

Original Article

A Comparative Study of Clonidine and Dexmedetomidine with 0.5% Levobupivacaine in Ultrasound-Guided Axillary Brachial Plexus Block for Upper Limb Surgeries

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Abstract

Background: Clonidine and Dexmedetomidine as an additive to Levobupivacaine in ultrasound-guided axillary brachial plexus block are not studied well. Hence, this study was designed to compare the efficacy of the Clonidine and Dexmedetomidine, used as an adjunct to Levobupivacaine in ultrasound-guided axillary brachial plexus block in upper limb elective surgery.

Materials and Methods: Eighty patients of the American Society of Anesthesiologists Grade I/II undergoing upper limb elective surgery were divided into two equal age/gender-matched groups. Group-LD received ultrasound-guided axillary brachial plexus block using injection 0.5% Levobupivacaine 20 ml+dexmedetomidine (1 µg/Kg), and group LC received ultrasound-guided axillary brachial plexus block using injection 0.5% Levobupivacaine 20 ml + Clonidine (1.5 µg/Kg). The onset and duration of sensory and motor block, hemodynamics, and side effects were recorded.

Results: The time for onset of sensory block and motor block in group LD was significantly faster than group LC (4.53±1.07 and 7.88±1.29 min vs. 5.90±0.81 and 8.85±1.81min, p<0.0001). The duration of motor block in group LD was significantly longer than group LC (Sensory and motor block: 662.50 ± 50.95 and 625.50 ± 51.95min, vs 567.75 ± 62.33 and 560.62 ± 67.19 min. p< 0.0001). The sedation score was highly significant at 30 min (p<0.0001) and was significant at 60 min (p<0.05), postoperatively.

Conclusion: The addition of Dexmedetomidine (1µg/Kg) as an adjuvant to Levobupivacaine (0.5%) for upper limb surgeries by axillary brachial plexus block had provided the rapid onset of sensory block and motor block and enhanced duration of sensory and motor block with arousable sedation without any adverse effects compared to clonidine (1.5µg/Kg).

Keywords: Axillary Brachial Plexus Block, Dexmedetomidine, Levobupivacaine, Clonidine

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Please cite this article as: Kumar VA, Raj BP, Chaitanya1 KK, Shamiq Muthaib SM. A Comparative Study of Clonidine and Dexmedetomidine with 0.5% Levobupivacaine in Ultrasound-Guided Axillary Brachial Plexus Block for Upper Limb Surgeries. J Cell Mol Anesth. 2021;6(3):249-58. DOI: <https://doi.org/10.22037/jcma.v6i3.34421>

Introduction

Upper limb surgeries below the shoulder joint are mostly performed under the brachial plexus block. Brachial plexus techniques include interscalene block, supraclavicular block, infraclavicular block, and axillary block. Brachial Plexus Block provides effective intraoperative anesthesia and prolonged postoperative analgesia without any side effects for upper limb surgeries (1).

Levobupivacaine is an S (-)- enantiomer of racemic bupivacaine. Compared with bupivacaine, it produces less vasodilation, less hypotensive episodes, less CNS toxicity, less negative inotropic effect, less prolongation of QTc interval, and a higher toxicity threshold (2). However, it has limiting factors like delayed onset, patchy and incomplete analgesia.

Various studies have investigated several adjuvants, including opioids, Alpha -2 agonists like Dexmedetomidine and Clonidine, neostigmine, hyaluronidase, dexamethasone (2, 3). Dexmedetomidine is a potent α -2 adrenoceptor agonist and about eight times more selective towards the α -2 adrenoceptor than Clonidine. It has been used as an adjuvant to local anesthetics to prolong block and postoperative analgesia in various peripheral blocks (4-6).

The use of USG guidance for the localization of nerve plexus has revolutionized regional anesthesia (7).

Levobupivacaine has a great success rate and safety along with a marked reduction of the dose of local anesthetics and adjuvants (8). Studies comparing Clonidine and Dexmedetomidine are reported for brachial plexus block, but a high dose of α -2 agonist is associated with side effects include hypotension and Bradycardia.

No studies have so far compared the efficacy of Clonidine and Dexmedetomidine as an adjuvant to Levobupivacaine in ultrasound-guided axillary brachial plexus block in upper limb surgeries.

Hence, we planned a double-blind prospective randomized clinical study at our institute to evaluate the comparative efficacy of Dexmedetomidine (1 μ g/kg), and Clonidine (1.5 μ g/kg) used in ultrasound-guided axillary brachial plexus block as adjuvants to Levobupivacaine in patients undergoing upper limb surgeries.

Methods

After the institutional ethics committee's approval and signed written informed consent, a randomized, double-blind study was done. A prospective, double-blind, randomized controlled study was conducted on 80 patients undergoing upper limb surgery with ASA grade I or II under ultrasound-guided axial, brachial plexus block. We have selected fractures of the radius, Fracture of the ulna, and both bone fractures of the forearm. Surgeries lasted for 2-4 hours.

Patients who underwent elective upper limb surgery posting to the anesthesia department during the study period and who satisfied the inclusion criteria were selected for the study.

The *sample size* was calculated by the formula: $N = \frac{z_1^2 \cdot 1 - \alpha / 2^{p(1-p)}}{d}$

Where, p=expected proportion; d=absolute precision; $1 - \alpha / 2$ =desired confidence level; P=0.6; Preci d=10; $1 - \alpha / 2$ =95. Required sample size (N)=80.

Eighty patients were randomly divided into two groups using the "slip in the box technique" each containing 40 patients (using a computer-generated randomization table).

The study was conducted between the duration of January 2019 to June 2020.

Group LC: (n=40) receive 20ml of 0.5 % of Levobupivacaine +1.5 μ g/kg of Clonidine.

Group LD: (n=40) receive 20ml of 0.5 % of Levobupivacaine + 1 μ g/kg of Dexmedetomidine.

Inclusion criteria were ASA Class I and II of age between 20 and 60 years, SBP - 100-140 mm of Hg, and DBP - 60-90 mm of Hg.

Exclusion criteria were ASA Class III and IV, Patients with complications like severe anemia, severe hypovolemia, shock, septicemia, Abnormal CT, BT, or anticoagulant therapy, Local infection at the site of axillary block, history of drug allergy to local anesthetics, Clonidine, or Dexmedetomidine, and the patient refusal.

Method: The technique, ultrasound-guided axillary brachial plexus, was conducted in the operation theatre. Investigations include Hb% estimation, TC, DC, Urine examination - Albumin, Sugar, and Microscopy, X-ray chest, RBS, Blood urea, Serum Creatinine, B.T, C.T, PT, aPTT, INR, E.C.G, HIV, and HBsAg.

Performance of Axillary Block under Ultrasound

Guidance: Patients were given alprazolam 0.5 mg a day before surgery. No sedative premedication on the day of surgery was given to avoid interference in scoring sedation.

The patient was placed in the supine position with the head turned away from the side of the block. The arm is abducted at 90°, and the elbow flexed to 90°. The axilla is prepared aseptically. We have used High-frequency, linear probes (13-6MHz) for imaging because the nerves are superficial (1 to 2 cm) below the skin. The most proximal location at the apex of the axilla may be the best for viewing all of the terminal branches of the brachial plexus. The probe is positioned perpendicular to the anterior axillary fold and in cross-section to the humerus at the bicipital sulcus (and at the level of the axillary pulse) to capture the transverse, or short-axis, given the neurovascular bundle.

A 5-cm, 22-gauge insulated needle is used. In-plane approaches are used for axillary blocks based on convenience. The in-plane method involves inserting the needle at an acute angle (20 to 30 degrees) to the skin in a lateral-to-medial direction. Typically, the block needle is advanced to the direction median nerve. It is then crossed over the axillary artery to the ulnar nerve superficially and finally behind the artery to the deeper radial nerve. The musculocutaneous nerve is usually blocked within the coracobrachialis, where its flat shape gives a large amount of surface area for a rapid block of both sensory and motor fibers after positioning the probe, the infiltration of LA distal to the probe subcutaneously is recommended to cover the injection site and also to block the intercostobrachial nerve. After carefully positioning the needle tip, gentle negative aspiration is done, and small aliquots of 4-5 ml of LA are injected around each nerve. All four nerves are blocked individually.

All patients were monitored for onset and duration of motor and sensory block and duration of analgesia up to 12hrs postoperatively. Sensory blockade was tested using the pinprick method along with the distribution of the four nerves.

Sensory block graded as follows: Grade-0: sharp pin felt, Grade-1: analgesia, dull sensation, Grade-2:

anesthesia, no sensation.

Sensory block was assessed corresponding to the median nerve, radial nerve, ulnar nerve, and musculocutaneous nerve dermatomal areas. Sensory onset is considered when there is a dull sensation to pinprick (grade-1). The sensory block duration is the time interval between the end of LA administration and the complete resolution of the anesthesia on the dermatomal areas corresponding to all nerves.

Motor blockade assessment was done using the Modified Bromage Scale (MBS) for upper extremities on a three-point scale.

Grade 0= normal motor function with the full extension of the elbow, wrist, and fingers.

Grade 1= the ability to move fingers and/or wrist only, decrease motor strength;

Grade 2=complete motor blockade with the inability to move fingers.

The onset of motor blockade was considered when there is a grade 1 motor blockade. Peak motor block was considered if there is a grade 2 motor blockade. The motor block duration is the time interval between the end of LA administration and the recovery of complete motor function. When the block is complete patient did not require any rescue analgesic intraoperatively.

The block was considered incomplete when any of the segments supplied by the median, radial, ulnar and musculocutaneous nerve did not have analgesia even after 30 min of drug injection. These patients were supplemented with intravenous fentanyl (1 µg/kg) and midazolam (0.02 mg/kg).

After local anesthetic injection, the onset, duration of motor and sensory blockade measurements, and vital parameters (pulse, BP, SPO₂) were carried out 0 min, 5min, 15min, 30 min, 60min, 120 min, 360 min, 720 min. Postoperatively, the patient's motor and sensory blockade and vitals were noted half hourly till the block completely wears off. Adverse effects like hypotension (i.e., 20% decrease relative to baseline), bradycardia (HR < 50 beats/min), nausea, vomiting, and hypoxemia (SPO₂<90) if occurred were noted and attended. The time between the complete sensory block and the first analgesic request was recorded as the duration of analgesia (DOA). The pain was assessed using a visual analog scale (VAS) 0-10 at an interval of every

30 min. The first dose of postoperative analgesia was based on VAS \geq 2 or on-demand made by the patient (whichever was early) and was noted for use as Analgesia time. The sedation score was assessed using a 5-point sedation scale. The scoring was recorded as follows:

Awake and alert [1], sedated but responding to verbal stimulus [2], sedated, responding to mild physical stimulus [3], sedated, responding to moderate or strong physical stimulus [4], and not arousable [5].

To detect a difference of 179 mins in the duration of motor and sensory block, with 80% power and 5% level of significance, a sample size of 40 per group was chosen (9). Data were analyzed using SPSS version 24 for Windows (IBM Corp., Armonk, NY, USA). Categorical data were represented as frequencies and proportions. Continuous data were represented as mean and SD. A χ^2 -square test was used to find the significance of association for qualitative data. The Independent t-test was used as the significance test to identify the mean difference between the two groups. A paired t-test was the test for paired data such as before and after surgery. p -value <0.05 was considered significance.

Results

A total of 80 ASA class I and II patients of both genders, aged between 18-60 years, posted for upper limb surgeries under axillary brachial plexus block were selected for the study.

In group LC, 75.00% of the patients and in group LD, 72.50% of the patients were ASA class I, whereas 25.00% of patients in group LC and 27.50% of patients in group LD were ASA class II (Table 1). The distribution of subjects based on ASA class was comparable. And no significant difference was observed between the groups, as the p -value was more than 0.05. The mean duration of onset of sensory block in group LD was significantly faster than group LC ($p < 0.0001$). The mean duration of motor block onset in the LD group was significantly faster than group LC ($p < 0.05$). The mean duration of

the group LD's sensory block was significantly longer than group LC ($p < 0.0001$). The mean duration of motor block in the LD group was significantly longer than group LC ($p < 0.0001$) (Table 1).

Sedation score: At sedation was observed between 30 min and 60 min from the time of drug injection in these two groups. At 15 min, 22.50% of patients in group LC are sedated (sedation score 2), whereas, in group LD, 27.50% of patients were sedated (sedation score 2). The difference in sedation score at 15 min was not statistically significant though few subjects in both the groups were sedated ($p=0.06$). At 30 min, in group LC, 37.50% of patients were sedated (with sedation score 2), whereas 80.00% of patients were sedated (65.00% of patients with sedation score 2 and 15.00% of patients with sedation score 3) in group LD which is statistically highly significant ($p=0.0001$). At 60 min, in group LC, 47.50% of patients were sedated (37.50% of patients with sedation score 2 and 10.00% of patients with sedation score 3), and in group LD, 80.00% of patients were sedated (65.00% of patients with sedation score 2 and 15.00% of patients with Sedation score 3) which is statistically significant ($p=0.01$). None of the patients had a sedation score of 4 and above during the study. χ^2 -square analysis showed that the difference in sedation score was significant ($p < 0.05$) at 30 and 60 min (Table 2).

Hemodynamics: There were no significant differences in the Pulse Rate between the groups measured at 0, 5, 15, 30, 60, 120, 360, and 720 mins ($p > 0.05$) (Table 3). There were no significant differences in the systolic BP between the groups measured at 0, 5, 15, 30, 60, 120, 360, and 720 mins ($p > 0.05$) (Table 3). There were no significant differences in the Diastolic BP between the groups measured at 0, 5, 15, 30, 60, 120, 360, and 720 mins ($p > 0.05$) (Table 3). There were no significant differences in the Oxygen saturation between the groups measured at 0, 5, 15, 30, 60, 120, 360, and 720 mins ($p > 0.05$) (Table 3).

Table 1: The demographics, surgical characteristics, comparison of Onset of sensory and motor block and sensory and motor block duration between two groups.

Variable	Group LC	Group LD	P-value
Age [years]	36.40±12.56years	39.10±13.37years	>0.05
Sex [M:F]	28: 12	32: 8	>0.05
Weight [kgs]	55.72±0.164	57.00±2.98	0.164
ASA [I:II]	30: 10	29: 11	>0.05
onset of sensory block (min)	5.90±0.81 ^a	4.53±1.07 ^a	<0.0001**
onset of motor block (min)	8.85±1.81 ^a	7.88±1.29 ^a	0.05
Duration of sensory block (min)	567.75±62.33 ^a	662.50±50.95 ^a	<0.0001**
Duration of motor block	560.62±67.19 ^a	625.50±51.95 ^a	<0.0001**

^aStudent's unpaired t-test; P value<0.05: significant; P value<0.001- highly significant.

Discussion

Ultrasound guidance has established its effectiveness and safety and revolutionized the practice of peripheral nerve blocks. USG helped us visualize the nerve roots and depositing the drug at the plexus (10, 11). Local anesthetics alone for brachial plexus block provide good operative conditions but have a shorter postoperative analgesia duration. Various studies used different types of local anesthetics such as bupivacaine, Levobupivacaine, ropivacaine, lignocaine. Chakraborty et al.(11) demonstrated the effect of Clonidine as an adjuvant in 0.5% 25 ml of bupivacaine-induced supraclavicular brachial plexus block. Esmaoglu et al. (12) studied the effect of mixing Dexmedetomidine to 0.5% 45 ml of Levobupivacaine for axillary brachial plexus blockade. Dexmedetomidine has been advised to use as an additive to local anesthetic for brachial plexus block from 1 µg/kg to 100 µg in adults (13, 14). As our study involved USG axial approach to brachial plexus block, 1 µg/kg of Dexmedetomidine was used as an adjuvant with 20 ml of 0.5% of Levobupivacaine.

Kaur et al. showed a decrease in the onset time of motor and sensory block and lower VAS pain scores with the administration of Dexmedetomidine as an adjuvant to Levobupivacaine in supraclavicular

brachial plexus block (15).

Kataria A.P et al. in their study on brachial block comprising patients of age ranging from 2.6 to 90 years. However, in our study, we included patients of age group >20 years (16).

In our study, the mean onset sensory block duration in group LC was 5.90 ±0.81 min, and in group LD was 4.53±1.07min. The mean onset motor block duration in group LC was 8.85±1.81min, and in the group, LD was 7.88±1.29 min. Our study showed that the onset time of sensory and motor was significantly faster in group LD. Similarly, in a survey by Manjunatha C, et al., the onset time of sensory and motor block was shorter in Group D than in Group C, which was significant (P<0.05) (17). Our results concur with Agarwal et al. and Ammar and Mahmoud (13, 14).

However, there are wide variations observed in the onset sensory and motor block durations in different studies. These variations may be due to the usage of other types of local anesthetics, their concentration and volumes, the dose of Dexmedetomidine, and the nature of blocks. Agarwal et al. used Dexmedetomidine 100 µg or 1 ml saline with 30 ml of 0.325% bupivacaine for a supraclavicular block. Ammar and Mahmoud compared bupivacaine 0.33% with dexmedetomidine 0.75µg/kg for infraclavicular

Table 2: Comparison of Sedation score between two groups.

Time of assessment	Score	Group LC (%)	Group LD (%)	χ^2 value, Significance
0 min	1	40 (100%)	40 (100%)	No Difference
	2	0 (0.00%)	0 (0.00%)	
	3	0 (0.00%)	0 (0.00%)	
5 min	1	40 (100%)	40 (100%)	No Difference
	2	0 (0.00%)	0 (0.00%)	
	3	0 (0.00%)	0 (0.00%)	
15 min	1	31 (77.50%)	29 (72.50%)	$\chi^2 = 0.2667$, $p = 0.60$
	2	9 (22.50%)	11 (27.50%)	
	3	0 (0.00%)	0 (0.00%)	
0 min	1	25 (62.50%)	8 (20.00%)	$\chi^2 = 17.71$, $p = 0.0001$, Highly Significant
	2	15 (37.50%)	26 (65.00%)	
	3	0 (0.00%)	6 (15.00%)	
60 min	1	21 (52.50%)	8 (20.00%)	$\chi^2 = 9.179$, $P = 0.01$, significant
	2	15 (37.50%)	26 (65.00%)	
	3	4 (10.00%)	6 (15.00%)	
120 min	1	40 (100%)	40 (100%)	No difference
	2	0 (0.00%)	0 (0.00%)	
	3	0 (0.00%)	0 (0.00%)	
360 min	1	40 (100%)	40 (100%)	No difference
	2	0 (0.00%)	0 (0.00%)	
	3	0 (0.00%)	0 (0.00%)	
720 min	1	40 (100%)	40 (100%)	No Difference
	2	0 (0.00%)	0 (0.00%)	
	3	0 (0.00%)	0 (0.00%)	

χ^2 test

Table 3: Comparison of Systolic, Diastolic blood pressure (mm of Hg), Oxygen saturation (SpO_2 percentage), and Pulse Rate (beats/min) between the two groups.

Time of assessment	Mean \pm SD		Mean Difference	t*	Value p-value
	Group LC	Group LD			
Systolic blood pressure					
0 min	120.20 \pm 6.77	119.40 \pm 6.65	0.800		0.59
5 min	119.50 \pm 7.02	116.90 \pm 6.33	2.625		0.08
15 min	120.00 \pm 5.78	117.78 \pm 5.03	2.225		0.07
30 min	117.85 \pm 5.92	117.52 \pm 5.32	0.325		0.79
60 min	117.80 \pm 6.27	118.70 \pm 5.77	0.900		0.50
120 min	119.15 \pm 6.82	118.45 \pm 6.69	0.700		0.64
360 min	117.07 \pm 5.58	117.70 \pm 5.42	0.625		0.61
720 min	116.85 \pm 17.96	119.15 \pm 5.06	2.300		0.43
Diastolic blood pressure					
0 min	79.35 \pm 6.06	78.75 \pm 5.83	0.50	0.39	0.06
5 min	79.41 \pm 6.25	78.31 \pm 6.23	1.10	0.83	0.072
15 min	79.26 \pm 6.66	79.66 \pm 6.65	0.40	0.26	0.08
30 min	80.55 \pm 6.83	77.85 \pm 6.69	2.700	1.785	0.07
60 min	79.11 \pm 5.91	79.51 \pm 6.43	0.40	0.29	0.065
120 min	79.56 \pm 7.31	79.66 \pm 7.08	0.10	0.06	0.62
360 min	82.15 \pm 5.78	80.10 \pm 6.04	2.050	1.549	0.12
720 min	80.75 \pm 7.01	79.30 \pm 6.71	1.450	0.944	0.34
Oxygen saturation					
0 min	99.03 \pm 0.78	98.81 \pm 0.85	0.22	1.26	0.07
5 min	98.96 \pm 0.91	98.89 \pm 0.63	0.07	0.41	0.06
15 min	99.15 \pm 0.92	99.00 \pm 0.90	0.150	0.70	0.46
30 min	99.12 \pm 0.91	98.87 \pm 0.93	0.250	0.14	0.23
60 min	98.97 \pm 0.90	98.77 \pm 0.80	0.20	1.16	0.07

120 min	99.37±0.83	99.10±0.54	0.275	1.740	0.08
360 min	98.90±0.84	98.83±0.61	0.07	0.43	0.08
720 min	99.03±0.78	98.92±0.75	0.10	0.56	0.063
Pulse Rate					
0 min	82.52±6.46	83.25±5.96	0.725	0.521	0.60
5 min	83.12±6.55	82.45±5.98	0.675	0.481	0.63
15 min	79.92±7.05	78.80±6.58	1.125	0.737	0.46
30 min	76.10±6.58	73.62±6.20	2.475	1.731	0.08
60 min	74.20±6.35	73.30±5.62	0.88	0.58	0.555
120 min	69.82±4.38	68.67±5.73	1.15	1.07	0.28
360 min	70.37±4.48	71.00±4.23	0.63	-1.54	0.12
720 min	76.85±5.48	74.75±5.03	2.100	1.783	0.07

*Students t-test, $p < 0.05$: significant

brachial plexus block against plain bupivacaine.

Another study reported that Dexmedetomidine as an adjuvant to Levobupivacaine is statistically highly significant in the onset of sensory and motor block than Clonidine as an adjuvant ($p < 0.001$) (18).

One research reported that Clonidine and Dexmedetomidine's analgesic efficacy for USG-guided supraclavicular brachial plexus block along with Levobupivacaine. The sensory and motor blockade onset time was significantly earlier in the dexmedetomidine group (3.58 ± 0.61 min. and 7.13 ± 0.89 min.) in comparison to the clonidine group (6.88 ± 0.59 min. and 8.75 ± 0.77 min.). The mean Onset of the sensory blockade and motor blockade was faster in group D in comparison to group C ($P < 0.001$) (19).

The statistical analysis showed that the sensory and motor block duration in group LD was significantly longer than in group LC ($p < 0.0001$). Similar results were found in other similar studies (19-21).

Hosali et al. (2015) also concluded that Dexmedetomidine's addition to Levobupivacaine significantly prolonged sensory block and motor block duration than Clonidine. Karthik et al. also (2015) also found that sensory and motor blockade

was prolonged by adding Dexmedetomidine to Levobupivacaine

In our study, the duration of sensory and motor block was 567.75 ± 62.33 and 560.62 ± 67.19 min, respectively, in group LC, whereas they were 662.50 ± 50.95 , and 625.50 ± 51.95 min, respectively, in group LD. Our results concur with the study of Manjunatha et al. (17).

Manjunatha et al. reported that the duration of sensory and motor block was prolonged (863.8 ± 106.8 min and 758.5 ± 121.6 min) in Group D compared to Group C (335.6 ± 58.6 min and 308.4 ± 71.8 min) ($p < 0.05$).

In group LC, the mean pulse rate ranged from 69.82 ± 4.90 to 83.12 ± 6.55 beats/min. In group LD, the mean pulse rate ranged from 68.67 ± 5.73 to 83.25 ± 5.96 beats/min. In group LC, the mean systolic blood pressure ranged from 116.85 ± 17.96 to 120.20 ± 6.77 mm of Hg, whereas in group LD, the mean systolic blood pressure ranged from 116.90 ± 6.33 to 119.40 ± 6.65 mm of Hg. In group LC, the mean diastolic blood pressure ranged from 80.55 ± 6.83 to 83.12 ± 7.31 mm of Hg, whereas in group LD, the mean diastolic blood pressure ranged from 77.85 ± 6.69 to 80.10 ± 6.04 mm of Hg.

Vinod Hosalli et al. conduct the study using Dexmedetomidine and Clonidine as adjuvants with 1 µg/kg each with Levobupivacaine in the axillary brachial plexus block. In Group LD, a significantly lower pulse rate was observed at 60, 90, and 120 min, compared with Group LC, but not less than 60 beats/min ($P < 0.001$). In Group D, systolic and diastolic blood pressure was significantly lower than the baseline from 30 to 120 min compared with Group C ($P < 0.001$).

There was a slight decrease in pulse rate in our study, 30 min, 60 min, 120 min in the dexmedetomidine group ($P > 0.05$). In our study, the statistical analysis showed no significant difference in systolic and diastolic blood pressure between the two groups ($p > 0.05$)—similar results seen by Gopal Krishan et al.

Esmaoglu et al. observed Bradycardia in their patient group in which 100 mcg of Dexmedetomidine was used with Levobupivacaine. In our study slight decrease in pulse rate in group Dexmedetomidine ($P > 0.05$). The statistical analysis showed no significant difference in systolic and diastolic blood pressure between the two groups ($p > 0.05$).

In our study, no patients had a sedation score of > 4 . Nevertheless, when compared to Clonidine, Dexmedetomidine showed greater arousable sedative effects with a sedation score of 3 was seen. Chi-square analysis showed that the difference in sedation score was highly significant ($p < 0.0001$) at 30 min and 60 min. No side effects in our study due to the low dose of Dexmedetomidine (1mcg/kg). Atul Dixit et al. no patients had a sedation score of 3 and above. Chi-square analysis showed that the difference in sedation score was significant ($P < 0.05$) (22).

In our study, no patients had any hemodynamic disturbances, Bradycardia, or Severe hypotension in either study group. None of the patients experienced any severe complications such as large hematoma, prolonged nerve paralysis, nausea, vomiting, or dry mouth, due to the blocking technique at doses 1.5µg/kg of clonidine and dexmedetomidine (1mcg/kg). No other side effects such as nausea, vomiting, local anesthetic toxicity, hematoma, and respiratory depression were found in either of the groups. In contrast, a study by Manjunatha et al. demonstrated that two patients who had Bradycardia

in Group D were treated with injection atropine-i.v. HR and blood pressure were lower in Group D. The decreased blood pressure is due to inhibition of central sympathetic outflow. The presynaptic α -2 receptors are also stimulated by Dexmedetomidine, thereby decreasing norepinephrine release and causing a fall in blood pressure and heart rate (23,24). Esmaoglu et al. reported the incidence of Bradycardia. In the study of Aggarwal et al. and Kaygusuz et al., Dexmedetomidine provided better hemodynamic stability (25).

In conclusion, dexmedetomidine (1µg/kg) has a superior clinical profile as an adjuvant to Levobupivacaine (0.5%) in contrast to Clonidine (1.5µg/kg) in the block of brachial plexus axillary due to Rapid onset time of sensory and motor block, prolonged duration of sensory and motor block, and greater arousable sedation compared to clonidine group.

Conclusion

We conclude that the addition of Dexmedetomidine (1µg/kg) as an adjuvant to 0.5% levobupivacaine for upper extremity surgeries under ultrasound-guided axillary brachial plexus block provided the rapid onset of sensory block and motor block and enhancement of duration of sensory and motor block with arousable sedation compared to Clonidine (1.5µg/kg) without any hemodynamic variations and adverse events.

Acknowledgment

None.

Conflicts of Interest

The authors declare that they have no conflict of interest.

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