per 24 h was permitted.

The time to first analgesic, defined as the period from the subarachnoid injection to the patient experiencing pain with a NRS score ≥ 4, was set as a primary outcome.

Secondary outcomes were: (i) the onset of sensory block (the period between the application of SA and the total loss of skin sensation to the pinprick test up to T10 level); (ii) the highest sensory level (the maximum level reached with complete loss of sensation to the pinprick); (iii) sensory loss duration (time to complete recovery of sensation); (iv) Onset of motor block (described as the time from the administration of SA to the extended modified Bromage scale of three.); (v) Motor loss duration (described as the duration from the administration of SA to the extended modified Bromage scale of zero); (vi) Time of ambulation was defined as the patient's capacity to walk independently. (vii) Total nalbuphine consumption in the first 24 h postoperative, (viii) NRS; (ix) perioperative HR and MAP; (x) SA-related complications such as shivering (treated with IV meperidine 30 mg), hypotension, bradycardia, local anesthetic toxicity, hematoma formation, and lower limb weakness (xi) Nausea and vomiting score, using a four-point verbal scale; treated with metoclopramide (10 mg)

Table 2: Postoperative analgesic requirement

Analgesic requirement	D5 (n = 23)	D10 (n = 23)	D15 (n = 23)	P value
First 12 hours	15 (65.2)	3 (13)	0 (0)	**< 0.001**
Second 12 hours	3 (13)	3 (13)	2 (8.7)	1.00
Time of first request of analgesia (h)	8 ± 6	15 ± 8	21 ± 4	**0.018**
Total 24-h analgesic requirement (mg)	4.67 ± 0.59	4 ± 0.89	2.5 ± 0.71*	**0.012**

*Data presented as number (percentage). Time of first request of analgesia and total 24-hour analgesic requirement are presented as mean ± SD; * denotes statistically significant from group D5; # denotes statistically significant from group D10. significant P < 0.05.*

Table 3: Numerical rating scale scores

Recording time	D5 (n = 23)	D10 (n = 23)	D15 (n = 23)	P value
2 h	0 (0:0)	0 (0:0)	0 (0:0)	…
4 h	2 (2:3)	0 (0:0) *	0 (0:0) *	**< 0.001**
6 h	5 (4:6)	0 (0:2) *	0 (0:0) *	
12 h	6 (5:6)	3 (2:4) *	1 (0:2) *#	
18 h	6 (6:7)	4 (3:4) *	2 (1:2) *#	
24 h	6 (6:7)	4 (3:4) *	2 (2:2) *#	

*Data are presented as median (interquartile range). * Denotes statistically significant from group D5. # Denotes Statistically significant from group D10. significant p value < 0.05*

and (xii) level of postoperative sedation by the Ramsay Sedation Scale (RSS) score.

2.2. Statistical analysis

Excel 2010 for Windows from Microsoft Office was used to tabulate the data. Data analysis was performed using the Statistical Package of Social Science Software Programme, version 25 (IBM SPSS Statistics for Windows, Version 25.0; Armonk, NY: IBM Corp.). The chi-square test was used to analyze categorical variables, which were expressed as frequencies (percentages).

The normality of the data distribution was ascertained using the Shapiro-Wilk test. A repeated measures general linear model analysis of variance (ANOVA) was used to analyze normally distributed data, which was subsequently presented as means and standard deviations (SD). For data not normally distributed, the Kruskal-Walli's test was applied for analysis, and the results were displayed as medians and interquartile ranges (IQR).

Repetitive measures analysis of variance was used to examine the NRS data. The repeated measures ANOVA model was used to compare the baseline and subsequent values of MAP and HR within the same group (pair-wise group comparison). The Bonferroni test was then used as a post-hoc test. A $P < 0.05$ was deemed significant.

3. RESULTS

To determine their eligibility, 78 adult patients underwent screening. Due to their inability to meet the inclusion criteria, nine patients were excluded. Sixty-nine (23 per group) were included and completed the study. All 69 adult patients were analyzed statistically. For each of the three study groups, the surgical time and demographic information were comparable (Table 1).

The time to first request for rescue opioids was significantly shorter in the D5 group (8 ± 6 h) compared to the D15 group (21 ± 4 h) ($P < 0.018$). On the other hand, there was a significant difference in the total dose of postoperative opioid consumption over the first 24 h, which was higher in the D5 group (4.67 ± 0.59 mg) compared to the D15 group (2.5 ± 0.71 mg) ($P = 0.012$) (Table 2). NRS at all time measurements (4 h, 6 h, 12 h, and 24 h postoperative) was significantly higher in the following order: Group D5 > D10 > D15 ($P < 0.001$) (Table 3).

The onset of sensory block was significantly earlier in groups D15 and D10 compared to Group D5 ($P < 0.001$) and ($P = 0.007$), respectively; however, no statistically significant difference was observed between groups D10 and D15 ($P = 0.067$). The time to attain the T10 level was significantly shorter in D15 and D10 compared with

Table 4: Spinal anesthesia block characteristic.

	D5 (n = 23)	D10 (n = 23)	D15 (n = 23)	P value
Sensory bock onset (min)	2.22 ± 0.48	1.74 ± 0.44*	1.41 ± 0.26*	**< 0.001**
Time to reach T10 (min)	3.36 ± 0.3	2.81 ± 0.38*	2.44 ± 0.28*#	**< 0.001**
Peak sensory level				
• T10	23 (100)	15 (65.2)	9 (39.1)	**< 0.001**
• T8	0 (0)	8 (34.8)	14 (60.9)	
Motor block onset	2.77 ± 0.38	2.21 ± 0.49*	1.91 ± 0.29*	**< 0.001**
Two segments regression time (h)	1.19 ± 0.17	1.48 ± 0.28*	1.84 ± 0.16*#	**< 0.001**
Duration of motor block (h)	1.17 ± 0.16	1.7 ± 0.3*	2.89 ± 0.41*#	**< 0.001**
Duration of sensory block (h)	3.42 ± 0.29	4.67 ± 0.49*	5.99 ± 0.53*#	**< 0.001**
Time of ambulation (h)	2.11 ± 0.32	2.61 ± .0.35*	3.58 ± 0.42*#	**< 0.001**

*Sensory onset, time to reach T10, motor onset, two segments regression time, duration of motor block, duration of sensory block and time of ambulation are presented as mean (standard deviation). Peak sensory level is presented as number (percentage). * Denotes statistically significant from group D5. # Denotes Statistically significant from group D10. significant p value < 0.05.*

Group D5 (P < 0.001). There was a significant difference with respect to 2 segment sensory regression time between Group D15 vs. D10 (P = 0.002), Group D15 vs. D5 (P < 0.001), and a significant difference between Group D10 vs. D5 (P = 0.004). Group D15 had a significantly longer duration of sensory blockade than Groups D10 and D5 (P =0.001 and P < 0.001, respectively). The duration of sensory blockade was significantly prolonged in Group D10 compared with Group D5 (P < 0.001) (Table 4).

The onset of motor block was significantly earlier in Groups D15 and D10 compared with Group D5 (P < 0.001 and P = 0.001, respectively); however, there was no significant difference between Groups D15 and D10 (P = 0.145). Group D15 had a significantly longer duration of motor blockade than Groups D10 and BD5

Table 5: Mean arterial blood pressure of the study groups

Time	D5 (n = 23)	D10 (n = 23)	D15 (n = 23)	P value
Preoperative baseline	93.38 ± 11.08	96.16 ± 9.24	91.12 ± 11.67	0.285
Intraoperative:				
Post-spinal	81.57 ± 8.64†	83.58 ± 12.78†	78.22 ± 9.91†	0.229
10 min	82.33 ± 10.54	79.19 ± 8.24†	77.91 ± 9.89†	0.282
20 min	82.41 ± 9.36	79.01 ± 6.81†	77.03 ± 12.9†	0.191
30 min	82.74 ± 8.96	79.2 ± 7.37†	79.28 ± 8.47†	0.262
40 min	83.38 ± 12.88	81.53 ± 9.44†	76.5 ± 9.53†	0.257
Postoperative:				
0 h	84.46 ± 7	82.39 ± 6.6	81.93 ± 7.61	0.438
2 h	86.03 ± 5.3	86.49 ± 7.37	83.88 ± 6.35	0.266
4 h	85.71 ± 5.7	87.83 ± 5.83	84.74 ± 5.31	0.171
6 h	88.96 ± 6.28	89.91 ± 7.72	86 ± 5.99	0.127
12 h	89.64 ± 5.21	88.9 ± 7.94	86.62 ± 6.52	0.284
24 h	88.09 ± 6.09	89.19 ± 7.55	88.72 ± 7.01	0.863

*Data are presented as mean (standard deviation). * Denotes statistically significant from group D5. # Denotes Statistically significant from group D10. †Denotes statistically significant from baseline preoperative values. significant p value < 0.05.*

Table 6: Heart rate of the study groups (HR)

Time	D5 (n = 23)	D10 (n = 23)	D15 (n = 23)	P value
Preoperative baseline	75.91 ± 11.45	78 ± 11.25	81 ± 11.1	0.312
Intraoperative:				
Post-spinal	71.39 ± 12.91†	73.26 ± 10.09	72.91 ± 9.96†	0.831
10 min	71.61 ± 9.51	71.48 ± 11.58	70.87 ± 10.82†	0.969
20 min	70.35 ± 10.13	71.04 ± 10.03	68.3 ± 12.01†	0.67
30 min	70.48 ± 8.64	69.83 ± 10.65	70.17 ± 10.69†	0.976
40 min	70.63 ± 7.46	67.3 ± 9.58	64.56 ± 10.08†	0.336
Postoperative:				
0 h	71.48 ± 8.55	71.78 ± 8.36	70.57 ± 10.29†	0.895
2 h	72.7 ± 7.14	72.52 ± 7.26	72.35 ± 8.22†	0.988
4 h	72.78 ± 5.69	73.78 ± 7.22	72.13 ± 7.4†	0.711
6 h	75.22 ± 6.04	75.91 ± 7.39	74.3 ± 6.01†	0.704
12 h	75.65 ± 5.89	76.48 ± 7.24	74.22 ± 6.27†	0.493
24 h	74.22 ± 6.76	75.7 ± 6.18	74.48 ± 6.57†	0.715

*Data are presented as mean (standard deviation). * Denotes statistically significant from group D5. # Denotes Statistically significant from group D10. †Denotes statistically significant from baseline preoperative values. significant p value < 0.05.*

(P < 0.001). The duration of motor blockade was significantly prolonged in Group D10 compared with Group D5 (P = 0.001) (Table 4).

Regarding baseline, intraoperative, and postoperative MAP, the three groups were comparable. Compared to the preoperative baseline values, all readings of the intraoperative MAP in groups D10 and D15 were statistically lower than the baseline values, while in Group D5, only the post-spinal reading was statistically lower than the baseline (Table 5).

Similarly, regarding baseline, intraoperative, and postoperative HR, the three groups were comparable. Compared to the preoperative baseline values, all readings of the intraoperative and postoperative HR in Group D15 were significantly lower than the baseline value; while in Group D5, only the post-spinal reading was statistically lower than the baseline (Table 6).

Two patients in Group D5 experienced intraoperative bradycardia; it was corrected by a single dose of 0.5 mg IV atropine. One patient in Group D10 and four patients in Group D15 experienced intraoperative hypotension, which was corrected by a single dose of ephedrine 5 mg IV. One patient each in Group D5 and Group D10, and two patients in Group D15 experienced intraoperative shivering, which was corrected by a single dose of meperidine 30 mg IV.

None of our patients experienced sedation (the RSS score was 2 in all patients). None of our patients had postoperative nausea and vomiting, signs of anesthetic toxicity, hematoma, or prolonged lower limb weakness.

4. DISCUSSION

The main findings of this study indicate that increasing the dose of dexmedetomidine to 15 µg with prilocaine in SA prolongs the time to first request of rescue analgesic, reduces opioid consumption in the first 24-hours,

decreases the onset time for sensory and motor blocks, and prolongs both sensory and motor block duration., compared to 5 µg and 10 µg dexmedetomidine.

When selecting a prilocaine dosage, a variety of criteria should be considered, including the type of surgery and the clinical characteristics of the patient. According to a prior review9], hyperbaric prilocaine is superior for the ambulatory situation because it provides a faster onset of the spinal block and hastens patient recovery. It has been determined that 10 mg of 2% hyperbaric prilocaine is suitable for small perianal surgery and that a dose of 40–60 mg of hyperbaric prilocaine is appropriate for procedures involving the lower extremities and lower abdomen that last up to 90 min.

To induce sensory block to T10 in outpatient surgery, Camponovo C. et al. compared the efficacy of 60 and 40 mg intrathecal dosages of 2% hyperbaric prilocaine with

60 mg of 2% ordinary prilocaine.[10] The onset times to T10 sensory block were significantly shorter for both 60 and 40 mg of 2% hyperbaric prilocaine than for 60 mg of 2% plain prilocaine. When compared to plain prilocaine, hyperbaric solution produced motor and sensory blocks faster, the anesthetic was established quicker, and patients recovered faster. Similar to this study, 100 patients scheduled for urologic procedures under SA were the subjects of an investigation by Ostgaard G. et al. into the analgesic effects of 80 mg of prilocaine and lidocaine.[11] In the prilocaine group, the mean onset of block was 13.4 ± 4 min, the mean duration of sensory block was 221 ± 49 min, and the mean duration of motor block was 197 ± 42 min.

Ratsch et al. examined the effects of 60 mg of 2% hyperbaric prilocaine against 0.5% hyperbaric bupivacaine in 88 patients undergoing lower limb procedures under SA.[12] Both groups were comparable in sensory onset, maximum sensory block level, and sensory duration of 360 ± 60 min in the bupivacaine group and 240 ± 90 min for the prilocaine group, whereas the motor block regression was 135 ± 90 min for the prilocaine group versus 210 ± 90 min for the bupivacaine group.

Under a variety of circumstances, neuraxial dexmedetomidine displayed a remarkable effect; its prolongation of sensory and motor blocks could be linked to the hyperpolarization of neuronal cation currents and the suppression of C-fibre transmitter release.[13] The lipophilic nature causes rapid binding to the adrenoreceptors in the spinal cord. Moreover, they also enhance the effects of local anesthetics and reduce the required doses. Compared to other α2-agonists, dexmedetomidine has a higher affinity for α2-adrenergic receptors (more than eight times that of clonidine) and a lower affinity for α1-receptors. It is a selective, short-acting agonist of the α2-adrenergic receptors. Its analgesic activity is also attributed to its strong selectivity for α2A-adrenergic receptors.[14,15]

As far as we know, there have never been any studies investigating the analgesic effect of intrathecal dexmedetomidine with prilocaine for SA.

The impact of intravenous dexmedetomidine was investigated by Tekin M et al. with 80 mg of prilocaine 2% in SA, demonstrating prolonged sensory and motor block duration with a moderate level of sedation.[16] Kol IO. et al. compared the effect of dexmedetomidine to lornoxicam in intravenous regional anesthesia with prilocaine in patients undergoing upper limb surgeries, reporting that dexmedetomidine had a more potent effect, hastening sensory block onset time and prolonging sensory block recovery time.[17]

In this dose-finding study, we selected 5, 10, or 15 μg of dexmedetomidine as an adjuvant to prilocaine in SA. The safe dose of intrathecal dexmedetomidine has been demonstrated in a review by Naaz et al. to be 0.1–0.2 μg/kg.[18]

According to the systematic reviews and meta-analyses of Liu S. et al., which analyzed 1478 patients from twenty-five clinical studies, they investigated the analgesic effect of a dose of 5 μg dexmedetomidine as an adjuvant to bupivacaine in SA, demonstrating that intrathecal dexmedetomidine shortened the onsets of sensory block and motor block by 0.80 min and 1.03 min, respectively.[6] It also prolonged the sensory block, motor block, and analgesic durations.

Sun S. et.al. conducted another meta-analysis on 639 patients from nine studies, which included studies comparing dexmedetomidine at different doses (3 and 5 μg) as a local anesthetic adjuvant to fentanyl indicating that dexmedetomidine increased the duration of SA, improved postoperative analgesia, decreased the incidence of pruritus, and did not increase the incidence of hypotension and bradycardia.[7]

According to the study's findings, intraoperative shivering developed in one patient each in Group D5 and Group D10, and two patients in Group D15. These shivering episodes were resolved by a single injection of 30 mg of meperidine intravenously. With an incidence of 40–70%, shivering is recognized as a common consequence in patients undergoing surgery under neuraxial anesthesia.[19] Dexmedetomidine administration lowers the hypothalamus's central thermosensitivity and decreases the spinal cord neurons' rate of spontaneous firing. Additionally, dexmedetomidine reduces vasoconstriction and enhances the shivering threshold. According to limited evidence, dexmedetomidine may also prevent shivering by lessening the hyperadrenergic response to perioperative stress.[20, 21]

Two patients in Group D5 experienced intraoperative bradycardia, which resolved with a single injection of atropine 0.5 mg. One patient in Group D10 and four patients in Group D15 experienced intraoperative hypotension, resolved by 5 mg of IV ephedrine. Rather than dexmedetomidine, the impact of SA may be the explanation for these results. Numerous studies have evaluated the analgesic, hemodynamic, and analgesic effects of dexmedetomidine on subarachnoid anesthesia.[22–24] The intrathecal approach offers greater advantages than the intravenous technique, although both maintain sufficient hemodynamic stability and prolong the sensorimotor effects of subarachnoid anesthesia.[25]

5. LIMITATIONS

The study had a limitation that there was no control group. Nonetheless, prior research has established the precise onset, duration, and analgesic impact of SA with prilocaine. We recommend that future research examine the effects of prilocaine, when used with various adjuvants, such as opioids, magnesium sulphate, ketamine, and midazolam, in spinal, epidural, and different nerve blocks.

6. CONCLUSION

The addition of 10 or 15 µg of dexmedetomidine as an adjuvant to prilocaine in spinal anesthesia shortened the onset of both sensory and motor blocks, prolonged the duration of sensory and motor blocks, and prolonged the time to the first analgesic request. The selection of either dose depends on the duration and type of surgery, the required sensory level, and the predicted severity of postoperative pain.

7. Availability of data

Data are available from the authors upon reasonable request after permission of Cairo University.

8. Conflict of interest

The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript. The authors did not receive any funding from any public, commercial, or not-for-profit agencies.

9. Consent for publication

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10. Ethics approval

The study was conducted in Almaza Military Hospital after the approval of the Institutional Review Board—Faculty of Medicine—Armed Forces College of Medicine (IRB number 76-2021).

11. Clinical trial registration

The study was registered on the Pan African Clinical Trial Registry (www.pactr.org) (ID: PACTR202204558879194-April 2022).

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13. Authors' contribution

All authors took part in conception of the idea, design of the study, data collection, analysis of the data, writing and revising the manuscript, and approval of the final manuscript.

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