ROLE OF DEXAMETHASONE IN BRACHIAL PLEXUS BLOCK

Muhammad Dawood, Amjad Iqbal*, Sarfraz Janjua, Arooma Dawood***, Jehangir Ahmed Afridi****

2-Mountain Medical Battalion Azad Jammu and Kashmir, *Combined Military Hospital Rawalpindi, ** Combined Military Hospital Gujranwala, ***Sheikh Zayed Hospital Lahore

ABSTRACT

Objective: To evaluate the effect of dexamethasone added to (lignocaine) on the onset and duration of axillary brachial plexus block.

Study Design: Randomized controlled trial.

Place and Duration of Study: Combined Military Hospital Rawalpindi, from September 2009 to March 2010.

Patients and Methods: A total of 100 patients, who were scheduled for elective hand and forearm surgery under axillary brachial plexus block, were randomly allocated to group A in which patients received 40 ml 1.5% lidocaine with 2 ml of isotonic saline (0.9%) and group B in which patients received 40 ml 1.5% lidocaine with 2 ml of dexamethasone (8 mg). Nerve stimulator with insulated needle for multiple stimulations technique was used to locate the brachial plexus nerves. After the injection onset of action and duration of sensory blockade of brachial plexus were recorded at 5 minutes and 15 minutes interval.

Results: Group A showed the onset of action of 21.64 ± 2.30 min and in group B it was 15.42 ± 1.44 min ($p< 0.001$). Duration of nerve block was 115.08 ± 10.92 min in group A and 265.42 ± 16.56 min in group B ($p < 0.001$).

Conclusion: The addition of dexamethasone to 1.5% lignocaine solution in axillary brachial plexus block prolongs the duration of sensory blockade significantly.

Keywords: Brachial plexus block, Dexamethasone, Lignocaine.

INTRODUCTION

The brachial plexus is formed by the ventral rami of C5-C6-C7-C8-T1, occasionally with small contributions by C4 and T2. There are multiple approaches to blockade of the brachial plexus, beginning proximally with the interscalene block and continuing distally with the supraclavicular, infraclavicular, and axillary blocks[1]. Advantages of using brachial plexus over general anaesthesia are that there are less chances of decrease in blood pressure, undesirable decreases in cardiac output, central nervous system depression, respiratory depression, loss of protective airway reflexes (such as coughing), need for tracheal intubation and mechanical ventilation, and residual anesthetic effects. Although brachial plexus block is not without risk, it is usually less invasive and affects fewer organ systems than general anesthesia[2].

Brachial plexus block is one of the most commonly used peripheral nerve blocks in clinical practice. It can be used as the sole anesthetic technique or in combination with general anaesthesia for intraoperative and postoperative analgesia. Continuous catheterization of the brachial plexus is one of the best methods of postoperative analgesia.

This block was performed by William Steward Halsted first time in 1889. He directly exposed the brachial plexus in the neck to perform the block and used cocaine[3,4].

After that Herschel[5] , Kulenkampff[6], Winnie and Collins[7] described the percutaneous, supraclavicular and perivascular approach to the brachial plexus block respectively.

The drugs commonly used for brachial plexus block are 2% lignocaine with adrenaline, 0.5% bupivacaine and by adding opioids to the local anesthetic.

Correspondence: Maj Muhammad Dawood, 2 Mountain Med Bn, Kotli, Azad Jammu Kashmir.
Email: dr.daud@hotmail.com
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Neuraxial blockade in elderly population needs less sedation, favoring early mobilization and excellent analgesia postoperatively[8]. Regional anesthesia for forearm surgery can be provided by brachial plexus block through axillary approach, where surrounding nerves are injected with local anesthetic agents[9]. Increasing the duration of local anesthetic action is often desirable as it prolongs surgical anesthesia and analgesia. Different additives have been used to prolong regional blockade[10]. Vasoconstrictors can be used to reduce vascular absorption of the local anesthetic. Synergistic interaction can also occur when drugs affect different critical points along a common pathway[11].

Different additives have been used to prolong regional blockade. It has been recently demonstrated that the nerve blocks are the ideal techniques for day case surgery[12]. Addition of dexamethasone to the local anesthetic increases the duration of the block[13,14].

The purpose of this study was to evaluate the effect of dexamethasone added to (lignocaine) on the onset and duration of axillary brachial plexus block.

PATIENTS AND METHODS

These randomized controlled trails were conducted in the department of anaesthesiology, CMH Rawalpindi, from Sep 2009 to Mar 2010.

Patients, who were scheduled for elective hand and forearm surgery under axillary brachial plexus block, of either sex, aged 20 to 80 years having ASA grade I and II, who were willing to undergo the trial were included.

Patients with a history of peptic ulcer disease, diabetes mellitus, hepatic or renal failure, pregnant women, morbidly obese with BMI more than 35, having coagulopathy or taking any premedications like opioids, benzodiazepines, and clonidine or having allergy to amide type local anaesthetics were excluded.

One hundred patients were included in the study and randomly divided into two equal groups using random numbers table. The study was conducted after approval from the Hospital Ethics Committee and all data was collected after the informed consent of patients. Group A received 1.5% lignocaine plain only and in group B 8 mg dexamethasone was added in 1.5% lignocaine. Neither epinephrine nor bicarbonate was added to mixtures. All local anesthetic solutions and adjuvant drugs were prepared by an anesthesiologist not involved in the performance of block, patient care, or data collection to control the bias.

On arrival to the operating room, standard monitoring was established (pulse oximetry, electrocardiography, and noninvasive arterial blood pressure monitoring) and oxygen was delivered via a Venturi facemask at a rate of 3 L/min. After insertion of an 18 gauge IV catheter in a peripheral vein in the contra- lateral arm and administration of 1 to 2 mg IV midazolam, axillary block was performed with the patient in the supine position and the upper arm abducted 90° and the elbow flexed at 110°. A nerve stimulator (Polymedic®) with a 24-gauge 7 cm sprotte needle was used for precise localization of each nerve. The stimulation frequency was set at 3 Hz, the duration of stimulation at 0.1 ms, and the intensity of the stimulating current was initially set to deliver 3 mA and then gradually decreased. The position of the needle was considered to be acceptable when an output current <0.5 mA still elicited a slight distal motor response in each of the nerve distributions (thumb opposition for median, thumb abduction for radial, thumb adduction or ulnar deviation of the hand for ulnar, and flexion of forearm on the arm for musculocutaneous nerves).

Increments of anesthetic mixture (10 ml/nerve in total) were injected through a stationary needle after identifying the stimulus response to each of the four nerves.

In case of blockade failure in any of the nerve distributions, the patients were excluded from the study. Sensory blockade of each nerve was assessed by pinprick and compared with the same stimulation on the contra-lateral hand.

Sensory blockade of each nerve was rated by the patient on a verbal analog scale from 100% (normal sensations) to 0% (no sensation). The onset time of the sensory blockade was defined as the time between the end of last injection and the total abolition of the pinprick response. The duration of sensory block was considered as the time interval between the administration of the local anesthetic and the first postoperative pain. The patients and the anesthesiologist who evaluated the onset and duration of sensory blockades were blinded as to the mixture used.

Data was entered in SPSS version 10.0 for statistical analysis. For quantitative variables, mean and standard deviation (SD) were calculated for description and independent samples' t-test was applied for comparison. For qualitative variables, frequency and percentage were calculated for description and chi-square test was applied for comparison. A *p*-value < 0.05 was considered as significant.

RESULTS

The mean age of patients in group A was 41.18 ± 13.34 years while in group B it was 40.42 ± 12.50 years. In group A, male to female ratio was 3.5:1 and in Group B, male to female ratio was 7.3:1. In Group A, 29 (58%) patients were of ASA I while in group B, 25 (50%) patients were of ASA I. Both the groups were comparable with respect to age (p = 0.769), gender (p = 0.813) and ASA status (p = 0.422). (Table-1)

The onset of action and the duration of nerve block showed a significant difference in the two groups. Group A showed the onset of action of 21.64 ± 2.30 min and in group B it was 15.42 ± 1.44 min (p <0.001). (Fig-1). Average duration of nerve block in group A was 115.08 ± 10.92 min while in group B it was 265.42 ± 16.56 min (p <0.001). Table-2 shows the frequency of duration or sensory block between two groups. In group A, all (100%) patients had the duration between 89-130 minutes, while in group B majority (64%) had duration > 256 minutes. This showed that group B had a more prolonged duration of brachial plexus sensory blockade as compared to group A.

DISCUSSION

The addition of corticosteroid to local anesthetic prolonged duration of blockade of the peripheral nerves. In this regard different studies were conducted to increase the duration of sensory and motor blockade and decreasing the onset of analgesia.

In a prospective study conducted on sixty adult patients undergoing various orthopedic surgeries on forearm and around the elbow under supraclavicular brachial plexus block by Maruf et al[15] have concluded that addition of dexamethasone as an adjuvant to 2% lignocaine or 0.5% bupivacaine results in significantly early onset and markedly prolonged duration of analgesia without any unwanted effects.

Parrington and colleagues[16] in their study added 8 mg of dexamethasone to 30 ml 1.5% mepivacaine during supraclavicular brachial plexus blockade. The dexamethasone group showed a longer duration of analgesia: 332 (225-448 min) vs. 228 (207-263 min) min in the control group, whereas the onset time of sensory and motor blockade were similar in both groups.

In our study, in group A onset of action was 21.64 minutes and duration of axillary brachial plexus block was 115.08 minutes and in group B onset was earlier i.e., 15.42 minutes and duration of sensory blocked was prolonged i.e., 265.42 minutes.

Our study results are comparable to the study by Movafegh et al[17] who, in a randomized double-blind, study evaluated the effects of dexamethasone added to lignocaine on the onset and duration of axillary brachial plexus block in sixty patients scheduled for elective hand and forearm surgery. However, in their study the onset times of sensory and motor block were similar in the two groups but the duration of sensory (242 ± 76 versus 98 ± 33 min) blockade was significantly longer in the dexamethasone than in the control group (p< 0.01).

In another study, by Adnan et al incorporation of dexamethasone into bupivacaine microspheres significantly prolonged intercostal nerve block in sheep[18]. Whereas another study suggested that methylprednisolone can increase the duration of sensory and motor block when added to mepivacaine and bupivacaine[19].

Hamid et al[20] found that butorphanol, a morphinan-type synthetic opioid analgesic exhibiting partial agonist at the μ opioid receptors and agonist activity at κ opioid receptors when added to 1% lignocaine can increase the duration of sensory blockade significantly. The mechanism of the analgesia induced by corticosteroids is not fully understood. This effect is suspected to be mediated by their anti inflammatory or immune-suppressive effects[21,22]. The use of corticosteroids as an adjuvant to local anesthetic for peripheral nerve blocks rarely has been described, and its mechanism of action is not clearly understood. Corticosteroids cause skin vasoconstriction on topical application. The vasoconstriction effects of topical steroids are mediated by occupancy of classical glucocorticoid receptors rather than by nonspecific pharmacological mechanisms[23,24]. According to the traditional theory of steroid action, steroids bind to intracellular receptors and modulate nuclear transcription. In our study, dexamethasone produced a relatively rapid effect which cannot be explained by the above mechanism[25]. Therefore, vasoconstriction, the presumed mechanism of action for epinephrine's adjunctive effect on local anesthetics, is probably not responsible for block prolongation by dexamethasone. Corticosteroids may have a local effect on the nerve; the dexamethasone effect may be related to this action[26].

One possibility is that prolongation of local anesthetic block occurs because of systemic effects of dexamethasone. Some authors believe that analgesic properties of corticosteroids are the result of their systemic effects[27,28]. To elucidate the mechanism of action of dexamethasone and to answer the question of whether these results

Table-1: Comparison of gender and ASA status between both the groups.

Variables	Group A (n=50)	Group B (n = 50)	*p*-value
Gender			
Male	39 (78%)	44 (88%)	0.183
Female	11 (22%)	6 (12%)	
ASA Status			
ASAI	29 (58%)	25 (50%)	0.422
ASAII	21 (42%)	25 (50%)	

Table-2: Comparison of duration of action between the groups.

Duration of Action	Group A (n=50)	Group B (n = 50)
< 130	50 (100%)	(0%)
131- 172	0 (0%)	0 (0%)
172 – 214	0 (0%)	0 (0%)
215 - 256	0 (0%)	18 (36%)
> 256	0 (0%)	32 (64%)

$p < 0.001$

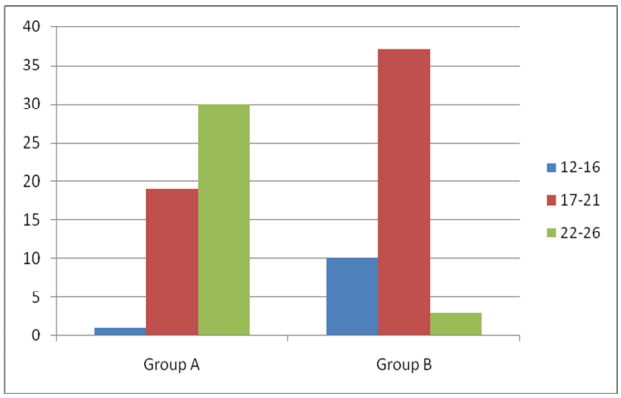

Figure-1: Comparison of time of onset between the groups.

were attributable to a local or systemic effect, well designed further studies are warranted.

Adding a steroid to local anesthetic solution may not be indicated for all patients. For example, diabetic patients may experience hyperglycemia and patients with a continuing infectious process may be detrimentally affected by the anti inflammatory effects of steroids. The use of dexamethasone to increase the duration of action of local anesthetics is not an indication of this drug. This study led us to hypothesize that it may be useful in situations in which epinephrine must be used with caution (e.g., hypertension, ischemic heart disease).

CONCLUSION

We conclude that, the addition of dexamethasone 8 ml to 1.5% lignocaine (38 ml) solution in axillary brachial plexus block prolongs the duration of sensory and motor blockade. However, further well designed studies are needed to evaluate the optimal dose and mechanism of action of dexamethasone to be used to prolong the brachial plexus block duration.

Conflict of Interest

This study has no conflict of interest to declare by any author.

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