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A Comparative Study of 0.5% Levobupivacaine with Dexmedetomidine 50 MCG and 0.5% Levobupivacaine Plain in Supraclavicular Brachial Plexus Block

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Abstract

Context: Several adjuncts are added to local anaesthetic agents to prolong the duration of regional blockade. In this study, we added dexmedetomidine to levobupivacaine for supraclavicular brachial plexus blocks. *Aim:* To compare the effectiveness of 50 mcg dexmedetomidine added to 0.5% levobupivacaine in supraclavicular brachial plexus block for upper limb surgeries with respect to onset and duration of motor and sensory blockade. *Settings and Design:* We conducted a randomized double blinded controlled trial on 40 patients, undergoing supraclavicular block. *Methods and Material:* Group A (n=20) received 30 ml of 0.5% levobupivacaine with 0.5 ml of normal saline and Group B (n=20) received 30 ml of 0.5% levobupivacaine with 0.5 ml of dexmedetomidine (50mcgs/0.5ml). Time of onset, completion and duration were noted for both sensory and motor blockade. *Statistical Analysis Used:* Chisquare test and student t-test and p value <0.05 was considered as significant. *Results:* Onset of motor and sensory blockade was significantly shorter with Group B (0.5% levobupivacaine with dexmedetomidine). The mean duration of sensory and motor blockade was significantly prolonged with 591± 30min. and 543±32min respectively. The mean sedation score was 2.60±0.503 (patients were sedated and arousable to verbal or mild physical stimulus) in Group B whereas the score was 1 (fully awake) in Group A. All patients were hemodynamically stable. *Conclusion:* We conclude that addition of dexmedetomidine 50mcgs to 0.5% levobupivacaine fastens the onset and prolongs the duration of motor and sensory blockade and provides good intraoperative sedation

Keywords: Supraclavicular Brachial Plexus Block; Levobupivacaine; Dexmedetomidine; Nerve Locator.

Introduction

Regional block offers several advantages over general anaesthesia for upper limb surgeries and is preferred by patients for intraoperative and postoperative analgesia. Levobupivacaine, s-enantiomer of bupivacaine, is less cardiotoxic and neurotoxic, attributed to its faster protein binding nature. Adjuvants are added to the local anaesthetic agents to augment the onset and duration of the blockade. An $alpha_2$ receptor agonist dexmedetomidine is one such adjuvant which can prolong the duration of blockade so that it reduces the analgesic requirement in the postoperative period.

We had used the lower dose of dexmedetomidine in our study to avoid unwanted side effects such as bradycardia and hypotension. In this study, we aimed to investigate the effects of adding dexmedetomidine to levobupivacaine for supraclavicular brachial plexus block using nerve locator. We hypothesized that adding dexmedetomidine will prolong the duration of anesthesia and analgesia with a shorter onset time [1-6].

Aim of the Study

The aim of the study is to compare the effectiveness of adding an adjuvant dexmedetomidine 50 mcg to

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0.5% Levobupivacaine over 0.5% levobupivacaine alone in supraclavicular brachial plexus block. The objectives of this study are to observe the time of onset, completion and duration of both sensory and motor blockade, hemodynamic stability and any adverse effects.

Materials and Methods

The study was performed after obtaining ethical committee approval and written informed consent of the patients. In this study, 40 patients with American Society of Anesthesiologists(ASA) physical status I/ II who were scheduled to undergo forearm and hand surgery in which an supraclavicular block was to be used were enrolled in this prospective, double blind, controlled trial. The study was done in a tertiary care hospital in Chennai.

Randomization

Patients were randomly allocated into two groups by using sealed envelopes. The group allocation was concealed in sealed envelopes which were opened just before administration of the block.

Inclusion Criteria

- ASA 1 and 2
- 20 – 50 years
- Both sexes
- Weight 50 – 70 kilograms
- Elbow, Forearm and Hand surgeries

Exclusion Criteria

- Patient refusal
- ASA 3 and above
- Allergy
- Infection
- Coagulopathy
- Pneumothorax

Group A (n=20): 20 patients received 30 ml of 0.5%levobupivacaine with 0.5ml of normal saline.

Group B (n=20): 20 patients received 30 ml of 0.5% levobupivacaine with 0.5ml of dexmedetomidine (50mcgs/0.5ml).

Blinding: The drug solution was prepared by an anaesthesiologist not involved in the study. An experienced anaesthesiologist who had previous experience >50 supraclavicular blocks with nerve stimulator technique, performed all the blocks. Both the patient and the anaesthesiologist performing the block, were unaware of the assigned group. An independent observer (not included in the study) then observed the onset and offset of sensory and motor blockade and VAS score post-operatively. Blinding was opened at the end of the study.

Patients were kept for overnight fasting for 6-8 hrs. Peripheral venous access was obtained in the non-operative hand with 18G cannula and connected to the standard monitoring ECG, Pulse Oximeter and NIBP.

The patient was placed in supine position with head turned to 45 degree contralateral side. A small roll was placed underneath the shoulder. Using the subclavian perivascular technique described by Kulenkampff, modified by Winnie and Collins, the supraclavicular brachial plexus block was performed. With the standard peripheral nerve stimulator, the brachial plexus was located with 2 Hz and 1.0 mA. Once the desired muscle response occurs, the current was then reduced to 0.6 mA and if still the muscle contraction persists, the local anaesthetic agent is injected in increments of 5ml after intermittent negative aspiration of blood. Complications like pneumothorax, Horner's syndrome, vascular puncture and phrenic nerve palsy were noted. Patient's heart rate, Blood pressure and SpO2 were recorded at the baseline, after the block is administered and every 5 min thereafter till the end of the surgery. Any episode of hypotension (fall in MAP of more than 20% from the baseline), bradycardia (HR<50) and desaturation (SpO2<90) was documented.

After injecting the local anaesthetic, motor block is evaluated by thumb abduction (Radial nerve), thumb adduction (Ulnar nerve), thumb opposition (Median nerve) and flexion of the elbow in supination and pronation of the forearm (Musculocutaneous) every 5 minutes till 30 minutes.

Sensory blockade was evaluated in the same time interval for same duration. Sensory blockade is graded according to Hollmen's scale: 0-normal sensation of pin prick, (+) -pin prick felt as sharp pointed but weaker compared with the same area in other extremity, (++) - pin prick is felt as touch with blunt object, (+++)- no perception of pin prick.

Motor blockade (Grade)

- Normal muscle function
- Slight depression in muscle function as compared with pre-anaesthetic power

- Very weak muscle action persisting in muscle
- Complete block with absent muscular function

Onset of sensory blockade is defined as the time since local anaesthetic administration until loss of pain sensation in all four nerve territories. Onset of motor blockade is defined as the time since local anaesthetic administration until the loss of movement in arm and forearm.

A successful blockade was defined as a complete sensory and motor blockade in all the regions within 30 minutes from the time of blockade. Patients with incomplete block or failed block were excluded from the study. Duration of sensory block was defined as the time interval between the sensory block onset and complete resolution of anaesthesia off all nerves. Duration of motor block was defined as the time interval between the motor block onset and recovery of complete motor function of the upper limb.

After completion of the surgery, the patients were monitored in Post Anaesthesia Care Unit for 24 hours by an independent anaesthesiologist not involved in the study. Heart rate and Blood pressure were recorded every 30 minutes till 2 hours and every hour for 6 hours, every 2 hours until 12 hours and then at 24 hours postoperatively.

Post-operative pain was assessed at the same time with visual analogue scale. Injection diclofenac 3ml IM was given if VAS >4. Sedation was assessed using the *sedation score* by *Culebras et al* where sedation was graded on a scale of 1 – 5 as follows.

- Awake & alert
- Sedated, responding to verbal stimulus
- Sedated, responding to mild physical stimulus
- Sedated, responding to moderate or severe stimulus, not arousable

Statistical Methods

Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean±SD and results on categorical measurements are presented in percentage (%).

Chi-square test has been used to find the significance of study parameters on categorical scale between two groups. Student 't' test has been used to determine the significance between two group means. All analysis were two tailed and $p < 0.05$ was considered significant. SPSS version 16.0 was used for data analysis.

Results

The demographic data were comparable in both the groups with respect to age, sex and weight.

The addition of dexmedetomidine to levobupivacaine has fastened the onset time of motor and sensory block in Group B ($p<0.05$). There was a significant difference in the completion of sensory and motor block among the groups.

The duration of sensory and motor block was prolonged with the dexmedetomidine group with 591.5±28.814 min. and 543±32.135 min respectively. Whereas the duration of sensory and motor block in plain levobupivacaine group was significantly less when compared to dexmedetomidine group.

Sedation scores differed between the two groups during the intraoperative period (at 30 minutes). None of the patients were sedated in group A with mean sedation score of 1.

The Mean sedation score was 2.60 ± 0.503 in Group B (patients were sedated and arousable to verbal or mild physical stimulus). No patients needed airway assistance to maintain the airway patency. Sedation score was statistically significant during the intraoperative period (p value < 0.05).

Group B patients recorded a lower mean VAS score than their counterpart. Likewise the rescue analgesic requirement was lower in Group B compared to Group A. Both were statistically significant ($p < 0.05$). The mean pulse rate is significantly low in group B at different time intervals except at 5 min and 10 min where there is no statistically difference exists among the groups (Figure-1). Bradycardia requiring atropine administration was not reported in any of the cases in the study.

Table 1: Demographic data

	Group A (n=20)	Group B (n=20)	p-value
Age(y), Mean ± SD	34.90±8.422	34.90±8.896	1.000
Weight(Kg) ± SD	59.60±4.570	60.70±5.202	0.482
Sex (M:F), n	15:5	14:6	1.00
Duration of surgery(min), Mean ± SD	134.75±19.25	137.25±22.911	0.711

Table 2: Parameters

Mean ± SD	Group A	Group B	P value
Onset of sensory blockade	7.70±0.979	5.10±0.788	0.000
Onset of motor blockade	10.45±1.050	6.55±0.759	0.000
Completion of sensory blockade	10.95±1.234	7.05±0.826	0.000
Completion of motor blockade	13.55±1.234	8.55±0.887	0.000
Duration of sensory blockade	372±28.023	591.5±30.826	0.000
Duration of motor blockade	327.50±28.814	543±32.135	0.000

Table 3: Sedation score

	Group	N	Mean	Standard deviation	p-value
Sedation Scores	Group A	20	1.00	.000	0.000
	Group B	20	2.60	.503	

Table 4: Vas Score

	Group	N	Mean	Std. Deviation	p-value
VAS 6hr	Group A	20	5.35	.875	0.000
	Group B	20	.20	.410	
VAS 12hr	Group A	20	7.65	.671	0.000
	Group B	20	3.70	.470	
VAS 24hr	Group A	20	10.00	.000	0.000
	Group B	20	8.80	1.005	

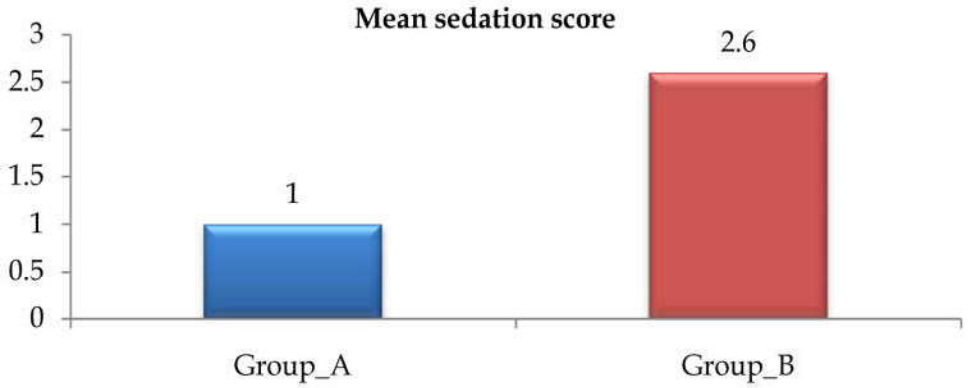

Fig. 1: Sedation score

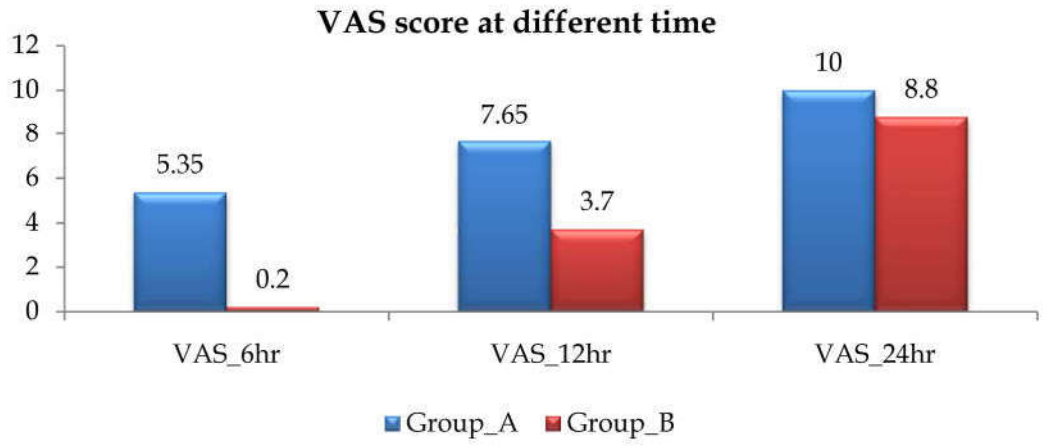

Fig. 2: VAS

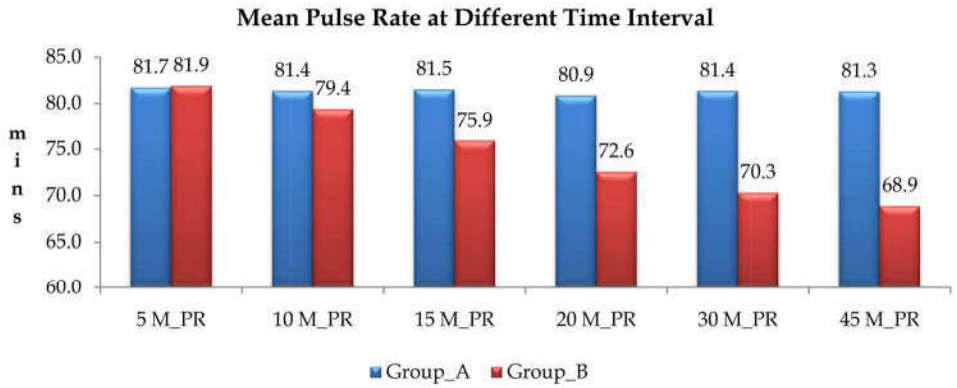

Fig. 3: Mean pulse rate

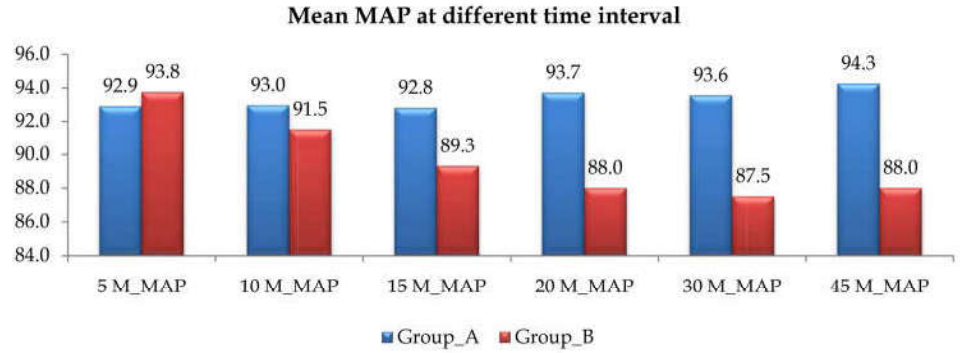

Fig. 4: MAP

Table 5: Pulse rate

	Group	N	Mean	Standard deviation	p-value
5 M_PR	Group A	20	81.70	6.974	0.931
	Group B	20	81.90	7.469	
10 M_PR	Group A	20	81.35	6.588	0.371
	Group B	20	79.35	8.087	
15 M_PR	Group A	20	81.50	4.926	0.018
	Group B	20	75.90	8.669	
20 M_PR	Group A	20	80.85	5.905	0.003
	Group B	20	72.55	10.065	
30 M_PR	Group A	20	81.35	5.566	0.000
	Group B	20	70.30	10.484	
45 M_PR	Group A	20	81.30	4.975	0.000
	Group B	20	68.85	9.483	

Table 6: MAP (Mean arterial pressure)

	Group	N	Mean	Standard deviation	p-value
5 M_MAP	Group A	20	92.90	5.025	0.605
	Group B	20	93.75	5.280	
10 M_MAP	Group A	20	92.95	3.993	0.346
	Group B	20	91.50	5.492	
15 M_MAP	Group A	20	92.80	3.139	0.012
	Group B	20	89.30	4.985	
20 M_MAP	Group A	20	93.70	3.080	0.000
	Group B	20	88.00	5.026	
30 M_MAP	Group A	20	93.55	2.762	0.000
	Group B	20	87.85	4.452	
45 M_MAP	Group A	20	94.25	3.307	0.000
	Group B	20	88.00	4.078	

The mean values of mean arterial pressure were statistically significant (p value < 0.05) except in 5 minutes, 10 minutes and 24 hours measurements. MAP levels in Group B were significantly lower than those in Group A .

No patient in either of the groups reported of hypotension and the usage of vasopressors. The mean values of SpO_2 were comparable among the groups and there was no incidence of desaturation. There was one case of arterial puncture while performing the block with no hematoma formation. No other complications were reported in the study.

Discussion

In our study, the addition of dexmedetomidine had significantly fastened the onset time for sensory and motor block and prolonged the duration of sensory and motor block and lowers the VAS score and rescue analgesia requirement in the post operative period.

Dexmedetomidine has been used as an adjunct in central neuraxial blockade and peripheral nerve blocks. The mechanism by which an $alpha_2$ receptor agonist Dexmedetomidine produces analgesia and sedation is multifactorial. It produces analgesia peripherally by reducing the nor-epinephrine release and inhibiting the nerve action potentials. Centrally, analgesia is produced by inhibiting substance P in the dorsal horn cells and by activating the $alpha_2$ receptors in the locus coeruleus.

Nallam et al [9] has compared the efficacy of two different concentrations of dexmedetomidine 50 mcg and 100 mcgs when used as an adjunct with 0.5% levobupivacaine in supraclavicular block and concluded that higher incidences of significant sedation and bradycardia were reported with 100 mcgs of dexmedetomidine. Hence in our study, we decided to evaluate the efficacy of dexmedetomidine in concentration of 50 mcg rather than 100 mcgs.

Esmaoglu et al divided 60 patients who had been scheduled to undergo forearm andhand surgery using an axillary block into 2 groups. They administered 0.5% 40 ml levobupivacaine plus 1 mL saline solution in 1 group and 0.5% 40 mL levobupivacaineplus 100 µg dexmedetomidine in other group. Their study differs from our study in the dexmedetomidine dose that we used (0.5µg/kg dexmedetomidine). Esmaoglu et al found that adding dexmedetomidine to levobupivacaine for an axillary brachial plexus block shortens both the sensory and motor block onset time, extends the block duration, and the analgesia period. Our correlates with Esmaoglu et al [1]. We showed that by adding 0.5µg/kg dexmedetomidine to 0.5% levobupivacaine had hastened block onset time and also the duration of the block significantly .We used 0.5% Levobupivacaine in our study, similar to other studies whereas Waindeskar et al used 0.325% levobupivacaine. As Waindeskar et al performed USG guided supraclavicular approach of brachial plexus block, he had reduced the concentration of levobupivacaine but the volume of drug was similar to our study. 30 ml was volume of the drug used in our study which was similar to Nallam et al, whereas Esmaoglu et al used 40 ml of 0.5% levobupivacaine with dexmedetomidine.

Haramritpal Kaur et al [8] conducted a study on 90 patients who were undergone upper limb surgeries under supraclavicular nerve block using nerve locator. They had shown that the onset of sensory and motor blockade was 7.6±1.006 min and 8.3± 0.877 min in group A, while it was 6.96±1.077 min an 7.6 ± 1.1 min in group B, respectively. The difference was statistically significant ($P < 0.05$). Which was similar to our study. They also concluded that addition of 1 mcg/kg dexmedetomidine to 0.25% levobupivacaine for supraclaviclar plexus block shortens sensory, motor block onset time and motor block durations, extends sensory block, and analgesia durations. Reduction in total levobupivacaine dose also increases the safety margin of the block. Duration of motor block was determined by the concentration of levobupivacaine used. Since they had used 0.25% they hadn't got any significant prolongation of motor block duration in contrast to our study.

The duration of sensory and motor block was prolonged with the dexmedetomidine group with 591.5±30.826 min. and 543±32.135 min (9 hours) respectively when compared to plain levobupivacaine group. This is in disagreement with other studies where the addition of 100 mcg of dexmedetomidine to levobupivacaine 0.5% has prolonged the duration of motor blockade for 13-14 hours and duration of sensory blockade for 14-15 hours. In a study done by Soumya et al[7] showed that duration of motor blockade was 512±60.13 in levobupivacaine (0.5% levobupivacaine 35ml)group compared to 840±50.23 in dexmedetomidine group (35ml levobupivacaine 0.5% added to 100µg of dexmedetomidine). Duration of sensory block also prolonged significantly (645±70.11 in group A & 898±32.33 in group B)

The patients in the dexmedetomidine group were sedated but arousable to verbal commands. None of the patients desaturated and required any airway

assistance. Dexmedetomidine produces sedation by central action by activation of presynaptic Alpha$_2$ receptors in locus cerulus by inhibiting the release of norepinephrine.

VAS and Rescue Analgesia requirement was less with dexmedetomidine group when compared to plain levobupivacaine group. Similar to other studies, the heart rate and mean arterial pressure was significantly low in the group B when compared to group A but none of the patients required any intervention. Hypotension and bradycardia were explained by the decrease in sympathetic activity due to the stimulation of alpha$_2$ receptors by dexmedetomidine. There was one case of arterial puncture while performing the block with no hematoma formation. No other complications were reported in the study.

Ultrasound guided brachial plexus block could have reduced the Levobupivacaine volume with the advantage of injecting the drug close proximal to the nerve fibres but it requires greater technical expertise.

Conclusion

We conclude that Dexmedetomidine 50mcg when added to 0.5% Levobupivacaine for brachial plexus block is found to shorten the onset of sensory and motor blockade and prolongs the duration of sensory and motor blockade. It produces a good intraoperative sedation and also helped to reduce the post-operative analgesic requirement.

Dexmedetomidine can be safely added as an adjuvant to local anaesthetic drugs for brachial plexus block with good perioperative monitoring

Acknowledgement

We like to acknowledge our patients who had participated in the study

Conflict of Interest: Nil

Key Messages

Dexmedetomidine prolongs the sensory blockade in supraclavicular block for >8hrs, making it an ideal choice of additive when catheter is not used to prolong analgesia.

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