Section: Anaesthesiology

Original Research Article

ULTRASOUND-GUIDED SINGLE INJECTION INFRACLAVICULAR BRACHIAL PLEXUS BLOCK USING BUPIVACAINE ALONE OR COMBINATION COMBINED WITH DEXMEDETOMIDINE FOR PAIN CONTROL IN UPPER LIMB SURGERY

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Abstract

Background: Dexmedetomidine, a selective 2-adrenoceptor agonist, is used with local anaesthetics as an adjuvant when performing local anaesthesia. This study aimed to evaluate the effectiveness of mixing dexmedetomidine with bupivacaine during infraclavicular brachial plexus blockade (ICB) placement. **Materials and Methods:** This prospective randomised study was conducted at the department of anesthesia, Madurai Medical College from March 2017 to July 2018. 60 Patients were randomly assigned to two groups. Group I patients received ICB using 30 mL of 0.33% bupivacaine, whereas Group II patients received ultrasound-guided ICB with 30 mL of 0.33% bupivacaine mixed with 0.75 µg/kg of dexmedetomidine. **Result:** In the present study, Group II patients showed a statistically significant shorter time to onset of a sensory blockade than Group I patients (14.167 vs 19.8 min) and longer sensory block duration (178.7vs 122.46 min). Group II also exhibited a statistically significant shorter onset time to motor blockade (15.53 vs 22.13 min) and longer motor block duration (155.33 vs 104.46 min). In addition, group II showed a statistically significant longer duration of postoperative analgesia (400.33 vs 232.6 min) and lower rescue morphine requirements 48 h after surgery. **Conclusion:** Dexmedetomidine can be used as an adjuvant combined with local anaesthetics for brachial plexus blocking to improve pain control and extend the duration of local anaesthetic anaesthesia according to improved analgesic effectiveness criteria.

INTRODUCTION

Surgery on the hand, forearm, elbow and distal humerus is possible with infraclavicular brachial plexus blockade (ICB). Before they branch from the brachial plexus sheath, the cord level is where the axillary and musculocutaneous nerves are impacted or inhibited. An infraclavicular block can pose a far lower danger of intravertebral, intrathecal, or epidural injection and a lower risk of phrenic nerve paralysis or stellate ganglion block than interscalene and supraclavicular brachial plexus blockade. Pneumothorax, hematoma, and nerve damage are nonetheless rare risks associated with ICBs.[1-2] Clonidine, an α2-adrenergic agonist, has been added to local anaesthetics used during regional anaesthesia. Analgesia and motor blockade are extended by around 2 hours when clonidine is combined with intermediate and long-acting local anaesthetics during a single-shot peripheral nerve or nerve plexus block, according to a recent meta-analysis.[3] A selective 2-adrenoceptor agonist, dexmedetomidine, has been utilised as an adjuvant during regional and local anaesthesia.4 Animal and human studies have demonstrated the effectiveness and safety of mixing dexmedetomidine with local anaesthetics during regional anaesthesia procedures, including subarachnoid, epidural, and caudal injections. However, other studies have found diminished or adverse analgesic effects when dexmedetomidine is used.[5-6] Use of dexmedetomidine as an adjuvant mixed with local anaesthetics has been performed with neuraxial anaesthesia in both adult and pediatric patients. Dexmedetomidine (5 µg) added to intrathecal bupivacaine during gynecologic surgeries has resulted in a longer sensory and motor block

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duration. In another study, dexmedetomidine (3 µg) in combination with bupivacaine for spinal anaesthesia has been shown to provide a shorter onset to motor blockade and prolongation of motor and sensory block along with preservation of hemodynamics and absence of sedation.[6-7] However, there remains limited knowledge on the analgesic efficacy and clinical utility of adding dexmedetomidine to local anaesthetics during peripheral nerve and nerve plexus blockade in humans.[8] Therefore, this study was designed to investigate the efficacy of dexmedetomidine as an adjuvant in combination with local anaesthetic solutions during an ICB for upper extremity surgery. So, it has been hypothesized that dexmedetomidine may improve overall efficacy during an ICB.

MATERIALS AND METHODS

This prospective randomised study was conducted at the department of anesthesia, Madurai Medical college from March 2017 to July 2018. A total of 60 patients aged from 18 to 60 years with ASA grade I and II admitted for elective upper limb surgery were enrolled. The written consent and Institutional ethical committee approval were taken before the start of the study. Patients whose medical history, laboratory data, or physical examination showed evidence of abnormal hepatic or renal function or severe cardiovascular, pulmonary, neurological, psychiatric, or metabolic disease were excluded from the study.

Sample Size

A power analysis was performed to determine the necessary number of patients for each group based on the duration of analgesia. With a 2- sided type I error of 5% and study power at 80%, it was estimated that 30 patients would be needed in each group to detect a difference of 35 min in the duration of analgesia between the 2 groups

Inclusion criteria:

All Adults patients aged 18 to 65 years of both sexes with ASA I / II grading, admitted for Upper limb surgeries, were included.

Exclusion Criteria

Patients with local infection, known allergy to local anaesthetics drug. Patient on anticoagulant therapy, psychiatric illness and Inadequate block requiring supplemental anaesthesia. Patients with a history of bleeding disorders who refused to participate in the study were excluded.

Methods

Patients scheduled for elective upper limb surgery were eligible for the study. 60 Patients were randomly assigned to two groups. Group I: Patients received ultrasound-guided infraclavicular brachial plexus block using 30 mL of 0.33% bupivacaine. Group II: Patients received ultrasound-guided infraclavicular brachial plexus block 30 mL of 0.33% bupivacaine mixed with 0.75 µg/kg dexmedetomidine. The following parameters were assessed during the study: HR, BP, SPO2 saturation, block success rate, sensory onset time and duration, motor block onset time and duration, analgesic pain scores using the verbal rating scale (VRS) for pain, duration of analgesia, and amount of supplemental intravenous (IV) morphine required.

Statistical Analysis

The information collected regarding all the selected cases was recorded in a Master Chart. Data analysis was done by using SPSS 16 and Sigma Stat 3.5. Using this software, range, frequencies, percentages, means, standard deviations, chi-square and 'p' values were calculated by one-way ANOVA. The Chi-square test was used to test the significance of the difference between quantitative variables. A 'p-value less than 0.05 is taken to denote a significant relationship.

RESULTS

Both groups of patients had comparable demographic variables like age distribution, gender, weight, height, and ASA grading I and II (P>0.05), as shown in [Table 1]

Table 1: Demographic variable of patients in both I and II groups

Variables		Group I	Group II
Gender	Male	15 (50%)	16 (53.33%)
	Female	15 (50%)	14 (46.67%)
Age Group	<30 years	4 (13.33%)	4 (13.33%)
	31- 40years	15 (50%)	16 (53.33%)
	>40 years	11 (36.66%)	10 (33.33%)
Mean (years ± SD)		37.3 ± 6.51	38.67 ± 5.97
ASA Class	ASA I	18 (60%)	17 (56.66%)
	ASA II	12 (40%)	13 (43.33%)
Weight (kg) Mean ± SD		65.07 ± 3.92	64.1 ± 5.02
Height (cm) Mean ± SD		162.93 ± 4.27	163.98 ± 4.69

The successful blockade was achieved in 96.7% of patients in both groups. In addition, all patients recovered uneventfully without sensory or motor deficit and no evidence of respiratory depression, bradycardia, or hypotension was reported.

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The dexmedetomidine group of patients (Group II) showed a statistically significant shorter time to onset of sensory blockade (14.167 vs 19.8 min, P=0.001), longer sensory block duration (178.7vs 122.46 min, P=0.001) [Table 2].

Table 2: Observation of onset and duration of sensory block in both groups

	Group I	Group II	p-value
The onset of sensory block (min)	19.8±2.73	14.16±4.17	<0.001
Duration of sensory block (min)	122.46±12.77	178±15.66	<0.001

The dexmedetomidine group of patients (Group II) showed a statistically significant shorter onset time to motor blockade (15.53 vs 22.13 min, P=0.001), longer motor block duration (155.33 vs 104.46 min, P=0.001) [Table 3].

Table 3: Observation of onset and duration of motor block in both groups

	Group I	Group II	p-value
The onset of motor block (min)	22.13±4.42	15.53±4.55	<0.001
Duration of motor block (min)	104.46±21.39	155.33±16.96	<0.001

The dexmedetomidine group of patients (Group II) showed a statistically significant longer duration of postoperative analgesia (400.33 vs 232.6 min, P=0.001) and lower rescue morphine requirements 48 h after surgery.

Table 4: Observation of duration of analgesia and IV morphine required in both groups

	Group I	Group II	p-value
Duration of analgesia (min)	232.66±37.59	400.33±44.21	<0.001
IV morphine needs over 48 hours (mg)	12.97±3.15	5.37±1.79	<0.001

DISCUSSION

Apart from the sedative, analgesic, hemodynamic-stabilizing properties, and sympatholytic pharmacologic effects, the alpha (a)-2-adrenergic receptor (a2-AR) agonists have been used to increase the duration of thermal anti-nociception and analgesia. In addition, dexmedetomidine decreases inflammation around peripheral nerves, thereby decreasing the potential for peripheral nerve injury.[6-7] In human beings, the beneficial effects of adding dexmedetomidine to local anaesthetics during regional anaesthesia and some peripheral nerve blockade procedures have proved to be efficacious for the surgical patients.[8] In our study, we have found that the addition of dexmedetomidine (0.75mic/kg) to 30 ml bupivacaine 0.33% in ultrasound-guided infraclavicular brachial plexus block resulted in a quick onset of sensory and motor block prolonged duration of both sensory and motor block delayed time to first request for analgesia supplementation, that is, prolonged duration of analgesia, and significantly decreased 48 hr. analgesic consumption and good quality of analgesia when compared with a control group (bupivacaine 0.33% alone in block) and without significant side effects. Dexmedetomidine (1 g/kg) was introduced to bupivacaine by Saadawy and colleagues for caudal anaesthesia in kids, resulting in prolonged analgesia, reduced need for rescue analgesics, and better sleep quality without any unfavourable clinically significant side effects.[9] In paediatric caudal anaesthesia for lower abdominal procedures, El-Hennawy et al. combined bupivacaine with either dexmedetomidine or clonidine.[10] Both dexmedetomidine and clonidine medications mixed with bupivacaine significantly prolonged analgesia when compared with using bupivacaine alone 16 h (15–19 h) for dexmedetomidine, 12 h (3–21 h) for clonidine, and 5 h (4–6 h) with plain bupivacaine; P<0.001). The study showed no difference in analgesia duration (P=0.796) between either dexmedetomidine or clonidine when added to bupivacaine. The effects of combining dexmedetomidine with local anaesthetics during peripheral nerve and nerve plexus blockade have been examined in recent studies. In research, Obayah et al. used bupivacaine and dexmedetomidine when inserting a larger palatine nerve block for cleft palate repair.[8] When compared to bupivacaine alone, adding dexmedetomidine resulted in lower pain ratings and longer analgesia (about 50%), with no adverse effects on hemodynamics. Dexmedetomidine and levobupivacaine were combined in different research by Esmaoglu et al. during the insertion of an axillary brachial plexus blockade, which led to a faster block start and a longer block duration, which enhanced postoperative analgesia.[11] The analgesic effect of 2-adrenoceptor agonists has been attributed to some potential modes of action. Some of these include vasoconstriction around the injection site, a complex interaction with axonal ion channels or receptors that directly suppresses impulse propagation through neurons, local release of enkephalin-like substances, a decrease in localised inflammatory mediators, and an increase in anti-inflammatory cytokines through a 2-adrenoceptor-

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mediated mechanism.[12-13] According to research by Duma and colleagues, there is no change in the analgesic effectiveness when levobupivacaine and clonidine are combined during the insertion of an axillary brachial plexus blockade.[14] These findings may be explained by the inherent vasoconstrictive properties of levobupivacaine, which may counteract or impede any beneficial vasoconstrictor effects of clonidine, or by a difference in the pharmacokinetic effects brought on by utilising various amounts of clonidine. The research findings might also be influenced by anatomical variances of the brachial plexus or by various nerve block techniques that could result in barriers or differences in the diffusion of local anaesthetics inside the brachial plexus neurovascular bundle. Variations in the pace and depth to which injected anaesthetic solutions penetrate neurons might also be a factor in the lack of effectiveness when clonidine is added to local anaesthetics.[15]

CONCLUSION

In infraclavicular brachial plexus block addition of dexmedetomidine (0.75 mic/kg) as an adjuvant to 0.33%, bupivacaine shortens the sensory and motor block onset time, prolongs both sensory and motor block duration. It also significantly delays the first demand for analgesia supplementation, decreases 48 hrs. analgesic consumption and is not associated with any major side-effect. The action of dexmedetomidine is most probably peripheral than centrally mediated.

Limitation of Study

The major limitation of this study was that we did not measure the levels of dexmedetomidine in the plasma, which could have further supported the hypothesis that dexmedetomidine has a peripheral action rather than a centrally mediated cost of ultrasound.

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