Prospective Study of the Efficacy of Clonidine Added to Ropivacaine as Compared with Ropivacaine Alone in Supraclavicular Brachial Plexus Block.

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ABSTRACT

Background: In recent times, the use of ropivacaine for supraclavicular brachial plexus block is increased. The effect of clonidine as adjuvant is extensively studied. Aim: To evaluate the effects of clonidine on nerve blockade during brachial plexus block with ropivacaine.

Methods: Forty patients were included in our study and they were randomly divided into two groups, Group A and B. Group A received 30 ml of 0.5% of ropivacaine with 0.5 ml normal saline, while Group B received same amount of ropivacaine with 0.5 ml (equivalent to 75 µg) of clonidine for supraclavicular brachial plexus block.

Results: There was a significant increase in duration of motor and sensory block and analgesia in Group B when compared to Group A patients (P < 0.0001). There was no significant difference in onset time in either group.

Conclusion: The addition of clonidine to ropivacaine for brachial plexus block prolongs motor and sensory block and analgesia without significant side effects.

Keywords: Anaesthesia, Brachial plexus, Clonidine, Ropivacaine.

INTRODUCTION

Anaesthetists prefer supraclavicular brachial plexus block to anaesthetise entire upper extremity consistently. Studies have revealed it to be a safe and valuable method for upper limb surgery under regional anaesthesia and also showed that simultaneous sympathetic blockade decreases post-operative pain, vasospasm and oedema.[1,2] Ropivacaine is an aminesio amide local anaesthetic prepared as “S” enantiomer. When compared with bupivacaine, ropivacaine is less lipophilic, less cardio toxic, less arrhythmogenic, less toxic to central nervous system (CNS) than bupivacaine, and it also has intrinsic vasoconstrictor property and less likely to penetrate large myelinated motor fibres, resulting in a relatively reduced motor blockade.[1,3]

In order to increase the efficacy of brachial plexus block, many adjuncts have been studied, which included opioids, hyaluronidase, midazolam, bicarbonate, neostigminestse, and α-2 agonists. Clonidine, an imidazoline with selective partial agonist activity at α-2 adrenergic receptors has been extensively studied. It has been shown that clonidine when combined with a local anaesthetic, extended the duration of nerve block. This might be due to centrally mediated analgesia, α-2 adrenoreceptor-mediated vasoconstrictive effect, attenuation of the inflammatory response and direct action on peripheral nerve.[3-5]

The aim of the present study is to evaluate the effects of clonidine used along with ropivacaine during brachial plexus block, with quality of sensory and motor blockade, duration of post-operative analgesia and intra and post-operative complications as parameters.

MATERIALS AND METHODS

We carried our study in 40 patients from December 2015 to November 2016, after obtaining institutional
ethical committee approval. Consent was obtained from all the patients. Initially 52 patients were enrolled in the study. 12 patients were excluded as they did not meet the inclusion criteria. 40 patients were divided randomly into two groups of twenty each. We followed the methodology used by Ali et al (2014). Preanaesthetic checkup and appropriate investigations were performed. Before starting the procedure, linear visual analogue scale (VAS) of 0-10 cm. was described to the patient for assessing pain (0 stands for no pain and 10 for worst pain imaginable).

Group A patients (n=20); Received ropivacaine 0.50% (30 ml) and placebo (0.5 ml NS) and
Group B patients (n=20); Received ropivacaine 0.50% (30 ml) and clonidine 75 µg (0.5 ml).

Inclusion criteria
1. Patients undergoing upper limb surgeries under brachial plexus block,
2. Patients above 18 years of age,
3. Patients without any history of brachial plexus injury,
4. Patients not allergic to the study drugs.

Exclusion criteria
1. Patients having chronic pain and on analgesic medications,
2. Patients with a history of coagulation disorders,
3. Patients with a history of brachial plexus injury,
4. Patients allergic to the study drugs,
5. Patients taking other medications with α-adrenergic blocking effect,
6. Patients having hepatic or renal insufficiency, systemic infection or infection at the site of injection and
7. Patients with bilateral upper limb fractures and previous shoulder surgery.

Technique
An experienced anaesthesiologist gave the brachial plexus block through the supraclavicular approach to all the subjects. Following negative aspiration, 30 ml of solution containing local anaesthetic combined with placebo or clonidine was injected.

Sensory block
Sensory block was assessed every three minutes for thirty minutes on a 3 point scale for pain using pinprick with 25 gauge needle.
1= sharp sensation
2= blunt sensation
3= no sensation

Motor block
Motor block was assessed every five minutes for thirty minutes by modified Bromage Scale.
3= extension of elbow against gravity
2= flexion of wrist against gravity
1= finger movement

Onset of sensory block was defined as time from injection till disappearance of pain by pinprick test (pinprick=3).
Onset of motor block was defined as time between injection and motor paralysis distal to injection site (modified Bromage Scale=0).
Readiness for surgery was defined as complete sensory and motor block in surgical territory (pinprick test=3 and modified Bromage scale =0).
Duration of sensory block was defined as the duration from onset of sensory block till complete regression of sensory block (pinprick test 3 to 1).
Duration of motor block was defined as the duration from onset of motor block till the complete regression of motor block (modified Bromage Scale 0 to 3).

At the end of surgery, patient was transferred to the post anesthesia care unit for further observation and management.

Post-operative observations
1. Patient was assessed every fifteen minute till the complete regression of sensory block.
2. Patient was assessed every fifteen minute till the complete regression of motor block
3. Patient was assessed every fifteen minute till fully awake (sedation score =1).

The data was analysed by SPSS for windows (version 17) statistical package (SPSS Inc., Chicago, IL). The data were expressed as mean ± standard deviation (SD).

Unpaired t-test was applied for demoFigureic data, onset and duration of sensory and motor blockade and duration of analgesia. Fisher exact test was applied for assessment of quality of block. P value was considered significant if < 0.05.

RESULTS

The demoFigureic profiles in both the groups were comparable [Table 1 & Figure 1].

<table>
<thead>
<tr>
<th>Character</th>
<th>Group 1</th>
<th>Group 2</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Age (Years)</td>
<td>42±15</td>
<td>39±14</td>
<td>0.517</td>
</tr>
<tr>
<td>Mean Weight (KG)</td>
<td>60.8±9.86</td>
<td>64.05±6.04</td>
<td>0.216</td>
</tr>
</tbody>
</table>

Figure 1: DemoFigureic profile of the patients in the two groups.
The mean time of onset of sensory block in Group A and Group B was not statistically significant, whereas the mean duration of sensory block in Group A and Group B was statistically significant (Table 2). The mean time of onset of motor block in Group A and Group B was not statistically significant, whereas the mean duration of motor block in Group A and Group B was statistically significant [Table 2 & Figure 2 & 3].

Table 2: Onset and Duration of Sensory and Motor Block In The Two Groups.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group A</th>
<th>Group B</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of sensory block</td>
<td>7.55±3.14</td>
<td>9.2±3.03</td>
<td>0.099</td>
</tr>
<tr>
<td>Duration of sensory block</td>
<td>495±92</td>
<td>714±101</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Onset of motor block</td>
<td>12±2.96</td>
<td>14.09±3.9</td>
<td>0.064</td>
</tr>
<tr>
<td>Duration of motor block</td>
<td>441.50±78</td>
<td>639±86</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Figure 2: Mean Onset of Sensory and Motor blocks.

Figure 3: Mean duration of Sensory and Motor blocks.

The mean pain score of patients in both the groups at 60 minutes post-operatively was zero. Whereas, at 2 hour post-operatively, mean pain score in Group A and B were 1 and 0 (P = 0.1544). 8 hour post-operatively it was 4.2 ± 2.3 and 1.6 ± 1.1 (P = 0.0001).

Complication in the form of nausea and vomiting was observed in Group A and sedation in Group B and the observed complications were statistically insignificant [Table 3].

Table 3: Complications observed in the two groups.

<table>
<thead>
<tr>
<th>Complication</th>
<th>Group A</th>
<th>Group B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of Patients</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Percentage of Patients</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>Nausea/vomiting (Intraoperativ e)</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Sedation (postoperative)</td>
<td>0</td>
<td>10</td>
</tr>
</tbody>
</table>

To find out the basic underlying mechanism for the beneficiary effects of clonidine several studies have been carried out. Gabriel et al have shown that clonidine increases the duration and intensity of pain relief in central neuraxial blocks and other regional blocks.[11] Romero-Sandoval suggested that the mechanism of action of clonidine is by decreasing the systemic and local inflammatory stress response.[12] Chakraboty et al showed that perineural administration of clonidine is better than subcutaneous or i.m. injections, indicating that the local anaesthetic-enhancing effect of clonidine is probably mediated at the neuron.[13] According to Gaumann et al complex interaction between clonidine and axonal ion channels or receptors brings about its beneficial effects.[14] Butterworth et al were of opinion that clonidine enhances the sodium channel blockade action of local anaesthetics by opening up the potassium channels resulting in membrane hyperpolarisation and by α 2-adrenocceptor-mediated local release of enkephalin-like substances.[15] The result in our study is in agreement with other studies which showed that sensory block lasts longer than the motor block. According to them large fibres require a higher concentration of local anaesthetic than small fibres. The minimal effective concentration of local anaesthetic for large (motor) fibres is greater than for small (sensory) fibres.[6,7,16] We found that the combination of ropivacaine with clonidine showed a significant difference in the pain scores. This is in accordance with Ali et al.[1]
Two patients in Group B were sedated in the post-operative period as has been observed in other studies but the difference being statistically insignificant.\textsuperscript{[1,17]}

Murphy et al concluded that clonidine in doses up to 150 μg increased the duration of postoperative analgesia with minimal adverse effects.\textsuperscript{[18]} Whereas McCartney et al concluded that clonidine was beneficial only when added to intermediate-acting local anaesthetics.\textsuperscript{[19]} Sidharth et al found that there was a significant decrease in onset and increase in duration of sensory and motor blockade in the clonidine group in comparison to saline group.\textsuperscript{[3]} However further studies with larger sample and more parameters should be carried out in future to determine the exact role of clonidine as an adjuvant with ropivacaine for supraclavicular block.

**CONCLUSION**

We found that clonidine as an adjunct with ropivacaine for supraclavicular block anaesthesia as ropivacaine produces good analgesia and motor blockade in supraclavicular brachial plexus block and increases the effect of analgesia and motor blockade considerably.

**REFERENCES**


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