A RANDOMISED DOUBLE BLIND PROSPECTIVE STUDY TO COMpare Clonidine and Dexmedetomidine As an ADJuvant IN SUPRA-CLAVICULAR BRACHIAL PLEXUS BLOCK

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ABSTRACT

The practice of Brachial Plexus Block has made the surgical procedures of upper limbs as a safe, low cost and effective form of anaesthetic procedure. This study was designed to compare the relative efficacy and efficiency of use of α₂ agonist drugs (Clonidine and Dexmedetomidine) as an adjuvant of choice. 60 ASA Grade 1 & 2 patients planned for elective upper limb surgery under Brachial Plexus Block were divided into two equal groups in a randomised double blinded fashion. Group I patients received Inj. Bupivacaine 0.25% 38 ml plus Inj. Clonidine 1μg/kg and Group II patients received Inj. Bupivacaine 0.25% 38 ml plus Inj. Dexmedetomidine 1μg/kg. Onset & recovery time of Sensory block and motor block and Duration of Analgesia was studied in both groups. It was found that dexmedetomidine is better and more effective as an adjuvant to bupivacaine in supraclavicular brachial plexus block.

Keywords: Clonidine, Dexmedetomidine, Adjuvant, Supra-Clavicular Brachial Plexus Block

INTRODUCTION

Modern era can be called as the era of regional and demanding anesthesia. Both patients and the surgeons are now more comfortable with this type of anesthesia. Brachial Plexus Block (Supraclavicular approach) through Winnie’s method is a popular anaesthetic procedure for surgeries of upper limbs. The Brachial Plexus is blocked at its most compact site (middle of Brachial Plexus) and it results in a homogenous spread of anaesthetic drug and hence early and complete block. With the advancement in the fields of surgery and orthopaedics, the complexity as well as duration of surgery has increased. Therefore we need an adjuvant to local anesthetics to increase total duration of block. Various opioids and non-opioids agents have been studied as adjuvants to Brachial Plexus blockade. Clonidine and Dexmedetomidine have shown greater affinity as an α₂ agonist. The Aim of this study is to compare the relative efficacy of Clonidine versus Dexmedetomidine (mixed with Bupivacaine) for intra-operative/ post-operative analgesic efficiency and safety.

MATERIALS AND METHODS

This study was conducted at IIMS&R, Lucknow from Sept’2013 to Dec’2013 after getting approval from Ethical Committee. Written informed consent was taken from all the patients in this study. The study group includes ASA grade I and II patients of either sex, between age group of 18 years to 60 years posted for surgery of the upper limb. This study excluded patients with neurological deficits, seizure disorder, bleeding disorder, pneumothorax and pregnant patients.

In the pre-operative room, an intra-venous line is secured with 18 G cannula on the normal arm. Baseline E.C.G [Electrocardiogram], NIBP [Noninvasive blood pressure] and SpO₂ [Oxygen saturation] recorded. The patients were premedicated with Inj. Ranitidine 1amp i.v. stat and Inj. Ondansetron 1amp i.v. stat, 30minutes before Surgery. The Anaesthetist performing the procedure was blinded to the study group and patients were selected by random chit selection method.

These 60 patients were randomly divided into 2 groups of 30 each- Group I: Inj. Bupivacaaine 0.25% 38 ml plus Inj. Clonidine 1μg/kg (2ml).

Group II: Inj. Bupivacaaine 0.25% 38 ml plus Inj. Dexmedetomidine 1μg/kg (2ml).

Supra-clavicular Brachial Plexus block was performed by using subclavian artery as a landmark, with 22 G, 38mm short bevel needle. Paresthesia was elicited and 40ml of anaesthetic solution was given after...
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bloodless aspiration. The onset of sensory block was assessed with cold Alcohol swabs. Assessment of motor block done by Modified Bromage Scale (Eledjam et al., 1991)

0- Normal motor function of flexion and extension of elbow, wrist and fingers.
1- Reduced motor strength with ability to move fingers.
2- Complete motor blockade with inability to move fingers.

Duration of sensory and motor block was assessed every hour till the recovery of sensation by an independent anaethesiologist blinded to the group. Assessment of postoperative pain was done by VAS (Visual Analogue Scale). VAS Score 0-no pain to 10-worst pain. Inj. Diclofenac Sodium was given intramuscularly in the dose of 1–5 mg/kg as rescue analgesic at VAS score of ≥ 5.

During the procedure the vital parameters like pulse, BP, SPO2 and ECG were monitored every 5 min. for first 30 min. and then every 10 min. till procedure was completed. Post operative monitoring was done every 30 min. The duration of analgesia was calculated from onset of block to the first complaint of pain. The incidence of side effects such as bradycardia, hypotension, sedation noted and managed accordingly. Blood Pressure of < 80 mm of Hg were recorded and managed with Inj. Ephedrine in aliquots of 6 mg. For Heart Rate of < 40/per minute injection atropine [0.6mg] was given.

Data was expressed in mean ± SD and p-value of <0.05 was considered statistically significant.

RESULTS

The Chi-square test was used to compare male/female ratio between the groups. The Unpaired t-test was used to compare the continuous variables between the groups. The p-value of < 0.05 was considered as significant. All the analysis was carried by using SPSS 16.0 version (Chicago, Inc. USA).

Demographic profiles of the studied groups are shown in Table 1. Average age in group I was 33.73±12.09 and in group II 36.6±25.4, the p-value (0.57) being insignificant. Weight in group I 58.4±4.3 and group II 55.2±8.11, p-value (0.06) being insignificant and gender group I male22/female8 and group II male19/female11, p-value (0.40) being insignificant [n=30]

Table 2 shows the Onset time and Duration of Sensory and Motor Blocks in both groups. The onset of the sensory block was faster and the duration too was longer (statistically significant 0.001 p-value) in group II as compared to group I. The onset of motor block was slower in group II as compared but duration of block was significantly more as compared to group I (p-value 0.0001)

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<tr>
<th>Table 1: Demographic Profile</th>
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<tr>
<td>Group I (n=30)</td>
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<td>Age (years)</td>
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<tr>
<td>Weight (Kg)</td>
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<td>Gender (Male:Female)</td>
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Note: N.S.- Not Significant; S- Significant.

<table>
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<tr>
<th>Table 2: Onset time and duration of Sensory and Motor block</th>
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<tr>
<td>Group I (n=30)</td>
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<tr>
<td>Onset Time (mins.) (Sensory Block)</td>
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<tr>
<td>Duration (mins.) (Sensory Block)</td>
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<tr>
<td>Onset Time (mins.) (Motor Block)</td>
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<td>Duration (mins.) (Motor Block)</td>
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</table>

Note: N.S.- Not Significant; S- Significant.

Table 3 depicts the total duration of analgesia and it was significantly more in group II (p-value 0.0001)

Intra-operative and post-operative vital monitoring did not show any significant variations in both groups and vitals remained stable except one case of bradycardia and three cases of hypotension in Group II.
DISSCUSSION

Many investigators have studied the role of clonidine as an adjuvant to local anesthetics in brachial plexus block (Eledjam et al., 1991; Murphy et al., 2000; Gaumann et al., 1992).

In this randomized, double-blinded study, we compared dexmedetomidine and clonidine (α2-agonist) as an adjuvant to Bupivacaine in supraclavicular brachial plexus block. The results of our study showed that addition of dexmedetomidine to bupivacaine provided significantly longer duration of sensory and motor block.

Although mechanism of action of clonidine on peripheral nerves remains unclear. Dalle et al., (2001) proposed that clonidine has some direct action on peripheral nerves. They opined that clonidine, by enhancing activity-dependent hyperpolarisation generated by the Na+/K+ pump during repetitive stimulation, increases the threshold for initiating the action potential causing slowing or blockage of conduction. The enhancing effect of low-dose clonidine on lidocaine-induced inhibition of action potential of C-fibers and Aδ fibers (Gaumann et al., 1992; Butterworth and Strichartz, 1993) together with synergistic mechanism of action with local anesthetics (Eledjam et al., 1991) may be the possible explanation to the direct peripheral action.

Another worker Kosugi et al., (2010) examined the effects of various adrenoceptor agonists like dexmedetomidine, tetracaine, oxymetazoline and clonidine, and an α2-adrenoceptor antagonist (Atipamezole) on compound action potential (CAP) recorded from frog sciatic nerve, and found that CAPs were inhibited by α2-adrenoceptor agents so that they are able to block nerve conduction.

As both dexmedetomidine and clonidine belong to same group i.e. α2 agonist, it is quiet possible that they both execute their analgesic effect via similar mechanism.

Although as reported by Kalso et al., (1991) affinity of dexmedetomidine to α2 adrenoceptor agonists is 10 times as compared to clonidine when dexmedetomidine is added to lidocaine for intravenous regional anaesthesia, it has been studied that it improves quality of anaesthesia and intraoperative, postoperative analgesia without causing side effects (Coskuner et al., 2007; Memis et al., 2004; Jaakola et al., 1991).

Clonidine has been used in doses between 0.5 to 1.5µg/kg in brachial block (Elliott et al., 1997; Singelyn et al., 1996). In our study we deliberately selected dose of 1µg/kg as no added benefits were found with doses exceeding 1.5µg/kg. Clonidine increases duration of sensory and motor block of lignocaine by evoked inhibition of C-fiber action potential.

We used same dose of dexmedetomidine i.e. 1mcg/kg, as clonidine so that both drugs can be compared.

Results similar to our study were reported by Esmaglou et al., (2010) who added dexmedetomidine tolevobupivacaine for axillary brachial plexus block and showed that it shortens the onset time of both sensory and motor block, prolongs the duration of block and the duration of post-operative analgesia.

Memis et al., (2004) in their study showed that addition of dexmedetomidine to lignocaine for intravenous regional anaesthesia improves both the quality of anaesthesia as well as intraoperative and postoperative analgesia.

**Conclusion**

Dexmedetomidine is more effective than clonidine when added to bupivacaine in supraclavicular brachial plexus block as it reduces onset of block and causes greater prolongation of both sensory and motor block.
REFERENCES


