A Comparative Study between Levobupivacaine with Dexmedetomidine Versus Levobupivacaine with Clonidine in Ultrasound Guided Supraclavicular Brachial Plexus Block for Upper Limb Surgeries: A Randomized Double Blind Placebo Controlled Study

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ABSTRACT

Introduction: Brachial plexus block is more advantageous for routine as well as emergency upper limb surgery. This provides a useful alternative to general anesthesia for upper limb surgeries. Brachial plexus block provides very good intraoperative anesthesia as well as postoperative analgesia without any significant systemic side effects. This study was conducted to compare the perioperative analgesic efficacy of clonidine and dexmedetomidine for supraclavicular brachial plexus block along with levobupivacaine.

Material and methods: A randomized double blind controlled study was done on 90 patients of ASA Grade I or II undergoing upper limb surgery. Group L – received levobupivacaine 0.5% (25ml) 125 mg and 1.0 ml normal saline, Group C–received levobupivacaine 0.5% (25ml) 125 mg and 1.0ml (150 microgram) clonidine and Group D – received levobupivacaine 0.5% (25ml) 125 mg and 1.0ml (100 microgram) dexmedetomidine. Onset and duration of both sensory and motor blockade and duration of analgesia were studied in all the three groups.

Results: It was observed that in group D, onset of motor and sensory blockade was faster than group L and C. Significant difference was not observed in heart rate and blood pressure in any of the Groups. Group D had longer duration of analgesia in comparison of group C and group L.

Conclusion: We concluded that dexmedetomidine added to levobupivacaine in supraclavicular brachial plexus block enhanced the duration of sensory and motor blockade and also the duration of analgesia.

Keywords: Clonidine, Dexmedetomidine, Levobupivacaine, Ultrasound, Supraclavicular Brachial Plexus Block

INTRODUCTION

Brachial plexus block is the most commonly practiced peripheral nerve blocks. Halsted first injected the cocaine in brachial plexus under direct vision in 1885. [1]

Brachial plexus block is more advantageous for routine as well as emergency surgery in upper limb. This provides a useful alternative to general anesthesia for upper limb surgeries. Brachial plexus block provides very good intraoperative anesthesia as well as postoperative analgesia without any systemic side effects. [2]

Brachial plexus block is very popular and widely used regional nerve block of upper extremity because it avoids the unwanted effects of anesthetic drugs used during general anesthesia and the stress of laryngoscopy and tracheal intubation. Patients can also enjoy a post operative period free from nausea, vomiting, cerebral depression and immediate postoperative pain.

This achieves near ideal operative conditions by producing complete muscular relaxation maintaining stable intraoperative conditions and sympathetic blockade which reduces post operative pain, vasospasm and edema. Levobupivacaine is most frequently used local anesthetic agent as it has a longer duration of action varying from 3-8 hours. However it has limiting factors like delayed onset, patchy and incomplete analgesia. To minimize these drawbacks many drugs have been added to levobupivacaine to improve quality and duration of action and postoperative analgesia.

Addition of vasoconstrictors like α-adrenergic agonists, opioids, neostigmine, haylorunidase etc may enhance the quality and prolong the duration of brachial plexus block. All these drugs prolong the duration of brachial plexus block but various side effects were recorded. So the search for ideal additive continues and novel alpha 2 adrenergic agonists were tried by many workers.

The purpose of this prospective randomized double blind placebo controlled study was to compare the onset of motor and sensory blockade, duration of motor and sensory blockade and duration of perioperative analgesia with levobupivacaine 0.5% alone and with dexmedetomidine 100 µg or clonidine 150 µg as an adjuvant to levobupivacaine 0.5%.

Alpha 2 adrenergic agonists have sedative, analgesic, sympatholytic and cardiovascular stabilizing properties and they also reduce intraoperative anesthetic requirements. These can be given in epidural, intrathecal and peripheral nerve blocks either alone or with local anesthetic agents to decrease the time of onset of blockade, to prolong the

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duration of blockade and to improve the quality of the nerve block.

Dexmedetomidine, a potent α2 adrenergic agonist, is 8 times more selective α2 agonist than clonidine. [3] In many animal and human studies, dexmedetomidine as well as clonidine, had been reported to enhance onset and duration of sensory and motor blockade and duration of analgesia when used as an adjuvant to local anesthetic agents in peripheral nerve blocks. [4-11]

The aim of this randomized double blind controlled study was to compare the effects of dexmedetomidine or clonidine as an adjuvant to levobupivacaine in supraclavicular brachial plexus block.

MATERIAL AND METHODS

The study was conducted on 90 patients of American Society of Anesthesiologists (ASA) grade I or II, adult of either sex, in the department of anesthesia and critical care, Rohilkhand Medical College and Hospital, Bareilly and cases were selected from orthopedics patients going to be operated under supraclavicular brachial plexus block. The study was conducted in three groups of 30 patients each. The patients were randomly assigned using "computer generated random number table" to one of the following groups:

Group L – Received levobupivacaine 0.5% (25ml) 125 mg and 1.0 ml normal saline.

Group C – Received levobupivacaine 0.5% (25ml) 125 mg and 1.0ml clonidine.

Group D – Received Levobupivacaine 0.5% (25ml) 125mg and 1.0ml dexmedetomidine.

Approval from institutional ethical committee was taken before starting the study and informed written consent was taken from all patients.

Exclusion Criteria

1. Uncontrolled diabetes mellitus.
2. Labile blood pressure.
3. Hypersensitivity to the drug.
4. Age younger than 14 years.
5. Pregnant and lactating women.
6. Infection at the site of puncture.
7. Patients on adrenoreceptor agonist or antagonist therapy.
8. Significant neurological, psychiatric, neuromuscular, cardiovascular, pulmonary, renal or hepatic disease.

Anesthesia technique

Pre-anesthetic checkup was done and patient was informed about the procedure. Tab. alprazolam 0.5 mg was given evening before surgery and at 5 Am in the morning with a sip of water. IV line was secured with 18 Gauze IV cannula in healthy forearm and IV fluid was started. The patient was connected to all the standard monitors to record pulse rate, O_2 saturation, NIBP and ECG. Premedication with inj. Midazolam 0.05 mg/kg body weight before the procedure was given. Drug solutions were prepared by an independent anaesthesiologist according to group of the patient. Base line heart rate, blood pressure and oxygen saturation were recorded.

After strict aseptic precautions, at a point 1.5 to 2.0 cm posterior and cephalad to midpoint of clavicle, subclavian artery pulsation was felt. A skin wheel was raised with local anesthetic cephalo-posterior to the pulsations. Ultrasound machine was prepared and using clavicle as landmark a high frequency linear probe was positioned in supraclavicular fossa and pulsation of subclavian artery was located. The area lateral and superficial to subclavian artery brachial plexus was identified as honey combing structure. The needle was inserted from lateral side of probe and advanced inside the ultrasound beam by in plane technique till the plexus was visualized. Following negative aspiration, 25 ml of prepared drug solution was injected.

The onset of sensory blockade was defined as the time between injection and complete loss of pin prick sensation in C_2 to T_2 dermatome. The time when complete sensory blockade achieved was noted. Sensory blockade was graded as-[0= Sharp pin prick sensation felt, 1= dull pin prick sensation felt, 2=No pin prick sensation felt].

Motor blockade was assessed by bromage three point score [0= normal motor function with full flexion and extension of elbow, wrist and fingers, 1= decreased motor strength with ability to move fingers and/or wrist only, 2= complete motor blockade with inability to move fingers or wrist]. The time when complete motor blockade achieved was noted.

Duration of sensory blockade (till appearance of pin prick sensations), duration of motor blockade (till complete return of muscle power) and duration of analgesia (first feel of pain by patient) was also recorded.

STATISTICAL ANALYSIS

Sample size was estimated before study using duration of analgesia as a primary outcome. A sample size of 90 patients was required at $\alpha = 0.05$, $\beta = 0.001$ and power of study 95%. Statistical analysis was performed using the SPSS software package (Chicago, IL, version 17). Patient characteristics were analyzed using Chi-square test (age), analysis of variance ANOVA(weight and duration of surgery), Pearson Chi-square test(gender) and Fisher's exact test(ASA Grade). Hemodynamic changes during intraoperative period were analyzed using one way ANOVA with Tukey-Kraemer corrections. The characteristics of sensory and motor blockade were analyzed by analysis of variance for repeated measures with Tukey-Kraemer test for multiple comparisons. Chi-square test with appropriate corrections was used for analysis of dichotomous variables. Continuous variables were presented as mean ± standard deviation. Ordinal data were presented as median (range) or as count (Percentage). P-value <0.05 was considered significant and P- value <0.001 was considered highly significant.

RESULTS

All the three groups were comparable in the terms of Age, Gender, Weight and ASA Grade as shown in the above table and no statistically significant difference was found (P-value >0.05) (Table 1).

Duration of surgery was comparable in all the three groups and did not show any significant difference. (P-value >0.05) (Table 2).

Onset of motor blockade was faster in group C and D in comparison to group B (P-value <0.001) (Table 3) and it was further faster in group D in comparison to group C (P-value <0.001) (Table 4).

Duration of motor blockade was longer in group C and D in comparison to group B (P-value <0.001) (Table 3) and it was further longer in group D in comparison to group C (P-value <0.001) (Table 4).

Onset of sensory blockade was earlier in group C and D in comparison to group B (P-value <0.001) (Table 3) and it was much earlier in group D in comparison to group C (P-value <0.001) (Table 4).

Duration of sensory blockade was longer in group C and D in comparison to group B (P-value <0.001) (Table 3) and it was longest in group D (P-value <0.001) (Table 4).

Duration of analgesia was longer in group C and D in comparison to group L (P-value <0.001) (Table 3) and it was further longer in group D in comparison to group C (P-value <0.001) (Table 4).

Mean pulse rate was comparable in all the three groups on starting of procedure. Pulse rate changes during the entire intraoperative period was statistically not significant in all the three groups (P-value > 0.05) (Figure 1).

Mean systolic blood pressure was comparable in all the groups on starting of procedure. There was slight decrease in systolic blood pressure in group D which was statistically not significant (P-value >0.05) (Figure 2).

Mean Diastolic blood pressure was comparable in all the groups on starting of procedure. Diastolic blood pressure changes during the entire intraoperative period was statistically not significant in all the three groups (P-value >0.05) (Figure 3).

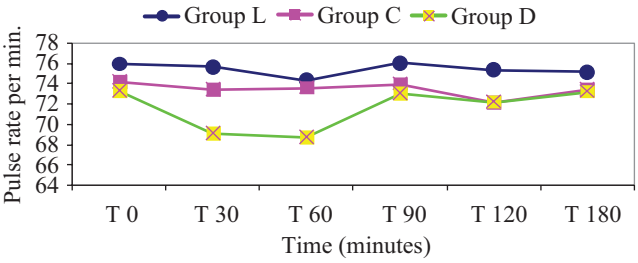

Figure-1: Comparison of mean pulse rate in group L,C and D

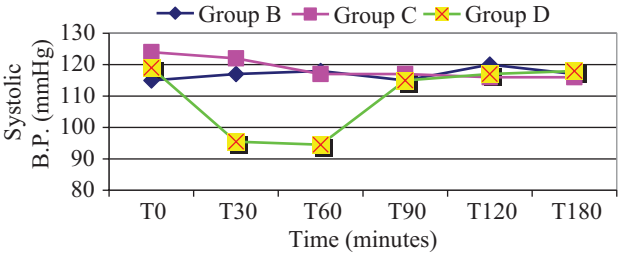

Figure-2: Comparision of mean SBP in group L,C and D

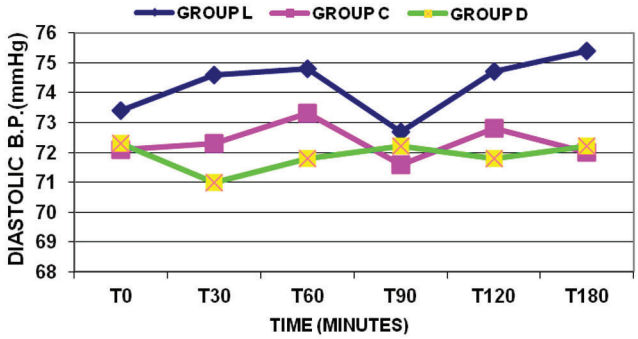

Figure-3: Comparision of diastolic blood pressure group L,C and D

Parameters	Group L (n=30)	Group C (n=30)	Group D (n= 30)	p value
Age (Years)	40.3±18.6	41.6±16.0	41.2±17.2	0.956
Weight(Kg)	57.0±5.7	59.5±6.0	58.7±5.7	0.239
Gender (M/F)	20/10	23/07	23/07	0.599
ASA (I/II)	26/04	26/04	25/05	0.914

Table-1: Demographic data

Group	Group B (n=30)	Group C (n=30)	Group D (n=30)	p value
Mean±SD (Minuts)	121.5±19.5	114.9±14.5	117.0±23.4	0.410

Table-2: Duration of surgery (minutes)

Variables	Group B	Group C	Group D	p value
Onset of sensory blockade	9.95±2.8	6.88±0.59	3.58±0.61	<0.001
Onset of motor blockade	15.06±4.35	8.75±0.77	7.13±0.89	<0.001
Duration of sensory blockade	107.3±9.4	242.5±11.7	471±15.1	<0.001
Duration of motor blockade	140.8±13.6	295.9±13.5	548.0±26.6	<0.001
Duration of analgesia	186.5±15.5	336.4±12.8	714.7±21.5	<0.001

Table-3: Onset and duration of motor and sensory blockade and duration of analgesia mean±S.D. (minutes)

Variables	Mean Difference	Standard Error	p value
Onset of sensory blockade	3.233	0.463	<0.001
Onset of motor blockade	3.5	0.684	<0.001
Duration of sensory blockade	228.5	3.521	<0.001
Duration of motor blockade	252.2	4.88	<0.001
Duration of analgesia	378.3	4.390	<0.001

Table-4: Comparison of clonidine and dexmedetomidine (Group C and Group D)

DISCUSSION

In this randomized double blind study we compared two α 2 agonist drugs dexmedetomidine and clonidine as an adjuvant to levobupivacaine in ultrasound guided supraclavicular brachial plexus block and found that there was significantly early onset, prolonged duration of sensory and motor blockade and analgesia in dexmedetomidine group as compared to clonidine group and placebo.

The role of clonidine as an adjuvant to local anesthetic in upper limb peripheral nerve blocks has been extensively studied. Eldgem et al and Murphy et al used dose range of 30-300 µg in various studies and found that doses up to 150 µg are associated with minimal side effects. [13-14] So we decided to use 150 ug of clonidine as an adjuvant to levobupivacaine in supraclavicular brachial plexus block.

In our study, the onset of sensory and motor blockade was significantly earlier in the clonidine group (6.88 ± 0.59 min. and 8.75±0.77 min.) in comparison to levobupivacaine alone (9.95±2.8min. and 15.06±4.35min) (Table no. 3).

Bernard et al, S. singh et al and Lohom et al also have reported early onset of sensory and motor blockade with the use of clonidine(1-2 µg /kg) as an adjuvant to Local Anesthetic in comparison to local anesthetic alone. [15-17] However, Gaumann et al, Singelyn et al, El Saied et al, Murphy et al, and Hutschala et al have reported that clonidine did not hasten the onset of block irrespective of the dosage used.[14, 18-21] Several explanations are possible for these variable observations. Duma A. et al. suggested that it may be due to responders and nonresponders to the drug and interpatient variation in anatomy of the plexus sheath or nerve, chosen block technique or unclear mechanism of action of clonidine in peripheral nerve blocks. [22]

In our study the mean duration of sensory and motor blockade was 242.5±11.7 min. and 295.9±13.5 min. in clonidine group and 107.3±9.5min. and 140.8±13.62 min. with levobupivacaine alone (Table no.3). The mean duration of analgesia was 336.4±12.8 min. in clonidine group and 186.5±15.5min. with levobupivacaine alone (Table no.3). This can be explained by the fact that clonidine increases potassium permeability and blocks the conduction of type A and C fibers. Besides, clonidine also enhances lidocaine induced inhibition of C-fiber compound action potential. Lipophilic nature of clonidine allows rapid absorption into cerebrospinal fluid and binding to alpha 2-adrenoceptor of spinal cord causing blockade at primary afferent terminals (both spinal as well as peripheral nerve endings). Similar to our study, both El Saied et al. and Hutschala et al. observed longer duration of analgesia and motor blockade with the use of clonidine as an adjuvant as compared to placebo.[20-21] An increase in the duration of postoperative analgesia was also observed by Bernard et al., Lohom et al., Singelyn et al. and Iskandar et al. [15, 17, 19, 23] They observed a linear increase in the duration of analgesia from 0.1 µg/kg to 0.5 µg /kg clonidine but not with 1 and 1.5 µg /kg, indicating no further increase in analgesia with increasing dose. Results contrary to our study were also reported by Gaumann et al. and Duma et al. [18, 22] Prolonged motor blockade with higher clonidine dose is beneficial in long duration surgeries. However, it can be detrimental in ambulatory settings, wherein early mobilization causes early rehabilitation.

S. chakra borty et al. in a randomized controlled trial used clonidine 30 mcg as an adjuvant to bupivacaine in supraclavicular brachial plexus block and found that duration of sensory and motor blockade (279.1±28.98 and 330.4 ±31.68 min.) was significantly prolonged as compared to bupivacaine alone (116.0±17.16 and 144.8±17.31 min.). Duration of analgesia (415.4 ± 38.18) was also significantly longer in comparison to bupivacaine alone (194.2±28.74 min.). [24]

In our study the hemodynamic parameters were comparable in all the groups (figure1-3). Hemodynamic parameters (heart rate, systolic and diastolic blood pressure and mean arterial pressure) remained stable at all times in most of the patients both intraoperatively and postoperatively in our study. Stable hemodynamic parameters were also reported by Murphy et al., Lohom et al., El Saied et al. and Duma et al. [14, 17, 20, 22] however Bernard et al. found 30% fall in SBP with 300 µg, 20% fall with 90 µg and 15% fall with 30 µg clonidine.[15] Similar to our study, S. singh et al., Duma et al. and kohli et al. concluded that 150 µg clonidine can be used as an adjuvant to bupivacaine in indoor patients without significant hypotension.[16, 22, 25] Few transient episodes of bradycardia and hypotension were observed by Iskandar et al. [23]

In our study sedation was not observed in any of the patient receiving clonidine 150 ug. Ramsay sedation score 1-2 was observed in maximum patients throughout the observation period. Sedative properties of clonidine is dose dependent, centrally mediated and is attributable to its lipophilic nature resulting in systemic absorption when administered perineurally. Santvana Kohli et al. also used up to 150 µg clonidine safely but in the same study, 4 out of 14 patients had episodes of hypoxemia (SpO_2 < 90% lasting 20 seconds or more) with the highest dose of clonidine (300 µg) due to sedation. [25] However Bernard et al. and Gaumann et al. reported significant sedation with the use of clonidine than with local Anesthetic alone. [15, 18]

Dexmedetomidine, another potent alpha 2 adrenergic agonist, is 8 times more selective alpha 2 agonist than clonidine. [3] In many studies, dexmedetomidine had been reported to enhance onset and duration of sensory and motor blockade and duration of analgesia when used as an adjuvant to local anesthetic agents in peripheral nerve blocks. [26-29] But very few studies are available to demonstrate its superiority in comparison to clonidine.

In our study the onset of sensory and motor blockade was significantly earlier in the dexmedetomidine group (3.58 ± 0.61 min. and 7.13 ± 0.89 min.) in comparison to clonidine group (6.88 ± 0.59 min. and 8.75±0.77 min.). The mean duration of sensory and motor blockade was longer (471.0±15.1 min. and 548.0±26.6 min.) in dexmedetomidine group in comparison to clonidine group (242.5±11.7 min. and 295.9±13.5 min.). The mean duration of analgesia was

also longer in dexmedetomidine group (714.7±21.5 min.) in comparison to clonidine group (336.4±12.8min.). (Table no.3)

Our results were same as Sarita S. Swami et al. who used dexmedetomidine 1 µg/kg and clonidine 1 µg/kg with bupivacaine 0.25% (35 cc), and found that duration of sensory and motor blockade was 227.00±48.36 and 292.67±59.13 min, respectively, in clonidine group, while it was 413.97±87.13 and 472.24±90.06 min. respectively, in dexmedetomidine group. The duration of analgesia in dexmedetomidine group was 456±97 min, while in clonidine group, it was 289±62 min. Statistically, this difference was significant (P-value <0.001). [30]

T. Archna et al. also concluded that the addition of dexmedetomidine to bupivacaine prolongs the durations of sensory and motor block and duration of analgesia and improves the quality of anesthesia as compared with clonidine when injected with bupivacaine in supraclavicular brachial plexus block. [31]

CONCLUSION

We concluded that both clonidine and dexmedetomidine can be used as an adjuvant to 0.5% Levobupivacaine in supraclavicular brachial plexus block as both the drugs shortened the onset of sensory and motor blockade and prolonged the duration of sensory and motor blockade and duration of analgesia without any significant side effects but dexmedetomidine is a better alternate to clonidine as an adjuvant to local anaesthetic agent in supraclavicular brachial plexus block.

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