Original Research Article

Dexmedetomidine as an adjuvant to levobupivacaine in ultrasound guided supraclavicular brachial plexus block

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ABSTRACT

Background: *Alpha-2 agonists are added to local anesthetic agents to extend the duration of peripheral nerve blocks.*

Objective: *We evaluated the effect of combining dexmedetomidine with levobupivacine with respect to duration of motor and sensory block and duration of analgesia.*

Material and Methods*: Sixty patients of ASA grade I or II aged between 18-60 years, posted for elective upper limb surgeries were enrolled for a prospective, randomized, double-blind 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% levobupivacaine + normal saline; and in group BD (n = 30), 30 ml of 0.325%levobupivacaine + 1µg/kg dexmedetomidine were given for ultrasound guided supraclavicular brachial plexus block. Duration of motor and sensory block and time to first rescue analgesia were recorded.*

Results*: Demographic profile and surgical characteristics were similar in both groups. The onset times for sensory and motor blocks were significantly shorter in BD group (*$p < 0.05$*), while the duration of sensory and motor blocks and duration of analgesia (DOA) was significantly longer in BD group. Heart rate level and SBP and DBP levels in group BD were significantly lower 15-20 min after block (*$p< 0.05$*). Bradycardia was observed in two patients in the group BD. No other adverse effects were observed in either of the groups.*

Conclusion*: Dexmedetomidine added as an adjuvant to levobupivacaine for supraclavicular brachial plexus block significantly shortens the onset time and prolongs the duration of sensory and motor blocks and duration of analgesia.*

Keywords: *Adjuvant, dexmedetomidine, supraclavicular brachial plexus block, levobupivacaine*

Introduction

Upper limb surgeries are preferably done under regional anesthesia. Peripheral nerve blocks not only provide intra operative anesthesia but also ensure analgesia in the post operative period without any systemic side effects. This approach is attractive due to its effectiveness in terms of cost and performance and margin of safety.

Kulenkampff in Germany performed the first percutaneous supraclavicular block in 1911, reportedly on himself, which was later modified as Winnie block. [1] A few months later, Herschel described a method of brachial plexus with an axillary approach. In 1928, Kulenkampff and Persky published their experiences with a thousand blocks without apparent major complications. The supraclavicular approach to the brachial plexus characteristically is associated with a rapid onset of anesthesia and a high success rate.

Adjuvant drugs are often added to local anesthetics for several reasons. [1, 2] 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. [3,4,5] Dexmedetomidine is being used for intravenous regional anesthesia (Bier's block), [5,6] intravenous (iv) sedation and analgesia for intubated and mechanically ventilated patients in intensive care units (ICUs). [7,8,9] Its use as an adjuvant in central neuraxial blocks has also been mentioned. [10,11,12,13] Its use in peripheral nerve blocks has recently been described. [14,15,16] However, the reports of its use in supraclavicular brachial plexus block are limited. [17] In this study we analyzed the effect of DEX on sensory and motor blocks, duration of analgesia and sedation.

Material and Methods

In this prospective and double blind study the effect of DEX on the sensory and motor blocks and duration of analgesia and the effect of DEX on sedation was analyzed. The Ramsay sedation scale was used for evaluation of the sedation state. [18,19,20,21] After obtaining approval from the institutional ethical committee, patients were explained about the drug and written consent was obtained. Sixty patients of ASA physical status I and II, 18-60 years scheduled for elective forearm surgery under supraclavicular brachial plexus block were included. 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. All patients were assessed preoperatively and investigated. Patients were premedicated with tablet alprazolam 0.25 mg and tablet ranitidine 150 mg on night before surgery. Control group B (n = 30) received 30 ml of 0.325% levobupivacaine with normal saline solution. Study group BD (n = 30) received 30 ml of 0.325% levobupivacaine 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.

Under all aseptic coditions, ultrasound guided supraclavicular brachial plexus block was administered to the patient using 23 G spinal needle and Sonosite machine with high frequency (13 MHz) linear probe. Spread of the drug was confirmed by USG. Sensory block was assessed by atraumatic 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. We assessed various parameters at 5, 10,15,20,25 and 30 min, and thereafter every 15 min for 2 hrs and then 30 min till block effect has resolved. It also includes sensory block onset and duration as well as motor block onset and duration along with duration of analgesia, level of sedation. 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.

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.

Results

The demographic data and surgical characteristics were comparable in both groups (Table 1). 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 2 dispicts 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 ($p < 0.05$). The mean duration time for sensory and motor blocks for group BD were 546.42±20.22 and 518.6±32.46min respectively; but for the group B, the mean duration were 202±30.4 and 172.4±41.26 min. The mean duration of analgesia (DOA) for group BD was 578.56±41.7min, it was 246±40.31 min for group B (Figure 3). DOA was significantly longer in group BD than group B ($p < 0.001$). HR, SBP, and DBP in group BD at 5, 10, 15, 30, 45 min were significantly lower than in group B ($p < 0.05$) (Fig. 4). In fact, when the percentage changes in the HR, SBP, and DBP were compared from 0-10min ($p>0.05$) to 15-120 min, they were significant ($p < 0.05$).

Table 1: Demographic parameters in group B and group BD

	Group B	Group BD	p-value
Age (Years)	38.43 ± 13.46	37.89 ± 12.23	>0.05
Sex			
Male	22 (73.3 %)	23 (66.67%)	>0.05
Female	08 (26.7%)	07 (33.34%)	
ASA Grade			
I	18 (60 %)	17 (56.67 %)	>0.05
II	12 (40%)	13 (43.33%)	

Table 2: 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 3: Comparison of time of duration of block, analgesia and level of sedation

	Group B	Group BD	p-value
Sensory Block (Min)	202±30.4	546.42±20.22	<0.001
Motor Block (Min)	172.4±41.26	518.6±32.46	<0.001
Analgesia (Min)	246±40.31	578.56±40.7	<0.001
Sedation Score (1-4)	1	2.8	

Table 4: Comparison of Mean Systolic Bp between group B and group BD

	GROUP B	GROUP BD	p-value
0 min	134.46 ± 2.42	132.3±6.29	>0.05
5 min	130.3±5.62	128±4.65	>0.05
10 min	127.26 ± 6.64	111.23±3.46	<0.05
15 min	122.76 ± 6.58	102.46 ± 6.19	<0.05
20 min	120.4 ± 4.41	104.7 ± 6.61	<0.05
25 min	118.3 ± 4.32	106.9± 6.17	<0.05
30 min	116.43 ± 4.65	104.16 ±2.41	<0.05
45 min	123.56 ± 3.12	102.86±3.30	<0.05
60 min	126.4 ± 2.41	102.72 ± 7.70	<0.05
90 min	118.3 ± 6.36	104.9± 6.27	<0.05
120 min	122.43 ± 4.65	106.16 ±2.42	<0.05
150 min	120.56±12.02	110.86±14.32	<0.05

Table 5: Intra and Postoperative Complications

	GroupB	Group BD
Hypotension	Nil	2(6%)
Bradycardia	Nil	2(6%)
Nausea and vomiting	Nil	Nil
Sedation	Nil	22(66%)
Respiratory depression	Nil	Nil

Discussion

Adding dexmedetomidine as an adjuvant to levobupivacaine has proved to be beneficial as it 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. Centrally, α-2 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 α-2 adrenoceptors in the locus coeruleus. [22] Peripheral action of dexmedetomidine was caused by activation of hyperpolarization activated cation 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 α-2 adrenorecptors activation in a concentration dependent manner and reversibly. [23,24,25] 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).

The α-2 receptor agonist such as dexmedetomidine has demonstrated a dose-dependent increase in the duration of thermal antinociception and analgesia in many animal studies. Dexmedetomidine 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. [21,26,27] Yoshitomi et al, demonstrated that α-2 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. [28] Our study also showed similar results. Swami et al. used dexmedetomidine and clonidine as an adjuvant to bupivacaine 0.25% in supraclavicular plexus block and demonstrated that dexmedetomidine prolongs the duration of sensory and motor block and enhances the quality of block as compared with clonidine. [17] Our study also showed similar results. Ammar et al, used dexmedetomidine 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. [29] Our study also showed similar results.

Masuki et al, suggested that dexmedetomidine 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. [30,31] Esmaoglu et al, reported prolongation of axillary brachial plexus block when dexmedetomidine 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. The limitation of our study was that we could not measure

the levels of levobupivacaine and dexmedetomidine 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.

Dexmedetomidine added as an adjuvant to levobupivacaine 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.

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