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Effects of clonidine and Dexmedetomidine on ropivacaine in ultrasound guided supraclavicular brachial plexus block

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ABSTRACT

Background: Several clinical studies have shown that clonidine prolongs sensory motor blockade when used as adjuvant to ropivacaine but it has its own limitations.**Aim**: To evaluate the effect of clonidine and dexmedetomidine on ropivacaine, for supraclavicular brachial plexus blockade.**Methods**: In a prospective randomised double blind study ultrasound guided supraclavicular brachial plexus blockade was performed in 100 patients using clonidine and dexmedetomidine with ropivacaine. Group C had 1 μg/kg clonidine(1ml) and in Group D 1 μg/kg dexmedetomidine(1ml) added to 30 ml of ropivacaine(0.5%). Sensory and motor blockade was assessed every 5 min till 30 min and at 15 min interval thereafter. **Results**: Mean sensory onset time in group D was 2.44 ± 1.7 min and in group C was 4.85 ± 1.55 min, which was statistically significant. Patients of group D had a mean motor onset time 4.35 ± 1.8 min and patients of group C had a mean motor onset time 8.55 ± 1.64 min, the difference being statistically significant. Mean duration of sensory block in group D was 584.15 ± 63.4 min and in group C was 490.85 ± 72.65 min, which was statistically significant. Patients in group D had a mean duration of motor block 450 $\pm$60.3 min and patients in group C had a mean duration of motor block 330.45$\pm$68.7 min, which was statistically significant.**Conclusion**: The addition of dexmedetomidine to ropivacaine increases the duration of motor and sensory block in brachial plexus blockade.

Keywords: Ropivacaine, Clonidine, brachial plexus, dexmedetomidine

Introduction

Brachial plexus block provides both introperative anaesthesia and postoperative analgesia without any systemic side-effects.[1] Ropivacaine has lower lipid solubility and have produced less central nervous system toxicity and cardiotoxicity than bupivacaine for which it is gaining popularity over bupivacaine for peripheral nerve blocks.[2] There has always been a search for ideal adjuvant to local anaesthetics for regional nerve block that prolong the analgesia with lesser adverse effects.[3].Several clinical studies have shown that clonidine can prolong the duration of analgesia when used in combination with local anaesthetic agents like ropivacaine.[4] But it has its own limitations and side effects.[5] Dexmedetomidine, a highly selective alpha-2 agonist, with an affinity eight times greater than clonidine, has better analgesic properties in peripheral nerve blocks. This research was planned to evaluate the effect of adding clonidine and dexmedetomidine to ropivacaine on sensory motor characteristics in supraclavicular brachial plexus block.

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 11, 2023

Methods

Following approval from the local hospital ethics committee, we conducted this study in a tertiary care hospital from Sept 2021 Sept 2022. 100 patients of American Society of Anesthesiologists (ASA) grade I or II posted for hand or forearm surgery were recruited to a prospective randomised, double blind study. Exclusion criteria were patients of age <18 yr or >60 yr, patients receiving anticoagulants, patients with history of hypertension, peripheral neuropathy and hypersensitivity to local anesthetic agents.Details of the anesthetic technique and the study protocol were fully explained to patients during preanaesthetic check up and informed written consent was obtained from each patient. Relevant investigations were performed as required. Before the procedure, visual analogue scale (VAS) on 0-10 cm. was explained to the patient for the assessment of pain where 0 denotes no pain and 10 denotes worst pain.Patients were randomly allocated by computer generated randomisation list and divided into two groups C and D.

Group C(n=50)- ropivacaine 0.5% (30 ml) with 1 μg/kg clonidine(1ml)

Group D (n=40)- ropivacaine 0.5% (30 ml) with 1 μg/kg dexmedetomidine(1ml)

Study drugs were prepared by the anaesthesiologist, not involved in the trial.On arrival in the operation room, baseline heart rate, blood pressure and oxygen saturation were recorded and monitored throughout the procedure. An intravenous line was secured in the unaffected limb and Ringer's lactate was started. All the patients received brachial plexus block through the ultrasound guided supraclavicular approach by an experienced anaesthesiologist. Following negative aspiration, 31ml of ropivacaine combined with clonidine or dexmedetomidine was injected. Sensory block was assessed by the pin prick method. Assessment of sensory block was done at every 5 minute after completion of drug injection in the dermatomal areas corresponding to median nerve, radial nerve, ulnar nerve and musculocutaneous nerve till complete sensory blockade. Sensory onset was considered when there was a dull sensation to pin prick with a 23G needle along the distribution of any of the above-mentioned nerves. Complete sensory block was considered when there was complete loss of sensation to pin prick.Sensory block was graded as-.[6] Grade 0: Sharp pin felt,Grade 1: Analgesia, dull sensation felt,Grade 2: Anaesthesia, no sensation felt.Assessment of motor block was carried out by the same observer at each 5 minute till complete motor blockade after drug injection. Onset of motor blockade was considered when there was Grade 1 motor blockade. Peak motor block was considered when there was Grade 2 motor blockade. Motor block was determined according to a modified Bromage scale for upper extremities on a 3-point scale. [7] 0 − normal motor function with full extension and flexion of elbow, wrist, and fingers, 1 − decreased motor strength, with ability to move only fingers, 2 − complete motor block with inability to move elbow, wrist, and fingers.The block was considered incomplete when any of the segments supplied by median, radial, ulnar and musculocutaneous nerve did not have analgesia even after 30 min of drug injection. These patients were supplemented with intravenous fentanyl (1 μg/ kg) and midazolam (0.02 mg/kg). When more than one nerve remained unaffected, it was considered a failed block. In this case, general anaesthesia was given .Patients were monitored for haemodynamic variables such as heart rate, blood pressure and oxygen saturation every 15 min after the block intraoperatively and every 60 min post-operatively. Sedation of patient was assessed by the Ramsay Sedation Score. [8] Assessment of blood loss was done and fluid was administered as per the loss. The intra- and post-operative assessment was done by an anaesthesiologist who was unaware of the drug used. Patients were assessed for duration of analgesia as per visual analogue scale of 0 to 10. The visual analogue scale was recorded post-operatively every 30 min till the score of 5. The rescue analgesia was given in the form of inj. IV paracetamol at the visual analogue scale of 5 and the time of administration was noted. All patients were observed for any side-effects like sedation ,nausea, vomiting, dryness of mouth and complications like pneumothorax, haematoma, local anaesthetic toxicity .The duration of sensory

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block was defined as the time interval between the end of local anaesthetic administration and the complete resolution of anaesthesia on all nerves. The duration of motor block was defined as the time interval between the end of local anaesthetic administration and the recovery of complete motor function of the hand and forearm.The sample size was calculated based on a pilot study on 10 patients in each group. To detect clinically significant difference between the two groups (1.8 ± 2) min in onset of motor block at 95% significance and 80% power, the required sample size was 45 participants in each group. To make good for attrition rate, 50 patients in each group were included for the study.Results are expressed as mean ± standard deviation .Unpaired t – test was applied for demographic data, haemodynamic parameters. Onset and duration of sensory and motor blockade and duration of analgesia was analysed by Chi-square test. Statistical analysis was performed by SPSS (version 21). P-value was considered significant if <0.05.

Results

There was no statistically significant difference between the demographics like age, sex, ,height,weight, ASA grade and duration of surgery**.** (Table I).

Table 1 : Distribution of subject according to demographic profile

Variables	Group D	Group C	P value
Age (year)	35.6±10.8	36.5±9.4	0.936
Sex ratio (M/F)	42/8	40/10	0.895
Weight (kg)	61.4±11.42	62.2±10.64	0.467
Height (cm)	164±12.67	165.3±11.68	0.400
ASA Grade (I/II)	40/10	40/10	0.895
Duration of Surgery (min)	92.6±28.4	91.8±29.6	0.922

No statistical difference was noted in the hemodynamic parameters (mean HR, MAP, SpO_2) before and after giving the block, throughout the surgery and postoperatively.

Table 2: Characteristics of sensory and motor block in group D and group C

Parameters	Group D Mean ±SD	Group C Mean ±SD	P value
Onset of sensory block (min)	2.44±1.7	4.85±1.55	0.002
Onset of motor block (min)	4.35±1.8	8.55±1.64	0.013
Duration of sensory block (min)	584.15±63.4	490.85±72.65	0.023
Duration of motor block (min)	450 ±60.3	330.45±68.7	0.016
Duration of analgesia (min)	686±56.8	565.8±62.5	0.101

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 11, 2023

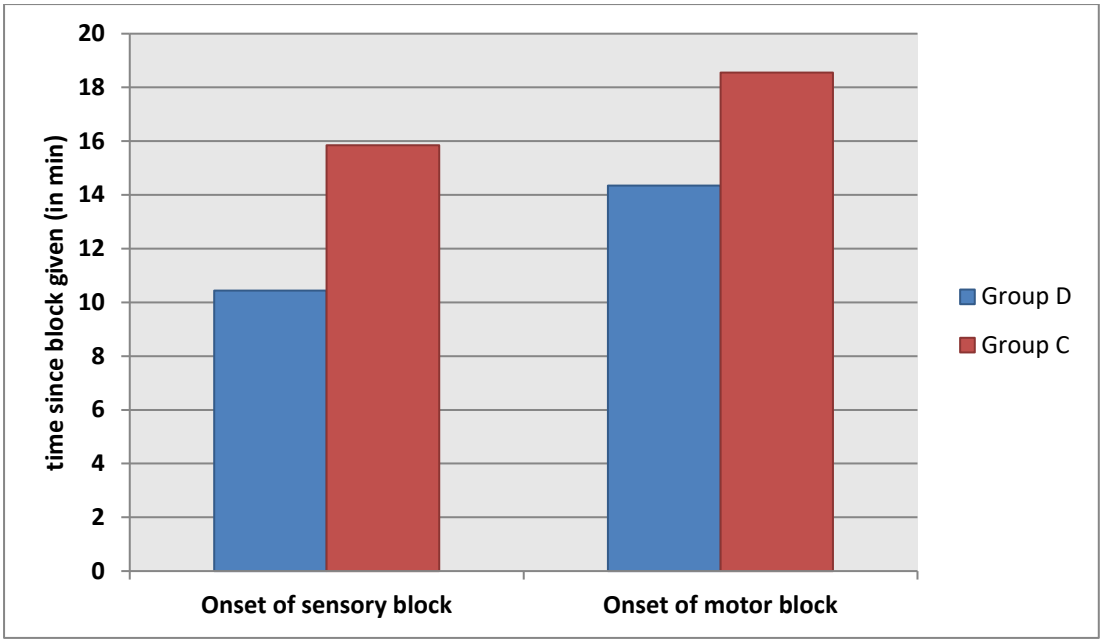

Figure1-Comparison between Group D and Group C for time of onset for sensory and motor block.

The onset of block was earlier in group D patients ,that was 2.44±1.7min for sensory block and 4.35±1.8 min for motor block than those in group C 4.85±1.55min for sensory block and 8.55±1.64 min for motor block, which was statistically significant (P < 0.05)(table 2)(figure1).

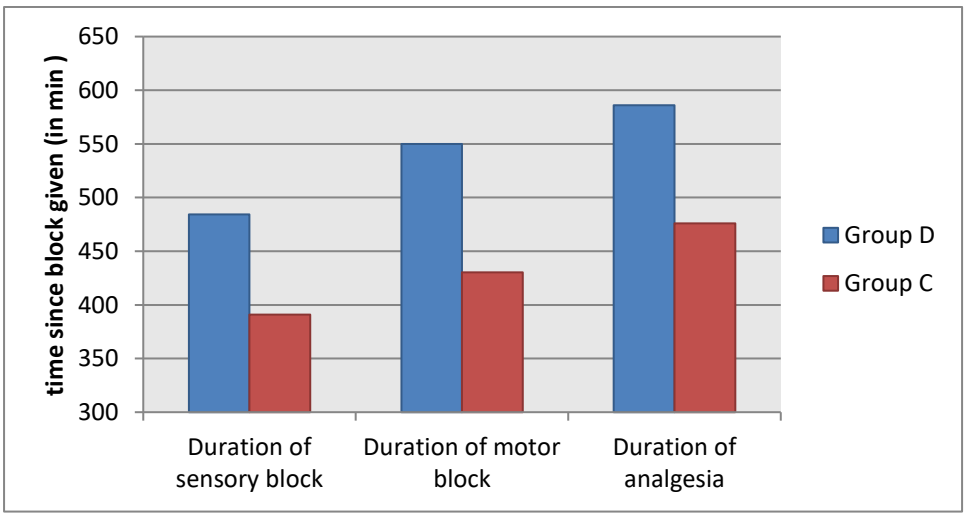

Figure 2-Comparison between Group D and Group C for duration of sensory , motor block and analgesia .

Mean duration of sensory block in group D was 584.15±63.4 min and in group C was 490.85±72.65min. Patients belonging to group D had a mean duration of motor block 450 ±60.3min and group C had a mean duration of motor block 330.45±68.7min, which was statistically significant.(table 2)(figure 2) Postoperative analgesia lasted 686±56.8min in group D and 565.8±62.5 min group C which was also statistically significant (P < 0.05).

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 11, 2023

Discussion.

The analgesic properties of clonidine when administered intrathecally or epidurally as adjuvant have been well demonstrated[9].They seem to be attributable to its α_2-agonist properties. Its action on large no α_2 receptors present in the central nervous system, at loecus coeruleus and dorsal horn of the spinal cord, is the main mechanism of centrally mediated sedation and analgesia.[10] Specific peripheral effects of clonidine appear less obvious because α_2-adrenoreceptors are not present on the axon of the normal peripheral nerve.[10] It has been postulated that clonidine improved the duration of postoperative analgesia only when used as an adjuvant to intermediate-acting local anaesthetics and that it was not worthwhile to combine it with long-acting local anaesthetics. In our study, we have observed that addition of dexmedetomidine significantly shortened the onset of sensory and motor block. Waindeskar V et al.,[8] concluded that dexmedetomidine significantly shortens the onset time and prolongs the duration of sensory and motor blocks and also postoperative analgesia when added to levobupivacaine in ultrasound guided block.This observation well matches with Kathuria et al.[9] Das et al.[10] who found that onset of sensory as well as motor blocks was earlier in dexmedetomidine group though the differences were not statistically significant.Murphy et al . [11]. analyzed randomized trials that investigated the usefulness of a variety of adjuvants, including clonidine added to local anaesthetics for brachial plexus block. On the basis of data from six trials (349 patients), they concluded that clonidine in doses up to 150 μg increased the duration of postoperative analgesia with minimal adverse effects .McCartney et al. [12] reviewed 27studies (1,385 patients) using clonidine as an adjuvant to local anaesthetics for a variety of peripheral nerve blocks. They concluded that clonidine was beneficial only when added to intermediate-acting local anaesthetics.Antonucci S [13]evaluated effects of tramadol used as adiuvant in brachial plexus block and compared with clonidine and sufentanil. He used ropivacaine for block and concluded that tramadol as adiuvant provides a significative redution of onset time of sensory motor block and also provides a prolongation of anesthesia and analgesia with a quality of block similar that obtained with clonidine and sufentanil.El Saied et al,[14] conducted a study in which axillary brachial plexus blockade was performed in 50 patients using 40 ml ropivacaine 0.75 %. Group (A) had 150 μg clonidine and Group (B) 1ml normal saline added to the local anesthetic. There was no difference in onset of sensory motor blockade. They concluded that the addition of 150 μg of clonidine to ropivacaine, for brachial plexus blockade, prolongs motor and sensory block and analgesia, without an increased incidence of side effects. Aggarwal S et al,[15] have also concluded that dexmedetomidine when added to local anaesthetic agents prolonged the duration of motor block. It also resulted in faster onset of sensory and motor block.Sebastian D et al,[16] compared the effects of clonidine and dexmedetomidine and observed that dexmedetomidine is a better agent than clonidine in terms of increased postoperative analgesia in supraclavicular block. Our study was also in agreement with studies by Patki et al [17], Kanvee et al [18], Harshavardhana et al [19] and Channabasappa et al [20].The above studies shows that selective α_2-adrenoceptor agonist like clonidine or dexmedetomidine when added as adjuvant to ropivacaine in different peripheral nerve blocks ,potentiates the sensorymotor blockade. The mechanism is not clear. Probably peripherally, α_2-agonists produce analgesia by reducing release of norepinephrine and causing α_2-receptor-independent inhibitory effects on nerve fibre action potentials. Centrally, α_2-agonists cause analgesia and sedation by inhibition of substance P release in the nociceptive pathway at the level of the dorsal root neurone and by activation of α_2-adrenoceptors in locus coeruleus. So the action of dexmedetomidine would then more likely be via a synergistic mechanism of action in combination with the local anesthetic resulting in the prolonged effect.

Conclusion

Dexmedetomidine, when added to ropivacaine for supraclavicular brachial plexus block produces quick onset of sensory motor block and provides prolonged postoperative analgesia which lasts longer than that produced by ropivacaine with clonidine and without any significant side-effects.

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ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 11, 2023

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