ORIGINAL ARTICLE

A Randomized Controlled Trial Study on the Effect of Adding Dexmedetomidine to Bupivacaine in Supraclavicular Block Using Ultrasound Guidance

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ABSTRACT

BACKGROUND: The benefits of regional anesthetic techniques are well established. Use of additives to local anesthetics can prolong these benefits. The aim of this study was to find out the effect of adding dexmedetomidine to bupivacaine for supraclavicular block.
METHODS: In this randomized, double-blind study, 70 ASA I & II patients of either sex undergoing elective surgeries on the upper limb were given supraclavicular block under ultrasound guidance. Group C (n=35) received 38 mL 0.25% bupivacaine + 2mL normal saline and group D received 38 mL 0.25% bupivacaine + 1 μg/kg dexmedetomidine (2mL). Patients were observed for, onset of motor and sensory block, duration of motor and sensory block, duration of analgesia, sedation score, hemodynamic changes and any adverse events.
RESULTS: In group D, the onset was faster (P< 0.001), durations of sensory and motor block duration of and analgesia were prolonged as compared to group C (P < 0.0001).There was a significant drop in heart rate (HR) from the baseline in group D (P < 0.05) at 30, 60, 90 and 120 min. However, none of the patients dropped HR below 50/min. Mean Arterial Pressure (MAP) remained unaffected. The patients in group D were more effectively sedated than those in group C (P < 0.05). No adverse event was reported in either group.
CONCLUSION: Dexmedetomidine as adjuvant to bupivacaine in supraclavicular block resulted in faster action, prolonged motor and sensory block, prolonged analgesia with hemodynamic stability and adequate sedation.
KEYWORDS: Dexmedetomidine, analgesia, supraclavicular block

INTRODUCTION

Surgeries of the upper limb are usually done under brachial plexus block. Various agents have been tried as adjuvant to increase the duration of post operative analgesia provided by local anesthetics (LA).We tried dexmedetomidine, an α2 adrenergic agent, as an adjuvant to bupivacaine as it has sedative, analgesic, sympatholytic and cardiovascular stabilizing effects. The aim of this study was to compare the postoperative analgesic efficacy and the safety of dexmedetomidine for brachial plexus blockade along with bupivacaine.

METHODS

After approval from Hospital Ethical Committee, a randomized, double-blind, prospective clinical study was initiated. Seventy ASA Grade I and II patients of either sex, aged 18–60 years, weighing 30-65 kg undergoing upper limb surgeries were selected. Patients with known hypersensitivity to LA, hypertension, diabetes mellitus, bleeding disorders, cardiac, liver or renal disease, pregnant women, infection at the site of injection and pre-existing peripheral neuropathy were excluded from the study. The patients were divided in two groups of 35 patients each on the basis of adjunct given to them. The randomization was achieved

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by random number table using a sealed envelope technique. The drug solutions were prepared by an anesthesiologist who was not involved in the study. The two groups were:
• Group C: control group; received injection bupivacaine (0.25%) 38mL plus 2mL normal saline, and
• Group D: study group; received injection bupivacaine (0.25%) 38mL plus dexmedetomidine 1 µg/kg diluted to 2mL.

Prior to surgery, all patients were fasted for six hours. Baseline heart rate, blood pressure and oxygen saturation were recorded. In the unaffected arm, after securing 18 G intravenous cannula, ringer's lactate was started. Premedication with anti-anxiety and sedative drugs was not given. All the patients received brachial plexus block through the supraclavicular approach by an experienced anesthesiologist under ultrasound (US) guidance (Sonosite Titan^{TM}TM US machine with a 6-13 MHz linear probe). After the brachial plexus was identified, the needle was introduced under US guidance. Correct position of the needle was confirmed by injecting saline boluses (0.5mL) before drug injection. After negative aspiration, 40mL of drug solution was injected.

Patients were assessed by a different anesthetist for hemodynamic changes, onset of motor and sensory blockade, duration of motor and sensory block, duration of analgesia, sedation score, and any adverse events. Both anesthetists were blinded to the treatment groups. Monitoring of vital parameters (HR and MAP) was done every five minutes for the first 30 minutes and thereafter every 10 minutes till the end of surgery and every 60 minutes(min) post-operatively. Bradycardia was defined as HR less than 50 beats/min which was treated with inj. atropine 0.6mg i.v.

Sensory block was assessed by the pin prick method at every three minute after drug injection by three point scale. According to this scale, (1) grade 0 was no block, grade 1 was sensory blockade with persistence of touch and grade 2 was complete sensory blockade. Onset of sensory block was the time from drug injection to grade 2 block. The duration of sensory block was the time from drug injection to the complete resolution of anesthesia.

Assessment of motor block was done using modified Bromage scale (2) by the same observer at every three minute until complete motor blockade. According to this scale, grade 0 was free movement of elbow, wrist and fingers, grade 1 was inability to extend the arm for 2 second, grade 2 was inability to extend the arm or flex the forearm, and grade 3 was inability to extend the arm, flex the forearm or move the fingers. Motor blockade of elbow was checked only in those cases in which it was possible; otherwise, only the movement of fingers was assessed.

The onset of motor block was the time from drug injection to grade 3 block. The duration of motor block was the time from drug injection to the complete recovery of motor function of the hand and forearm. The block was considered incomplete when any of the segments supplied by median, radial, ulnar and musculocutaneous nerve did not have complete sensory or motor block even after 30 minutes of drug injection. These patients were then excluded from the study and were given general anesthesia. For assessment of sedation of patients, Ramsay Sedation Score (3) was used.
Grade 1: Patient anxious and agitated or restless, or both
Grade 2: Patient cooperative, oriented and tranquil
Grade 3: Patient responds to commands only
Grade 4: Patient exhibits brisk response to light glabellar tap or loud auditory stimulus
Grade 5: Patient exhibits a sluggish response to light glabellar tap or loud auditory stimulus
Grade 6: Patient exhibits no response.

The assessment of postoperative pain was done hourly in the recovery room and in surgical ward with the help of Numeric Rating Scale (1-10) (4). Zero was considered as no pain, 1-3 as mild pain, 4-6 as moderate pain and 7-10 as severe pain. At score of 4, rescue analgesic (inj. diclofenac sodium (1.5 mg/kg) intramuscularly) was given. Duration of analgesia was the time from drug injection to the time of first request of analgesia. An attending nurse was advised to repeat Inj. diclofenac 1.5 mg/kg every eight hours thereafter. All patients were observed for any side-effects like nausea, vomiting, dryness of mouth, pneumothorax, hematoma, local anesthetic toxicity and post-block neuropathy in the intra- and postoperative periods.

Keeping the time of first analgesic request as primary variable, sample size was calculated. A pilot study with five patients in each group was conducted. Presuming the difference in

the time of first analgesic request and effect size obtained to be true, we calculated that 35 patients would be required in each group for the study with power 0.8 and significance of 0.05.The data was coded, entered and analysed using SPSS (standard statistical software SPSS) software Inc., version 16.0 for windows. Data was summarized using mean, standard deviation, minimum and maximum for quantitative variables and relative frequencies (percentages) for categorical variables. Normally distributed variables between the two groups were analysed using t-tests. Non-normally distributed variables were analysed using Mann Whitney test. Categorical variables were analysed using the Pearson's Chi-square ($\chi2$) test. Fisher exact test was used instead when the expected frequency is less than 5. $P < 0.05$ was considered as statistically significant and $P < 0.001$ as highly significant.

RESULTS

The patients in both groups were comparable with respect to the demographic parameters. The baseline hemodynamic parameters were comparable in both groups. In group D, HR was significantly decreased from the baseline at 30, 60, 90 and 120 minutes as compared to group C (Figure 1) ($P <0.001$) but none of the patients developed bradycardia. No statistical difference was observed on comparison of mean arterial pressures (MAP) between the two groups (Figure 2) ($P > 0.05$).
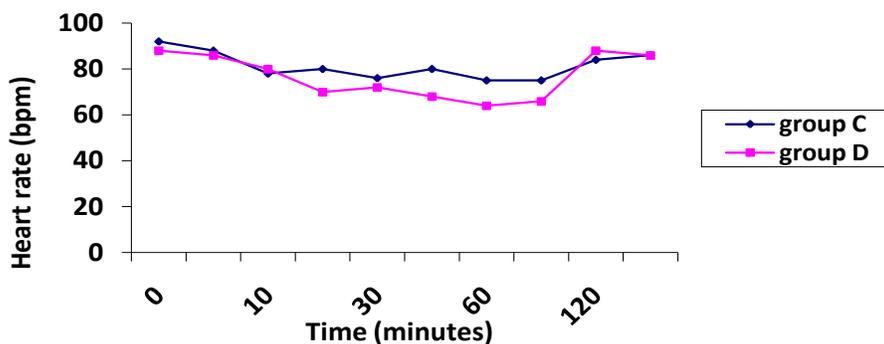

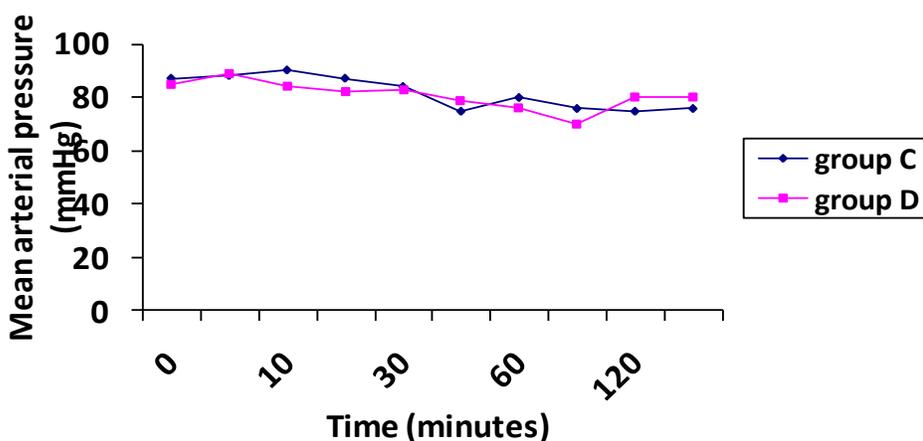*Figure 1: Comparison of heart rates between groups*

Figure 2: Comparison of Mean arterial pressures between groups

The onset of sensory and motor block was faster in group D as compared to group C (P <0.001) (Table 1). The duration of sensory and motor blockade was significantly prolonged in group D (P <0.0001) (Table 1). Similarly, the duration of analgesia was also significantly prolonged in group D as compared to group C (P <0.0001)

(Table 1). In group D, none of the patients was anxious. RSS 2 was seen in 83% of patients of group D while in group C, 85% of the patients had sedation score of 1 (Table 3) No adverse effects of drugs or complications of the technique were observed in any group.

Table 1: Demographic characteristics

Demographic parameters	Group C (n=35) Mean± S.D	Group D (n=35) Mean± S.D
Age (years)	38± 10.9	37.6± 9.3
Weight (kilograms)	50± 8.9	52± 8.7
Gender (Male/ Female)	17/18	20/15

Table 2: Onset, duration of sensory & motor block & duration of analgesia

Parameter	Group C (n= 35) Mean± S.D	Group D (n=35) Mean± S.D	t- value	P –value
Onset of sensory block (min)	14.76± 3.24	12.24± 2.8	3.48	0.001
Onset of motor block (min)	20.53± 2.43	15.8± 1.9	9.07	0.0001
Duration of sensory block (min)	200± 33.85	698± 43.9	53.14	0.0001
Duration of motor block (min)	188± 28.67	673± 79.46	33.96	0.0001
Duration of analgesia (min)	210± 35.88	722± 88.45	31.73	0.0001

Table 3: Comparison of Sedation scores

Sedation score (RSS)	Group C (n= 35)	Group D (n= 35)
1	30(85%)	0
2	5 (15)	29(83%)
3	-	6 (17%)
4	-	-
5	-	-
6	-	-

RSS= Ramsay sedation score

DISCUSSION

Dexmedetomidine, a highly selective alpha-2 agonist, has sedative, analgesic, sympatholytic and cardiovascular stabilizing effects (5). It causes significant opioid sparing as well as a decreased requirement of inhalational agents during anesthesia. (6) Various studies have shown that dexmedetomidine when added to LA in regional blocks (spinal, (7) epidural,(8) caudal,(9,10) axillary,(11) supraclavicular,(12) Bier's(13) and greater palatine nerve block(14)) causes

prolongation of duration of block and postoperative analgesia.
The highly selective action of dexmedetomidine on alpha-2 adrenoreceptors (α2:α1= 1620:1) results in sedation and analgesia without unwanted vascular effects from activation of alpha1-receptors. In addition, a reversal drug (Atipamezole) for the sedative effect of dexmedetomidine is also available. These properties make dexmedetomidine a suitable agent for sedation and analgesia during the perioperative period. (15) Dose of 1 μg/kg of dexmedetomidine was used for supra clavicular block in our study.

Similar dose were also used in previous studies. (11, 12) The use of ultrasound gave us the advantage of real time monitoring of drug injection around the plexus. However, the volume of bupivacaine used was not decreased because volume rather than the concentration of LA was important to achieve an effective nerve blockade. (16)

Significant drop in HR from the baseline was observed in patients of group D. However, none of the patients developed bradycardia or hypotension. The reduction in HR occurred due to the sympatholytic effect of alpha-2 agonists (11). Various studies which used dexmedetomidine in dose similar to our study, few of them had reported cases of bradycardia requiring treatment, (11,17,18) while others had not reported any incidence of bradycardia (12).

The onset of sensory and motor blockade was faster in the group receiving dexmedetomidine than in the control group. The faster onset could be due to local action of dexmedetomidine on nerve compound action potential (19) as well as enhancement of anesthetic action of LA (20). The results were in agreement with previous studies in which dexmedetomidine was added to bupivacaine in supraclavicular block (11,12,18,21).

The duration of motor and sensory block as well as post operative analgesia was prolonged in group D. The result can be explained by peripheral and central actions of dexmedetomidine. Peripherally, it produces analgesia by decreasing the release of norepinephrine which causes inhibition of ~~on~~ nerve action potentials. Centrally, it causes inhibition of the release of substance P in the nociceptive pathway at the level of the dorsal root neuron which produces analgesia (11).

The patients in group D were comfortable and did not require any sedative. This was due to the sedative effect of dexmedetomidine by virtue of its action on the subtype A and C of alpha-2 adrenoreceptors in the CNS (locus coerulus) which results in sedation, analgesia and anxiolysis. (15)

In conclusion, dexmedetomidine is an effective adjuvant to bupivacaine for supraclavicular block. It results in faster onset of block with prolonged duration of analgesia, effective sedation and stable hemodynamics

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