Alpha 2 agonist dexmedetomidine as an adjuvant to bupivacaine in supraclavicular brachial plexus block

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Abstract

Background: Regional anesthesia is a recommended technique for upper limb surgeries with better postoperative profile. Alpha-2 agonists are added to local anesthetic agents to extend the duration of nerve blocks. We evaluated the effect of combining dexmedetomidine with bupivacaine with respect to duration of motor and sensory block and duration of analgesia. **Materials and Methods:** Sixty patients posted for upper limb surgeries were enrolled for a prospective, randomized, study. Patients were divided into two groups, the control group B and the study group BD. In group B (n = 30), 30 ml of 0.325% bupivacaine + normal saline; and in group BD (n = 30), 30 ml of 0.325% bupivacaine +1µg/kg dexmedetomidine given ultrasound guided supraclavicular brachial plexus block. **Results:** The onset times for sensory and motor blocks were significantly shorter in BD than B group ($p < 0.05$), while the duration of sensory and motor blocks was significantly longer in BD group. The duration of analgesia was significantly longer in BD group than B group ($p < 0.0001$). SBP and DBP levels in BD group were significantly lower than in B group 20 min after block ($p < 0.001$). Bradycardia was observed in two patients in the group BD. **Conclusion:** Dexmedetomidine added as an adjuvant to bupivacaine for supraclavicular brachial plexus block significantly shortens the onset time and prolongs the duration of sensory and motor blocks and duration of analgesia. Patients in group BD were adequately sedated with no adverse effects except bradycardia in two patients.

Keywords: Adjuvant, Dexmetomidine, Supraclavicular brachial plexus block.

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Introduction

Upper limb surgeries are preferably done under regional anesthesia. Peripheral nerve blocks not only provide for intra operative anesthesia but also ensure analgesia in the post operative period without any systemic side effects.

Adjuvant drugs are often added to local anesthetics for several reasons. Like clonidine, the α-2 receptor agonist dexmedetomidine (DEX) has been reported to have a rapid onset time, to prolong the duration of local anesthetics, and to increase the quality of analgesia in a regional block [1,2]. Dexmedetomidine is being used for intravenous (iv) sedation and analgesia for intubated and mechanically ventilated patients in intensive care units (ICUs) [3,4], intravenous regional anesthesia (Bier's block) [5]. Its use in peripheral nerve blocks has recently been described. However, the reports of its use in supraclavicular brachial plexus block are limited. In this prospective and double blinded study we analyze the effect of DEX on the sensory and motor blocks and duration of analgesia and the effect of DEX on sedation [6]. The Ramsay sedation scale was used for evaluation of the sedation state.

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Methodology

After obtaining approval from the institutional ethical committee, patients were explained about the drug and after taking written consent were included in the study. Sixty patients of ASA physical status I and II, 18-60 years scheduled for elective forearm surgery under

supraclavicular brachial plexus block were included in a prospective double blind randomized comparison. Patients with diabetes, peripheral neuropathy, with known allergy to LAs, coagulopathy, infection at the site of block, pregnancy, and patients on beta blockers were excluded from the study.

Control group B (n = 30) received 30 ml of 0.325% bupivacaine with normal saline solution. Study group BD (n = 30) received 30 ml of 0.325% bupivacaine and 1μg/kg of dexmedetomidine. The anesthesiologist performing the block and observing the patient was blinded to the treatment group. Standard anesthesia monitoring in the form of the baseline measurement of heart rate, noninvasive arterial blood pressure, and peripheral oxygen saturation (SpO_2) was started. Intravenous access was achieved using 20 G cannula in the nonoperative arm.

Sensory block was assessed by a traumatic pin prick test using a 3-point scale: 0 - normal sensation, 1 - loss of sensation of pin prick (analgesia), and 2 -loss of sensation of touch (anesthesia). Motor block was assessed by using modified Bromage [18] 3-point scale: Grade 0: Normal motor function; Grade 1: Decreased motor strength with ability to move the fingers only; Grade 2: Complete motor block.

Both sensory and motor blocks were assessed every 5 min for first 30 min and thereafter every 15 min for 2 hrs and then every 30 min and then hourly till 24 hours. Sensory block onset and duration as well as motor block onset and duration along with duration of analgesia, level of sedation were measured. Sedation score was assessed by Ramsay sedation scale 4. This has scoring from 1 to 4. Score1 –fully awake and oriented and follows verbal command; Score2 – drowsy, eyes closed but arousable only to commands; Score3- eyes closed but arousable to mild physical stimulation; Score 4- eyes closed and unarousable to mild physical stimulation.

Any need for rescue analgesia was noted intraoperatively. Pain was assessed using visual analogue scale (VAS) 0-10. Inj. diclofenac sodium 3 mg/kg intramuscular was administered when VAS ≥ 3 (rescue analgesia). The time between the complete sensory block and the first analgesic request was recorded as duration of analgesia (DOA). Total amount of diclofenac sodium used in first 24 h period postoperatively was noted

Results

Table 1: Comparison of time of onset of complete sensory and motor block.

Onset time	Group B	Group BD	p-value
Sensory block (min)	8.42± 2.41	6.24± 1.22	< 0.05
Motor block (min)	14.6± 3.6	10.2 ±1.69	< 0.05

Table 2: Comparison of time of duration of block, analgesia and level of sedation.

	Group B	Group BD	p-value
Sensory Block (Min)	202±30.4	456.42±20.22	<0.001
Motor Block (Min)	172.4±41.26	418.6±32.46	<0.001
Analgesia (Min)	346±40.31	668.56±40.7	<0.001
Sedation Score (1-4)	1	2.8	

The demographic data and surgical characteristics were comparable in both groups. Onset time was shorter while duration of sensory and motor blockade was longer in BD than B group and the difference was statistically significant (p < 0.05). Table 1 depicts the mean onset time for sensory and motor blocks in group BD were 6.24± 1.22 and 10.2 ±1.69 min respectively, and for group B were 8.42± 2.41 and 14.6± 3.6 min, respectively. The mean duration time for sensory and motor blocks for group BD were 456.42±20.22 and 418.6±32.46 min respectively; but for the group B, the mean duration were 202±30.4 and 172.4±41.26 min respectively. The mean duration of analgesia (DOA) for group BD was 488.56±41.7min, it was 246±40.31 min for group B (Table 2). DOA was significantly longer in group BD than group B (p < 0.001). HR, SBP, and DBP in group BD at 15, 30, 45 min were significantly lower than in group B (p < 0.001) (Table 3). In fact, when the percentage changes in the HR, SBP, and DBP were compared from 10 to 150 min, they were highly significant (p < 0.001).

Table 3: Comparison of Mean Systolic Bp between both groups.

	Group B	Group BD	p-value
0 min	134.46 ± 2.42	132.3±6.29	>0.05
5 min	130.3±5.62	126±4.65	>0.05
10 min	127.26 ± 6.64	111.23±3.46	<0.001
15 min	122.76 ± 6.58	102.46 ± 6.19	<0.001
20 min	120.4 ± 2.41	102.7 ± 6.61	<0.001
25 min	118.3 ± 4.32	106.9± 6.17	<0.001
30 min	116.43 ± 4.65	102.16 ±2.41	<0.001
45 min	123.56 ± 3.12	102.86 ±38.30	<0.001
60 min	126.4 ± 2.41	102.72 ± 7.70	<0.001
90 min	118.3 ± 6.36	104.9± 6.27	<0.001
120 min	122.43 ± 4.65	106.16 ±2.42	<0.001
150 min	120.56 ± 12.02	106.86 ±14.32	<0.001

Table 4: Intra and Postoperative Complications.

S. No.	Complication	Group B	Group BD
1	Hypotension	Nil	2
2	Bradycardia	Nil	2
3	Nausea and vomiting	Nil	Nil
4	Sedation	Nil	8
5	Respiratory depression	Nil	Nil

Statistical Analysis: After all parameters, patient's age and duration of surgery were analyzed by student's unpaired '*t*'-test. Sex distribution and ASA grading were analyzed by chi-square test. Time for onset of adequate sensory block, duration of sensory and motor block was analyzed by student's unpaired '*t*' test. Comparison of intraoperative complications like bradycardia and hypotension were analyzed by Fisher exact test. The data was compiled and subjected to statistical analysis using Statistical Package for Social Sciences (SPSS), version 17. Demographic and hemodynamic data were subjected to Students 't-test' and for statistical analysis of onset time and duration of sensory and motor blocks, and DOA unpaired *t*-test. p-value was considered as significant as shown below .$p > 0.05$ not significant, $p < 0.05$ significant, $p < 0.0001$ highly significant.

Discussion

Since the advent of regional anaesthesia, the anaesthesiologists were in constant search to improve the quality and duration of brachial plexus blocks. From the age old practice of adding adrenaline, adding sensorcaine, soda bicarbonate; to now using various other adjuvants like clonidine, Fentanyl etc. by using ultrasound guided needles; the brachial plexus blocks have come a long way [1] [2]. Adding dexmedetomidine as an adjuvant to bupivacaine has proved to be beneficial as it can significantly: shortens the sensory and motor block onset time; reduce the offset time for motor block; prolong the duration of postoperative analgesia; provides significantly lower postoperative VAS pain scores, and provided comparable overall satisfaction scores among patients. The main purpose is to achieve a good analgesia with good duration of action and minimal side effects. Dexmetomidine an α-2 adrenoreceptors agonist seems to cater to all the above requirements [3] Shehabi et al elaborated the selectively, specificity & potency of dexmetodine. They actually studied the sedative cardiovascular effects in patients receiving dexmetodine infusion for more than 24 hrs [4] Abo Sediza et al compared clonidine / dexmetodine in Biers block [5] Various researchers till now have added adjuvant to local anaesthetic agents for giving brachial plexus blocks. Dexmetomidine from the very outset has been used in ICU for continuous infusion, side effects being hypotension, sedation and bradycardia Mantz J, Singer M et al studied the orientation and arousablity of patients in ICU who are being given Dexmetomidine similar works have been by M. Shurtey, JA Miller who

studied use of Dexmetomidine in non intubated patients [6] [7]. α2-AR agonist such as Dexmetomidine have demonstrated a dose-dependent increase in the duration of thermal antinociception and analgesia in many animal studies. Dexmetomidine in clinically effective doses lacks respiratory depression, but maintains its analgesic properties that may make it useful and safe adjunct in many diverse clinical applications.

Esmaoglu *et al*., reported prolongation of axillary brachial plexus block when Dexmetomidine was added to levobupivacaine. In our study, while the onset time of both sensory and motor blocks were shortened in the drug group, the duration of analgesia was significantly prolonged. In our study, two patients developed bradycardia, which did not require any treatment [8]. Others added Dexmetomidine to bupivacaine for greater palatine nerve block after deft palate repair [9]. M.P. Rancort PM Berraurd et al added Dexmetomidine to ropivacaine in lower limb surgeries by blocking posterior tibial nerve [10]. Centrally, α2 agonists produce analgesia and sedation by inhibiting substance P release in the nociceptive pathway at the level of the dorsal root neuron and by activating α2 adrenoceptors in the locus coeruleus. Peripheral action of dexmedetomidine was caused by activation of hyperpolarization activated caution current which prevents the nerve from returning from hyperpolarized state to resting membrane potential for subsequent firing. Kousugi et al in their study found high concentrations of dexmedetomidine inhibit compound action potentials in frog sciatic nerves without α2 adrenorecptors activation in a concentration dependent manner and reversibly. The efficacy of perineural dexmedetomidine for analgesia has been established. This effect is dose dependent and the effect is peripheral (not caused by centrally mediated or systemic analgesia). Perineural administration of Dexmetomidine in combination with bupivacaine enhanced sensory & motor blockade in sciatic nerve without inducing neurotoxicity, was studied by CM Brummett, R lydic et al in rats [11].

Dexmetomidine was added to lidocaine and its effects studied by T. Yoshitomi et al. They demonstrated that α2-AR agonist enhanced the local anesthetic action LA's via peripheral α-2A adrenoceptors. Studies have shown that clonidine when added to bupivacaine prolongs the duration of anesthesia and analgesia in brachial plexus block, but was associated with bradycardia, hypotension, and respiratory depression as side effects [12]. D. Memis also added Dexmetomidine to lidocaine but they did it for biers block that is IVRA [13]. Other researchers did similar studies in branchial plexus block such as N. Merle, D Nader et al added Dexmetomidine with bupivacaine for interscalene block [14].

Ammar *et* al. used dexmetomidine with bupivacaine and compared it with plain bupivacaine and demonstrated enhancement of onset of sensory and motor blockade, prolonged duration of analgesia, increased duration of sensory and motor block, lower VAS pain scores, and reduction in supplemental opioid requirements. They studied ultrasound guided infraclavicular brachial plexus block using bupivacaine alone and then combined with Dexmetomidine for pain control in upper limp surgeries [15].

The above studies were similar to our study in which onset and duration of motor/sensory block was studied results were correlating with our studies. Similar prolongation of action was also seen by EL Hermawy et al when Dexmetomidine was added to bupivacaine for caudal analgesia in children [16].

When compared with clonidine, Dexmetomidine was found slightly better for supraclavicular block in a study done by SS Swami R Rao et al. They used Dexmetomidine and clonidine as an adjuvant to bupivacaine 0.25% in supraclavicular plexus block and demonstrated that Dexmetomidine prolongs the duration of sensory and motor block and enhances the quality of block as compared with clonidine [17]. Masuki *et al*., suggested that Dexmetomidine induces vasoconstriction via α2 adrenoceptors in the human forearm possibly also causing vasoconstriction around the site of injection, delaying the absorption of local anesthetic and hence prolonging its effect [18].

M. Shurkey et al and Yazbek- Karam et al both studied Dexmetomidine in detail and inferred that it has emerged as a very useful drug for sedation in non intubated patients. Yazbek on other hand enumerated the perioperative uses of Dexmetomidine [19, 20].

The limitation of our study was that we could not measure the levels of bupivacaine and dexmedtomidine in the blood due to non availability of the facility in our institute. Levels of the drugs in the blood would have supported our conclusions. In a nutshell; use of Dexmetomidine is now very diverse. It could be now termed as a wonder drug in the armaterium of anesthesiologist [21,22].

Conclusion

Dexmedetomidine added as an adjuvant to bupivacaine for supraclavicular brachial plexus block significantly shortens the onset time and prolongs the duration of sensory and motor blocks and duration of analgesia in patients undergoing upper limb surgeries compare to saline group. Patients in group BD were adequately sedated (modified Wilson Sedation Score, RSS = 2/6 or 3/6) with no adverse effects except bradycardia in two patient of group BD. Significant difference was seen in postoperative diclofenac and opioid requirement.

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