Dexmedetomidine as an adjuvant to ropivacaine in supraclavicular brachial plexus block-A randomized, double blind, prospective study

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Abstract: Different adjuvants are used with local anesthetics to improve quality of peripheral nerve blocks. We evaluated the effect of addition of dexmedetomidine with ropivacaine in supraclavicular brachial plexus block with respect to onset of sensory and motor blockade and duration of blockade and analgesia. Sixty patients of ASA grade I and II, aged 18-60 yrs of either sex scheduled for upper limb surgeries under supraclavicular brachial plexus block were randomly divided into 2 equal groups. Patients in group R (n = 30), received 30 ml of 0.5% ropivacaine with 1ml normal saline (control) and in group RD (n = 30), received 30 ml of 0.5% ropivacaine with 1 ml (100µg) dexmedetomidine. The onset and duration of sensory and motor block, duration of analgesia were analyzed in both the groups. Statistical analysis done with Medcalc software and p value<0.05 taken as significant. The mean time of onset of sensory blockade were 5.5±0.94 min and 3.1±0.80 min respectively in group R and RD. Whereas time of onset of motor blockade were 12.27±1.84 min and 6.47±0.94 min respectively in group R and RD. The durations of sensory and motor block were 459.33±19.38 and 369 ± 18.45 min respectively in group R and RD. The duration of analgesia was 487.33± 16.01 min in group R compared to 719.33 ± 11.26 min in group RD (p<0.001). Statistically significant difference was present in respect of onset and duration of sensory and motor blockade and duration of analgesia between 2 groups. We conclude that in supraclavicular brachial plexus block addition of dexmedetomidine in ropivacaine shorten the onset of sensory and motor block and prolongs the durations of sensory and motor block and also increase duration of analgesia.

Keywords: Dexmedetomidine, Supraclavicular brachial plexus block, Ropivacaine

INTRODUCTION

Supraclavicular brachial plexus block provide an ideal operative condition with adequate muscle relaxation, stable intraoperative hemodynamics and post-operative analgesia for upper extremities surgeries. It is carried out at the level of distal trunks of brachial plexus. Various local anaesthetic agents of short, intermediate and longer duration of action have been used in brachial plexus block for upper limb surgeries and postoperative analgesia. Most commonly used local anaesthetic agents were lignocaine and bupivacaine. Lignocaine has faster onset of action but short duration of analgesia and bupivacaine is associated with higher incidence of cardiovascular toxicity. Different additives have been used to prolong the duration, decreasing the dose and side effects of local anesthetic agent. Lots of adjuvants like morphine [1], pethidine [2], neostigmine [3], midazolam [4], dexamethasone, etc has been used in combination with lignocaine and bupivacaine previously, but the results are either inconclusive or associated with side effects [5-7].

A newer long acting local anaesthetic ropivacaine hydrochloride, a chemical congener of bupivacaine is used for providing brachial plexus block. It is a pure S-enantiomer, provides greater local anaesthetic potency and it is less toxic than ropivacaine but more toxic than lidocaine [8, 9]. It is less likely to penetrate large myelinated motor fibers, resulting in a relatively reduced motor blockade and has greater degree of motor sensory differentiation.

Dexmedetomidine hydrochloride, a highly selective α2 agonist is 8 times more selective α2 adrenoreceptor agonist as compare to clonidine. It is found to be safe and effective in various neuraxial and regional
anaesthetic technique in human [10, 11]. It has been shown to have both sedative and analgesic effects [12, 13]. This study was planned to evaluate the effect of addition of dexmedetomidine hydrochloride to onset and duration of anaesthesia, analgesia and motor blockade following supraclavicular brachial plexus block with ropivacaine hydrochloride.

MATERIAL AND METHODS

After Institutional Ethics Committee’s approval, this randomized, double blind study was conducted on sixty patients scheduled for upper limb surgeries.

Selection criteria:
- 60 patients of ASA grade I and II.
- Male & non-pregnant females of age group 18-60 yrs without any renal, hepatic, metabolic and neuromuscular disease.

Exclusion Criteria
- Known history of allergy or sensitivity or any other reaction to local anaesthetic of amide type.
- Treatment with α adrenergic antagonist, history of arrhythmias and labile hypertension.
- Progressive neurological disorder, kidney or liver dysfunction.
- History of bleeding disorder.
- Poor compliance with the study procedure.
- Preanaesthetic checkup of these patients was done with complete history, general examination, and systemic examination.
- Routine investigations like complete blood count, blood sugar, blood urea, serum creatinine, chest X-ray and ECG were done.

After taking written informed consent selected patients were subsequently randomized by slip in a box technique into 2 groups of 30 each.
- Group R (control) (n=30) : 30 ml of 0.5% ropivacaine with 1ml normal saline
- Group RD (n=30): 30 ml of 0.5% ropivacaine with 100 mcg dexmedetomidine (1ml).

After securing an IV line with 18 G cannula, RL solution was started. Various monitoring devices like NIBP, Pulseoxymeter, 3 lead ECG were connected and basal readings were recorded.

Patients were placed in supine position with the head turned to contralateral side and the arms were extended and pulled towards the knee. The midclavicular point and subclavian artery pulsation were identified. Under aseptic precautions & after local infiltration of 2% lidocaine 2 ml, a 22G 1.5 inch needle was introduced 2 cm above the mid-clavicular point directed just lateral to subclavian artery pulsation caudal and medially until paresthesia was elicited. After negative aspiration of blood, the study drug was injected.

After the drug was injected, the following parameters were recorded: Pulse rate (PR), systolic blood pressure (SBP), diastolic blood pressure (DBP), were noted at 0, 5, 10, 15, 20, and at 30 min interval up to 90 min and then every hour till 750 min.

A. Onset of sensory block

Onset of sensory block is defined as the time elapsed between injection of drug and complete loss of sensation as analysed by pinprick.

B. Duration of sensory block

The time interval between complete sensory block till first postoperative pain.

A. Onset of motor block:

Defined as the time elapsed from injection of drug to complete motor block. Measurements were performed using a modification of the Lovett rating scale [14] at every 1 min interval from the time of injection of test drug until the block was established.

- Grade 6: Normal muscular force
- Grade 5: Slightly reduced muscular force
- Grade 4: Pronounced reduction of muscular force
- Grade 3: Slightly impaired mobility
- Grade 2: Pronounced mobility impairment
- Grade 1: Almost complete paralysis
- Grade 0: Complete paralysis

Only patient with complete motor block (grade 0) were included in study.

B. Duration of motor block:

Time elapsed between injections of the drug to complete return of motor power (grade 6).

Postoperative pain was assessed using a visual analogue score scale in which gradations marked as ‘0’ means no pain at all and ‘10’ means unbearable pain. When VAS score reached ≥ 4, inj. diclofenac 75 mg given intramuscularly. The time between end of local anaesthetic given and first analgesic requirement was noted as duration of analgesia.
VAS score rating [15]:

VAS score was recorded every 30 min in the postoperative period till the conclusion of study.

Adverse events such as nausea, vomiting, bradycardia (below 20% of basal value), hypotension (below 20% of basal value) & desaturation were noted. The observations were recorded and statistical analysis was carried out using independent student’s t-test by Medcalc software. p-value <0.05 was taken statistically significant.

RESULTS

The groups were well matched for age, weight & male: female ratio. Both groups had male patients predominantly. The statistical difference was insignificant (p>0.05).

### Table-1: Demographic profile of 2 groups

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group R</th>
<th>±SD</th>
<th>Group RD</th>
<th>±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>34.5</td>
<td>11.39</td>
<td>35.6</td>
<td>14.53</td>
</tr>
<tr>
<td>Weight (kgs)</td>
<td>60.17</td>
<td>6.64</td>
<td>59.1</td>
<td>6.41</td>
</tr>
<tr>
<td>Sex (m:f)</td>
<td>23:7</td>
<td></td>
<td>19:11</td>
<td></td>
</tr>
</tbody>
</table>

### Table-2: Comparison of study parameters between 2 groups

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group R</th>
<th>±SD</th>
<th>Group RD</th>
<th>±SD</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset time of sensory blockade (min)</td>
<td>5.5</td>
<td>0.94</td>
<td>3.1</td>
<td>0.80</td>
<td>0.00</td>
</tr>
<tr>
<td>Duration of Sensory blockade (min)</td>
<td>459.33</td>
<td>19.38</td>
<td>695</td>
<td>11.06</td>
<td>0.00</td>
</tr>
<tr>
<td>Onset time of motor blockade (min)</td>
<td>12.27</td>
<td>1.84</td>
<td>6.47</td>
<td>0.94</td>
<td>0.00</td>
</tr>
<tr>
<td>Duration of motor blockade (min)</td>
<td>369</td>
<td>18.45</td>
<td>657</td>
<td>18.27</td>
<td>0.00</td>
</tr>
<tr>
<td>Duration of Analgesia (min)</td>
<td>487.33</td>
<td>16.01</td>
<td>719.33</td>
<td>11.26</td>
<td>0.00</td>
</tr>
</tbody>
</table>

This table shown that mean onset time of sensory blockade was 5.5±0.94 min in group R, and 3.1±0.80 min in group RD. Duration of sensory blockade was 459.33±19.38 min in group R and 695±11.06 min in group RD. Mean onset time of motor blockade was 12.27±1.84 min in group R & 6.47±0.94 min in group RD. The duration of motor blockade (mean ±SD) was 369 ±18.45 min in group R & 657±18.27 min in group RD. Duration of analgesia was 487.33±16.01 min in Group R and 719.33±11.26 min in group RD. There was highly significant difference between control group and group RD in respect of onset time of sensory and motor blockade (p<0.001). Duration of sensory and motor blockade were significantly prolonged in group RD. Duration of analgesia was also longer in group RD than control group (p<0.001).

Baseline haemodynamic values were comparable in both groups as compared to control group. Heart rate was significantly lower in group RD upto 270 min and blood pressure was lower upto 330 min after that the difference was insignificant (p>0.05). No side effect such as nausea, vomiting, hypotension and bradycardia were seen in control group. Two patients had bradycardia in group RD. Inj. atropine 0.6

mg was given IV as treatment for these patients. No significant difference was found between 2 groups in respect of adverse events (p>0.05).

**DISCUSSION**

Peripheral nerve blocks produce long lasting site specific effective analgesia and anaesthesia. They avoid side effects of anaesthetic drugs used in general anaesthesia. Supraclavicular block is a common nerve block used for upper limb surgeries. Many long acting local anaesthetics have evolved by research techniques. Ropivacaine is a long acting amide which has less cardiotoxic potential. Dexmedetomidine an alpha-2 agonist has sedative, analgesic and perioperative sympatholytic and haemodynamic stability properties. It decreases anaesthetic and analgesic requirements. In our study we evaluated the effect of addition of dexmedetomidine (100mcg) to ropivacaine in supraclavicular block.

Results of our study shown that onset of sensory & motor blockade were rapid with adding dexmedetomidine in ropivacaine. Our observations are in accordance with the findings of Esmaoglu A et al.; [16] who evaluated the effect of dexmedetomidine added to levobupivacaine for brachial plexus block and observed that the onset of sensory & motor blockade was shorter (9.03±1.15 min ,9.50 ±1.04 min respectively ) in dexmedetomidine group than (10.46±1.30 min,11.10±1.24min respectively) in control Group. Similar findings observed by Ammar and Mahmoud [17]. Our findings revealed that duration of sensory& motor blockade prolonged in group RD as compared to group R. This may be because peripheral α2 agonist produces analgesia by reducing the release of norepinephrine leading to α2 receptor independent inhibitory effects on nerve fibers action potentials. Centrally, α2 agonist produces sedation and analgesia by abolition of Substance P release in nociceptive pathway at the level of dorsal horn and activation of α2 receptor in locus ceruleus [18].

Our observations are in accordance with the findings of Swami SS et al.; [19]. They used α2 agonist with local anaesthetic to extend the duration of peripheral nerve blocks. Ammar and Mahmoud [17] observed that adding dexmedetomidine (0.75 mcg/kg) to bupivacaine (0.33 %) significantly prolonged the duration of sensory & motor blockade as compared to bupivacaine alone.

Duration of analgesia as assessed by VAS score was prolonged in group RD as compared to control Group. Our observations are in accordance with the findings of Kathuria S et al.; [20], Esmaoglu A et al.; [16] &Swami SS et al.; [19].Our findings showed that heart rate and blood pressure were significantly lower in RD group. Our observations are in accordance with the findings of Esmaoglu A et al.; [16] & Swami SS et al.; [19]. No complication was found in group R while in group RD 6.66% patients had bradycardia. Similar findings observed by Esmaoglu A et al.; [16].

**CONCLUSION:**

Addition of dexmedetomidine 100 µg to ropivacaine shortens the onset of sensory and motor blockade, prolongs the duration of sensory and motor blockade & increase duration of analgesia. Thus dexmedetomidine is an attractive choice as an adjuvant to ropivaicane for supraclavicular brachial plexus block

**REFERENCES**

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