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Ketamine: As an Adjuvant to Supra Clavicular Brachial Plexus Block

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Abstract

Regional anesthesia provides a safe anesthesia for upper extremity surgery. Supraclavicular brachial plexus block is one of the most effective anesthetic procedures for the upper extremity procedures. Ketamine has been reported to enhance the analgesic effects of local anesthetics.

Aims and Objectives: This study was used to assess the efficacy of ketamine in prolonging the local analgesic effect of bupivacaine in the supraclavicular brachial plexus block for patients undergoing elective upper extremity surgery.

Materials and Methods: 50 adult patients undergoing elective surgery of the elbow, forearm, wrist or hand were randomly allocated in two groups of 25 patients each. Group I (ketamine group) received 2 mg/kg bupivacaine 0.5% plus 2 mg/kg ketamine, Group II (control group) received 2 mg/kg bupivacaine 0.5% and saline.Visual analog scale (VAS, 0 = no pain 10 cm = the most severe pain) was used to assess the severity of pain. Time of first request for analgesia and total dose of postoperative rescue analgesia was calculated. The data was analyzed using the student's t-test.

Results: Patients in the control group had a higher VAS than patients who received ketamine during the first 24 hours after surgery. The time (in hours) of first rescue analgesia in the ketamine group was significantly more than in the control group [5.83(±1.33) vs 3.53(±1.14) , respectively].

Conclusions: The addition of ketamine as an adjuvant to bupivacaine in the brachial plexus block could decrease the postoperative pain without any side effects and also decrease the need for rescue analgesia.

Keywords: *ketamine, bupivacaine.*

Introduction

The use of adjuvants in combination with local anesthetics for peripheral nerve blocks enhances the quality and duration of anesthesia and postoperative analgesia [1–5].

Bupivacaine is a long-acting amide local anesthetic agent when used in 0.5% concentration.[6] It prevents conduction of nerve impulses by reducing sodium permeability & increasing action potential threshold. The effect of 0.5% bupivacaine may persist for 2-3 hours if administered alone. [7] Ketamine is a noncompetitive antagonist of the N-methyl-D aspartate receptor (NMDAR). It acts by blocking the voltage gated sodium channels . It is used for premedication, sedation, induction, and maintenance of general anesthesia.[8] Ketamine is a well-known anesthetic agent with potent local effect on peripheral nerves. Intravenous (IV) administration of low dose ketamine decreases postoperative opioid use and improves analgesia[9]. The addition of ketamine to epidural lidocaine or bupivacaine

increases the duration of regional anesthesia and postoperative analgesia.[10]

Aim of The Study

To determine the effect of the addition of 2mg/kg ketamine to 0.5% bupivacaine in supra-clavicular brachial plexus block on the time of onset, duration of block, postoperative analgesia time and any side effects.

Materials and Methods

Fifty patients, from the age of 20-60 years, with ASA I&II, undergoing upper limb below elbow surgery, had been chosen to be enrolled in the study. They had been divided randomly according to a random allocation table, into 2 groups. Group I received 30 ml of solution 1 (20 ml of 0.5% bupivacaine with 10 ml of normal saline) as placebo or control group. Group II received 30 ml of solution 2 (20 ml of 0.5% bupivacaine with 10 ml of fluid containing 2mg/kg body weight ketamine) as ketamine group. After arrival to the anesthetic room, the peripheral intravenous (IV) line was placed in the non-operative upper limb and an infusion started with normal saline. Blood pressure, pulse rate, respiratory rate, ECG and SpO2 was monitored from the beginning to the end of the operation. Supplemental oxygen (4 L/minute) was delivered by a nasal cannula. IV Midazolam 0.04 mg/kg was administered to all patients as premedication. All local anesthetic solutions and adjuvant drugs were prepared by an anesthesiologist not involved in performing brachial plexus block or data collection. Both the patient and the surgeon involved had been blinded from the drug mixture that had been given to the patient. All observations were carried out by a single investigator, who was also blinded to the treatment group. The skin of the supraclavicular fossa was properly cleaned and draped. Ultrasound-guided supraclavicular brachial plexus nerve block was carried out with a linear high frequency (8-13 MHz) probe (covered with a sterile dressing). The brachial plexus was visualized as a group of round or oval hypoechoic nodules (trunks and/or divisions) superficial and lateral to the round pulsating hypoechoic subclavian artery. A 22-gauge insulated needle was attached to the nerve stimulator and then introduced lateral to the ultrasound probe, and parallel to the long axis of the probe, in the same plane as the ultrasound beam. Once the needle penetrated the brachial plexus sheath and its tip was positioned among the nerves, the nerve stimulator was turned on and set to deliver a 1.0 mA current at 1 Hz frequency. The current was decreased slowly when motor responses in the biceps or triceps were obtained. After a negative aspiration the local anesthetic was injected at the site for over three to five minutes. Local anesthetic dispersion at the time of injection was seen by ultrasound. Evaluation of the sensory and motor blocks was performed after administration of the local anesthetic. The sensory block was quantified as:

0 = Anesthesia (no sensation), 1 = Analgesia (decreased /dull sensation), and 2 = no block (normal sensation), by using the pinprick test and comparing with the contralateral limb. The time from injection to loss of sensation was taken as onset of sensory block. The motor block was evaluated by assessing the flexion and extension of the forearm, opposition of the thumb and second digit, and opposition of the thumb and fifth digit. It was scored as follows: 0 = complete block (no muscle activity). 1 = partial block (decreased muscle activity), and 2 = no block (normal muscle activity), when compared with the contralateral limb. A score of 0 was taken as a complete motor block and the interval between the injection and block completion was considered as the onset of the motor block. For patients in whom block success was not obtained 30 minutes after injection of the local anesthetic or the patient complained of pain at the surgical site during surgery, the patient was excluded from

data analysis. The time when the patients experienced the first postoperative pain was considered to be the end of the sensory block,[11,12]. The visual analogue scale (VAS) was assessed and recorded at 30 minutes ,1,6, 12, and 24 hours after the operation, by an anesthesiologist, who was not aware of the group of study drugs (0 = no pain and 10 = the worst pain experienced). During postoperative recovery in the hospital, pain (VAS >4) was treated with IV tramadol 50mg. The time to the first rescue analgesia and the total tramadol dose were documented. Side effects (if any) were also noted and documented.

Results

Fifty subjects aged 20-60 years participated in this study. All the demographic and operative data were similar between groups [Table 1]. No significant differences were noted between groups in the onset and duration of the motor and sensory blocks [Table 2]. Time of administration of the first dose of tramadol was significantly more in the ketamine group, but the total tramadol dose used as rescue analgesia was less. [Table 3]. Comparison of VAS between groups at various times, postoperatively, has been presented in Table 4. The most significant and highest VAS pain scores were found in the control group at all time points ($P < 0.05$). There were no significant differences between the groups in heart rate (HR) or mean arterial pressure (MAP) when they were recorded before anesthesia (baseline), every 10 minutes after brachial plexus block, upon arrival to the Recovery Unit and at 1,6,12 & 24 hrs. No adverse effects were recorded.

Table 1: Demographic & Operative data:

Variables	Bupivacaine	Bupivacaine + Ketamine
Weight	67.7(±9.8)	66.8(±10.7)
Age	32.7(±14)	33.1(±12.2)
Duration of surgery(min)	72(±25)	75(±26)
ASA [I/II]	17/8	19/6
Sex [male/female]	18/7	20/5
Type of surgery [elbow/forearm/hand]	5/11/9	6/8/11

Table 2: Onset and duration of sensory & motor blocks:

Variable	**Bupivacaine**	**Bupivacaine+ Ketamine**
Onset of sensory block (min)	6.9(±2.98)	7.12(±1.23)
Onset of motor block (min)	9.57(±3.79)	9.38(±3.90)
Duration of sensory block(min)	186.45(±24.56)	192.45(±30.19)
Duration of motor block(min)	200.12(±25.66)	213.83(±28.61)

Table 3: Time of administration of first dose and total dose of tramadol

Variable	Bupivacaine	Bupivacaine + Ketamine
Time of administration of first dose of tramadol (min)	212.1(±68.9)	349.8(±79.9)
Total tramadol dose (mg)	134.6(±47.8)	99.7(±39.9)

Table 4: Pain scores (measured by VAS) in each treatment group at different time intervals postoperatively

Variable	Bupivacaine	Bupivacaine + Ketamine
1hour postoperatively	1.654(±0.897)	0.875(±0.107)
6hours postoperatively	2.981(±1.11)	1.991(±0.087)
12hours postoperatively	3.120(±1.24)	2.180(±1.053)
24hours postoperatively	3.781(±1.20)	2.558(±1.001)

Discussion

Our results suggest that the addition of ketamine to 0.5% lidocaine for ultrasound-guided brachial plexus block reduces the postoperative pain. The onset time and duration of sensory or motor block is not affected. There was no statistically significant difference in the measured hemodynamic variables (heart rate,blood pressure) between the two groups. The first rescue analgesia was required earlier in bupivacaine group [212.1(±68.9)minutes] compared to the bupivacaine+ketamine group in which the request for first rescue analgesia was made in 349.8(±79.9) minutes. The total dose of rescue analgesia was less in bupivacaine+ketamine group [99.7(±39.9)mg]. It was 134.6(±47.8) mg in bupivacaine group.

The pain scores (measured by VAS) were less in bupivacaine+ketamine group at various time intervals after surgery .

Ketamine is used for premedication, sedation, induction, and maintenance of general anesthesia. The duration of regional anesthesia is prolonged by the addition of ketamine (10-50 mg) to epidural bupivacaine or lidocaine . This enhancement of anesthesia by ketamine is more likely the result of the direct action of ketamine on the nerve root fibres rather than the action on the spinal cord. Local anesthetic properties of ketamine were demonstrated by Dowdy et al.,[13] who reported that ketamine could produce reversible inhibition of the compound action potential in the stimulated frog sciatic nerve. The effect of ketamine on nerve conduction was confirmed by Weber et al.,[14] who reported that the subcutaneous infiltration of ketamine caused a loss of thermal and pain sensations for eight to ten minutes.

In our study the addition of ketamine to bupivacaine did not improve the onset or duration of

sensory or motor block. These findings are similarly to the study of Lee et al.,[15] who showed that the addition of 30 mg of ketamine to 30 ml of 0.5% ropivacaine in the brachial plexus block did not improve the onset time or duration of sensory and motor block. However, contrary to the same study , postoperative pain and need for analgesics in the ketamine group were decreased in our study. The analgesic effect could be the result of the local anesthetic effect of ketamine at the level of surgical trauma. Tverskoy et al.[16] showed that in patients whose wound were infiltrated with a solution of bupivacaine 0.5% and ketamine 0.3%, enhancement of the local anesthetic and analgesic effects of bupivacaine could not be explained by a central action of ketamine, and therefore, this effect was most likely peripheral. The role of NMDAR in processing the nociceptive input could explain the analgesic properties of ketamine. The effect of analgesia by ketamine is prolonged when administered with the local anesthetic agents due to the higher concentration of ketamine in the pre-neural fat than in the systemic circulation. It exerts the effect on neuronal transmission by blocking the Na-K ion gates at the peripheral nerves.[17,18]

In contrast, in some studies, the addition of ketamine to local anesthetics has not improved the peripheral,

regional, or local analgesia. Rahimzadeh et al.[19] compared the analgesic effects of peri-femoral nerve infusion of ketamine plus ropivacaine versus ropivacaine, after operation, in patients who underwent elective knee surgery for repairing the ACL, under spinal anesthesia. They reported that the addition of ketamine 1 mg/kg to 0.1% ropivacaine could not improve postoperative pain relief in the first 48 hours after the operation. Zohar et al.[20] reported that ketamine added to local bupivacaine did not enhance analgesia after wound infiltration following Cesarean section. The addition of ketamine to local anesthetics failed to improve analgesia after intra-articular injection for knee arthroscopy.[21] The variable effect of ketamine in various studies probably came from the different ketamine concentrations and sites of injection. We administered 2mg/kg ketamine and it was more than what the previously mentioned studies had used.Our study showed that ketamine decreased the severity of postoperative pain till 24 hours after surgery. The long-lasting analgesic effect of ketamine could be due to the inhibition of central sensitization or due to the blockade of neuronal transmission peripherally.

Conclusion

Our study showed that the addition of ketamine 2 mg/kg to bupivacaine in the brachial plexus block decreased the postoperative pain and need for analgesics, without causing any adverse effects. Therefore, it could be considered a good choice as an adjuvant to local anesthetics in a brachial plexus block.

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