Effect of Dexmedetomidine as an Adjuvant to Bupivacaine in Supraclavicular Brachial Plexus Block

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ABSTRACT

Introduction: The objective of this study was to evaluate the effect of 50µg dexmedetomidine on the onset and duration of block and duration of analgesia when used as an adjuvant to bupivacaine in ultrasound guided supraclavicular brachial plexus block.

Methods: Eighty patients of ASA physical status I and II undergoing elective upper limb surgery under ultrasound guided supraclavicular brachial plexus block were randomly divided into two groups: Group D and Group B. Group D (n=40) received 19.5 ml of 0.5% bupivacaine with 0.5 ml (50 µg) dexmedetomidine. Group B (n=40) received 19.5 ml of 0.5% bupivacaine with 0.5 ml normal saline. Onset time of sensory and motor block, duration of sensory and motor block and duration of analgesia was recorded.

Results: Onset time of sensory block (10.55±4.84 min in Group D vs 12.50 ±5.20 min in Group B) and motor block (15.85±5.9min in Group D vs 18.35±5.6min in Group B) though earlier in Group D as compared to Group B was not statistically significant (p value =.087 for sensory block and p value=.058 for motor block). The duration of sensory block (772.20 ±167.84 min in Group D vs 398.38 ±129.839min in Group B) and motor block (725.63±140.964min in Group D vs 361.88±128.764 min in Group B) was statistically significantly prolonged in Group D (p value= .000 for sensory and p value =.000 for motor block). The Duration of analgesia (845.93±184.545min in Group D vs 430.04±121.307 min in Group B) was also statistically significantly prolonged in group D (p value= .000).

Conclusions: Dexmedetomidine (50 µg) as an adjuvant to 0.5% bupivacaine solution in ultrasound guided supraclavicular brachial plexus block prolongs the duration (sensory and motor) of block as well as the duration of analgesia with no effect on the onset time of block.

Keywords: *brachial plexus block; bupivacaine; dexmedetomidine.*

INTRODUCTION

Brachial plexus block (BPB) provides several advantages over general anesthesia like excellent pain control, better hemodynamic stability, less postoperative nausea vomiting, early recovery thereby shortened stay in the post anesthesia care unit and no or minimal systemic side effects of anesthesia drugs.[1,2] With the use of ultrasound and peripheral nerve stimulator, efficacy and safety of the BPB has been greatly improved. To further improve the quality of block and prolong the duration of analgesia, various adjuvants to local anesthetic drugs have been tried, such as opioids,[3] epinephrine,[4] midazolam,[5] magnesium,[6] dexamethasone,[7]. Lately Alpha 2 receptor agonists clonidine and dexmedetomidine are the drugs of new interest in regional anesthesia. Dexmedetomidine is highly selective (8 times more selective than clonidine),[8] specific and potent alpha 2 adrenergic agonist having analgesic, sedative, antihypertensive, and anesthetic sparing effects when used via systemic route.[9] It has also been reported to improve the quality of

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intrathecal and epidural anesthesia.[10,11,12,13] There are clinical studies that have reported that perineuraldexmedetomidine facilitates a better effect on Brachial plexus block as well.[14,15]

To test the effect of dexmedetomidine as an adjuvant to bupivacaine in supraclavicular brachial plexus block in our population this study was designed.

METHODS

This was a randomized controlled trial done in NMCTH from January 2018 to June 2018. After obtaining permission from institutional ethical committee, patients were explained about the drug and the procedure and only those who gave willful written consent were included in the study. Eighty ASA physical status I and II patients, 20-60 years, weighing more than 50 kg undergoing upper limb surgery under supraclavicular brachial plexus block were enrolled in this study.

Exclusion criteria were patient refusal, any known hypersensitivity or contraindication to bupivacaine and dexmedetomidine; pregnancy, lactating mothers, hepatic, renal or cardiopulmonary abnormality, alcoholism, diabetes, long term analgesic therapy, local skin site infections were excluded from the study. Patients with a history of significant neurological and psychiatric disorders were also excluded.

Patients were randomly allocated using a sealed envelope technique into two groups: Group D and Group B. Group D (n=40) received 19.5 ml of 0.5% bupivacaine with 0.5ml (50µg) dexmedetomidine. Group B (n=40) received 19.5ml of 0.5% bupivacaine with 0.5ml normal saline. The drug solutions were prepared by a consultant anesthesiologist who was not further involved in the study.All the blocks were performed by the principal investigator of the study (who was experienced in providing USG guided nerve blocks). Data collection during intraoperative period and follow up of the patients in the postoperative ward was done by a third year anesthesiology resident. The anesthesiologists performing the block as well as the resident involved in data collection was unaware of the group allocation.

All the patients were examined a day before surgery, when whole procedure was explained. 10cm visual analog scale (VAS) (0, no pain and 10 worst pain imaginable) was also explained to the patient. All patients were kept fasting as per standard guidelines.

On the day of surgery, standard monitors were connected. Basal heart rate (HR); noninvasive arterial systolic blood pressure (SBP) and diastolic blood pressure (DBP); and peripheral oxygen saturation (SpO2) were recorded. An 18 gauge(G) i.v. cannula was inserted in nonoperated arm and lactated Ringer's solution was started at 5ml/kg/h. Patient was positioned supine with head turned away from the limb to be operated. The skin surface over the supraclavicular fossa was cleansed with povidone iodine. A 6-13 mHz linear ultrasound probe was used to identify the brachial plexus in relation to the pulsating subclavian artery and the hyperechoic first rib. Local anesthesia at the injection site was provided with 3 ml 2% plain lignocaine. The plexus was then approached using an in-plane (IP) technique with a 5cm 22G regional block needle. Once the needle tip reached the nerve sheath and following negative aspiration for blood/air, 20 ml of the study drug was injected around the plexus under vision. Plexus block was considered successful when all the four nerve injection territories (ulnar, radial, median, and musculocutaneous) were effectively blocked for both sensory and motor block.

Sensory block (four nerve territories) was assessed by pin prick test using a 3-point scale:

0= normal sensation

1=loss of sensation of pin prick (analgesia),

2=loss of sensation of touch (anesthesia).

Motor block was determined by thumb abduction (radial nerve), thumb adduction (ulnar nerve), thumb opposition (median nerve), and flexion of elbow (musculocutaneous nerve) according to the modified Bromage scale on a 3-point scale:

Grade 0: normal motor function with full flexion and extension of elbow, wrist, and fingers.

Grade 1: decreased motor strength with ability to move the fingers only

Grade 2: complete motor block with inability to move the fingers.

Both sensory and motor blocks were assessed every 3 min till their onset and at 15, 30, 45, 60, 90, and 120min; and then hourly till the effect of block had completely resolved. Patients were asked to note the subjective recovery of sensation and movements which was then certified by anesthesiologist on duty.

Onset time for sensory block was defined as the time interval between the end of local anesthetic administration and complete sensory block (score 2 for all nerves). Duration of sensory block was defined as the time interval between the complete sensory block and complete resolution of anesthesia on all the nerves (score 0)

Onset time for motor block was defined as the time interval between the end of the local anesthetic administration and complete motor block (grade 2). Duration of motor block was defined as the time interval from complete motor block to complete recovery of motor function of hand and forearm (grade 0).

An unsuccessful block was defined as absence of grade I sensory or motor block till 30 min after injection of drug. In such a circumstance of inadequate or patchy block, it was to be supplemented with general anesthesia and the patients excluded from the study.

HR, SBP, and DBP were also recorded every 5 minutes till completion of surgery.

Hypotension was defined as 20% decrease in mean arterial pressure relative to baseline, and was treated by fluid bolus or injectionmephentermine 6 mg. Bradycardia was defined as HR < 50 bpm and was managed with injection atropine 0.6 mg. Any other adverse effects like nausea, vomiting or hypoxemia (SpO2<90%) were noted. Blood loss was noted and replaced if more than the allowable blood loss. Pain was assessed using visual analogue scale (VAS) 0-10. Nursing staff was directed to administer inj. Diclofenac sodium 2mg/kg intramuscular when VAS≥ 3 (rescue analgesia). The time between the complete sensory block and the first analgesic request was recorded as duration of analgesia (DOA).

Using the following formulasample size was calculated

Number of cases in each group (n) =

$$\frac{2(z_{\alpha}+z_{\beta})^2 S^2}{d^2}$$

Based on a study done by Zhenqinget al[16] in which the mean standard deviation of duration of analgesia was 38.6min and the difference in mean was 58.1, minimum sample size required was 20. Because of the chances of failure of block, we conducted the study in total 80 patients.

The data was compiled and subjected to statistical analysis using Statistical Package for Social Sciences (SPSS), version 16. Demographic data was subjected to Student's t-test and for statistical analysis of onset time and duration of sensory and motor blocks, and duration of analgesia, unpaired t-test was applied.

RESULTS

Total 80 patients were recruited in the study. There was no block failure in any of the

patients. Demographic data (age, weight and gender) and ASA PS of patients were comparable in both the groups. (table 1) The mean duration of surgery was also similar in both the groups. (Table 2) The onset of both sensory and motor block was earlier in Group D than Group B (Table 3), but they were not clinically significant. Duration of sensory and motor block were significantly longer in Group D than in Group B. Duration of analgesia was also significantly longer in dexmedetomidine group.

Table 1. Patient characteristics

Parameter	Group B	Group D	P value
Age(years (Mean± SD)	40.15±11.432	35.55±12.549	.091
Body weight(kg) (Mean± SD)	61.33±7.357	63.50±10.427	.284
Gender (male/female)	31:9	30:10	1
ASA physical status (I/II)	30/10	33/7	.586

Table 2. Duration of surgery

Parameter	Group B	Group D	P value
Duration of surgery (min)	77.95±18.657	75.65±16.255	.558

Table 3. Onset of sensory and motor blockade

Onset of block	Group B	Group D	P value
Sensory onset (min)(mean ±SD)	12.50 ±5.20	10.55±4.84	.087
Motor onset (min)(mean ±SD)	18.35±5.6	15.85±5.9	.058

Table 4. Duration of blockade and analgesia

Duration of block and analgesia	Group B	Group D	P value
Sensory blockade (min)(mean± SD)	398.38 ±129.839	772.20 ±167.84	.000
Motor blockade (min) (mean ±SD)	361.88±128.764	725.63±140.964	.000
Duration of analgesia (min)(mean± SD)	430.04±121.307	845.93±184.545	.000

DISCUSSION

In this study we determined the effect of dexmedetomidine 50µg as an adjuvant to bupivacaine in ultrasound guided supraclavicular brachial plexus block in terms of onset, duration of motor and sensory block and also total duration of analgesia.

The hypothesized mechanism of action of dexmedetomidine in peripheral nerve block is: it inhibits the function of sodium channels and neuronal potassium current and blocks the hyperpolarization- activated cyclic nucleotide-gated channels, resulting in the enhancement of activity- dependent hyperpolarization and leading to the inhibition of substance P release in the nociceptive pathway at the dorsal root neuron. The other possible mechanism by which the α2 agonists improve local anesthetic action include vasoconstriction around the site of injection, thus delaying the absorption of local anesthetic drug, resulting in a prolongation of the local anesthetic effect. Other mechanism include release of local inflammatory mediators and increase in the release of anti - inflammatory cytokines.

The exact volume of local anesthetic agent for effective block remains controversial.Brenner et al[17] in their study named estimation of the minimum effective volume of 0.5% bupivacaine for ultrasound-guided axillary brachial plexus block have reported that under ultrasound guide use of 1ml per nerve bupivacaine 0.5% results in acceptable block onset times, adequate surgical anaesthesia and excellent postoperative analgesia in patients undergoing surgical procedures with extended tissue damage. Kothari in his lateral approach technique of supraclavicular brachial plexus block has found that only 20ml of local anesthetic mixture can produce block lasting 180-200min without any adjuvants.[18] Arun Kumar et al19 have studied the effect of dexamethasone in low volume supraclavicular brachial plexus block in which brachial plexus block was done under ultrasound guidance with only 20ml of 0.5% plain bupivacaine. In our institute prior to the

availability of Ultrasound, BPB was done by elicitation of paresthesia in which 30 to 40 ml of local anesthetic mixtures was given. With the above mentioned studies as a reference we decided to perform the brachial plexus block with 20 ml of 0.5% plain bupivacaine.

Dexmedetomidine has been used as an adjuvant to bupivacaine in brachial plexus block in different doses. Zhang Y et al 20in their study compared dexmedetomidine in a dose of 50 mcg and 100mcg as an adjuvant to ropivacaine. They found the incidence of side effects like bradycardia, hypertension and hypotension to be significantly higher in the patients who received 100mcg dexmedetomidine. Similarly JithendraChinnappa et al[21] have also reported statistically significant incidence of hypotension and bradycardia with the use of 1 µg/kg of dexmedetomidine in brachial plexus block. In order to avoid these hemodynamic complications we chose to use a lower dose of dexmedetomidine.

In our study duration of sensory block (772.20 ±167.84 min in Group D vs398.38 ±129.839min in group B) and motor block (725.63±140.964min in Group D vs361.88±128.764 min in Group B) was significantly prolonged in the dexmedetomidine group as compared to the Bupivacaine only group. Duration of analgesia (845.93±184.545min in group D vs430.04±121.307 min in group B) was also significantly longer in dexmedetomidine group in our study. The results of our study are similar to the study done bySandhyaet al22. They studied the effect of adding dexmedetomidine (100 µg) to 0.325% bupivacaine and found that dexmedetomidine as an adjuvant to bupivacaine significantly prolongs the duration of sensory and motor blocks and duration of analgesia. Dixitkumar B et al [23] added 1 µg/kg of dexmedetomidine to a mixture of 10ml 2%lignocaine and 20ml of 0.5% bupivacaine and compared it with a mixture of 10ml 2%lignocaine and 20ml of 0.5% bupivacaine. They found a significant prolonged duration of sensory and motor block.Rashmi et al [24] studied the effect of adding 50 µgdexmedetomidine to ropivaciane in brachial plexus block and their results were similar to our study. They found that dexmedetomidine prolongs the duration of sensory and motor blockade.

In our study the onset time of sensory block (10.55±4.84 min in Group D vs12.50 ±5.20 min in Group B) and motor block (15.85±5.9min in Group D vs 18.35±5.6min in Group B) were comparable in both the groups. Though the onset time was earlier with the use of dexmedetomidine as an adjuvant,it was not statistically significant. This finding correlates with the study done by Rachana et al [25] who studied the effect of a low dose dexmedetomidine (30 µg) as an adjuvant. They found a prolonged duration of block (sensory and motor) and a longer duration of analgesia with dexmedetomidine but it failed to hasten the onset time of block. Similarly Mathew et al [26] when used dexmedetomidine in dose of 1 µg/kg as an adjuvant found that the onset time of motor and sensory block was similar to that of the group without dexmedetomidine. The lower dose of dexmedetomidine may be the reason of failure to hasten the onset of block in our study.

However Sandhyaet al[22] noticed that the onset time of sensory and motor blockade was shorter in the group receiving dexmedetomidine as an adjuvant to bupivacaine. In their study they used dexmedetomidine in a dose of 100 µg. A higher dose irrespective of the body weight may be the reason for quicker onset in their study. Similarly Esmaoglu et al[27] also found a quicker onset of sensory and motor blockade in the group receiving dexmedetomidine as an adjuvant in a dose of 100 µg irrespective of the body weight.

There were no episodes of bradycardia, hypertension or hypotension. Though these episodes have been reported with the use of dexmedetomidine, no such event occurred during our study as we used a lower dose of dexmedetomidine.

CONCLUSIONS

Based on this study, we can conclude that dexmedetomidine (50 µg) as an adjuvant to 0.5% bupivacaine solution in ultrasound guided supraclavicular brachial plexus block prolongs the duration (sensory and motor) of block as well as the duration of analgesia with no effect on onset of block.

CONFLICT OF INTEREST: None.

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