Comparative study of supraclavicular axillary nerve block with or without clonidine as an adjuvant

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Abstract

Background: Local anesthetics administered as regional nerve blocks are utilized in providing postoperative pain relief in many surgical procedures by blocking signal traffic to the dorsal horn. Certain drugs like opioids, alpha2 adrenergic agonist, sodium bicarbonate, neostigmine, adrenaline, ketamine etc. are used as adjuvant to local anesthetics to lower doses of each agent and enhance analgesic efficacy while reducing the incidence of adverse reactions. Clonidine is a selective alpha 2 adrenergic agonist with some alpha 1 agonist property. In clinical studies, the addition of clonidine to local anesthetic solutions improved peripheral nerve blocks by reducing the onset time, improving the efficacy of the block during surgery and extending postoperative analgesia. In clinical studies, the addition of clonidine to local anesthetic solutions improved peripheral nerve blocks by reducing the onset time, improving the efficacy of the block during surgery and extending postoperative analgesia. Clonidine possibly enhances or amplifies the sodium channel blockade action of local anesthetics by opening up the potassium channels resulting in membrane hyperpolarization, a state in which the cell is unresponsive to excitatory input.

Material and methods: Present study was carried to evaluate efficacy of injection clonidine (150 µg) as adjuvant to supraclavicular brachial plexus block (30 ml lignocaine adrenaline 1.5%, 10 ml bupivacaine 0.5%) in adult patients (ASA Grade I and II). Patients of both groups were assessed in terms of: Onset time of sensory blockade, Onset time of motor blockade, Perioperative hemodynamic status, Duration of post-operative analgesia, Time of 1st rescue analgesia, Adverse effects of drugs if any.
Results: The mean time of onset of sensory and motor block was significantly lower in Group B compared to Group A. Mean duration of motor block and sensory block were significantly longer in Group B than in Group A. Mean time for analgesic requirement for Group B was $11.85 \pm 1.54$ hours and it was significantly longer than that in Group A ($5.62 \pm 0.358$ hours ($p < 0.05$). No incidence of nausea, vomiting, hypotension, tachycardia or bradycardia was observed in any group. One patient in Group B had pulse rate <60/min which was clinically not significant and did not require treatment. No incidence of decline in SPO2 perioperatively.

Conclusion: When clonidine 150 µg is added to local anesthetic solution in supraclavicular brachial plexus block, it provides rapid onset of block, better analgesia, good hemodynamic stability and profound and longer analgesia without any adverse effects. Clonidine is a good adjuvant to local anesthetic agent for brachial plexus block via supraclavicular approach for various upper limb surgeries.

Key words
Clonidine, Supraclavicular brachial plexus block, Lidocaine, Bupivacaine.

Introduction
Acute postoperative pain is the result of a complex physiological reaction to tissue injury. The dorsal horn of the spinal cord is the site of termination of primary afferents and there is complex interaction between such afferent fibers, intrinsic spinal neurons, descending pain modulating fibers and various associated neurotransmitters such as serotonin, nor epinephrine, acetylcholine, adenosine, and glutamate in the dorsal horn. Local anesthetics administered as regional nerve blocks are utilized in providing postoperative pain relief in many surgical procedures by blocking signal traffic to the dorsal horn. Certain drugs like opioids, alpha2 adrenergic agonist, sodium bicarbonate, neostigmine, adrenaline, ketamine etc. are used as adjuvant to local anesthetics to lower doses of each agent and enhance analgesic efficacy while reducing the incidence of adverse reactions [1]. Tramadol and fentanyl had been successfully used as adjuvants to local anesthetics in brachial plexus block [2]. The concurrent injection of alpha adrenergic agonist drugs has been suggested to improve the nerve block characteristic of local anesthetic solutions through either local vasoconstriction and facilitation of C fiber blockade or a spinal action caused by slow retrograde axonal transport or simple diffusion along the nerve. Clonidine is a selective alpha 2 adrenergic agonist with some alpha 1 agonist property [3]. In clinical studies, the addition of clonidine to local anesthetic solutions improved peripheral nerve blocks by reducing the onset time, improving the efficacy of the block during surgery and extending postoperative analgesia. Clonidine possibly enhances or amplifies the sodium channel blockade action of local anesthetics by opening up the potassium channels resulting in membrane hyper polarization, a state in which the cell is unresponsive to excitatory input. The present study is performed to compare the effect of clonidine v/s placebo as adjuvant to lignocaine for brachial plexus block, by supraclavicular approach, for different upper limb surgeries.

Present study was carried to evaluate efficacy of injection clonidine (150 µg) as adjuvant to supraclavicular brachial plexus block (30 ml lignocaine adrenaline 1.5%, 10 ml bupivacaine 0.5%) in adult patients (ASA Grade I and II). Patients of both groups were assessed in terms of: Onset time of sensory blockade, Onset time of motor blockade, Perioperative hemodynamic status, Duration of post-operative analgesia, Time of 1st rescue analgesia, Adverse effects of drugs if any.

Materials and methods
The present study was conducted in 50 patients of ASA I or II status in the age group of 18 – 50
years under brachial plexus block by supraclavicular approach [4] for various upper limb surgeries, emergency or planned, after receiving institutional ethical committee approval. Patients excluded from the study were for whom supraclavicular brachial plexus block or the study medications were contraindicated or those who had a history of significant neurological, psychiatric, neuromuscular, cardiovascular, pulmonary, renal or hepatic disease or alcohol or drug abuse, as well as pregnant or lactating women.

After the establishment of block, surgery was started and time of beginning of surgery was noted. Intravenous fluids were continued intraoperatively at a rate of 2 ml/kg/hour. Intraoperatively, pulse, BP, SPO2 and ECG were monitored every half hourly. Any complication like tachycardia, bradycardia, hypotension, nausea, vomiting, breathlessness, cough, discomfort and sedation were noted. During the procedure, anesthesia was considered satisfactory if patient did not complain of any pain or discomfort. Any patient requiring supplemental anesthesia was excluded from the study. All 50 patients were monitored for anesthesia and analgesia up to 15 hours in the post-operative period. Duration of sensory block (the time elapsed between injection of the drug and return of pinprick sensation) and duration of motor block (time elapsed between injection of the drug to complete return of motor power evaluated by finger and shoulder movement) were recorded. Intensity of postoperative pain was evaluated using VAS (Visual Analogue Scale), Grade 0 (No pain) to 100 (Worst pain). Analgesia was considered satisfactory if the score was 30 or less. If the score was more than 30, analgesia was judged unsatisfactory and rescue analgesic inj. Diclofenac sodium 75 mg iv was administered. Time for first analgesic was noted. Postoperatively, heart rate, blood pressure, respiratory rate, oxygen saturation and VAS were recorded at 0 min, 30 min, 1 hour, 2 hour, 3 hour, 4 hour, 6 hour, 9 hour, 12 hour and 15 hour.

Patients were observed carefully for any complications of supraclavicular block like pneumothorax, local anesthetic toxicity and complications of clonidine like sedation, bradycardia, nausea, vomiting etc. In each patient, a chest x-ray was done 6 hours postoperatively to rule out pneumothorax. Any neurological complication was noted. Both groups were compared for the duration of satisfactory analgesia from the time when the block was performed and the time for first administration of rescue analgesic. Data were presented as mean values and mean + S.D and analyzed using unpaired ‘t’ test with p value <0.05 considered statistically significant.

**Results**

The mean time of onset of sensory and motor block was significantly lower in Group B compared to Group A. (Table – 1)

Mean duration of motor block and sensory block are significantly longer in Group B than in Group A. Mean time for 1st analgesic requirement for Group B is 11.85 ± 1.54 hours and it is significantly longer than that in Group A (5.62 ± 0.358) hours (p <0.05). (Table – 2)

No incidence of nausea, vomiting, hypotension, tachycardia or bradycardia was observed in any group. One patient in Group B had pulse rate <60/min which was clinically not significant and did not require treatment. No incidence of decline in SPO2 perioperatively.

<table>
<thead>
<tr>
<th>Onset of Anesthesia</th>
<th>Group A</th>
<th>Group B</th>
<th>P value</th>
<th>Inference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Sensory (min) Block</td>
<td>12.72 ±1.33</td>
<td>11.32± 0.852</td>
<td>&lt;0.05</td>
<td>S</td>
</tr>
<tr>
<td>Mean Motor (min) Block</td>
<td>6.48±0.82</td>
<td>5.98±0.89</td>
<td>&lt; 0.05</td>
<td>S</td>
</tr>
</tbody>
</table>

**Table – 1: Onset of Anesthesia.**
Table – 2: Duration of analgesia and anesthesia.

<table>
<thead>
<tr>
<th>Time (hours)</th>
<th>Group A</th>
<th>Group B</th>
<th>P value</th>
<th>Inference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean duration of Motor Block</td>
<td>3.68 ± 0.33</td>
<td>7.15 ± 0.53</td>
<td>&lt;0.05</td>
<td>S</td>
</tr>
<tr>
<td>Mean duration of Sensory Block</td>
<td>4.59 ± 0.32</td>
<td>9.61 ± 1.63</td>
<td>&lt;0.05</td>
<td>S</td>
</tr>
<tr>
<td>Mean time of 1st analgesic</td>
<td>5.62 ± 0.358</td>
<td>11.85 ±1.54</td>
<td>&lt;0.05</td>
<td>S</td>
</tr>
</tbody>
</table>

Discussion

The onset of motor block was found to be faster than the onset of sensory block in the both groups. Winnie, et al. [5] observed this also, and attributed this to the somatotrophic arrangement of fibres in a nerve bundle at the level of the trunks in which motor fibers are located more peripherally than sensory fibers. Hence a local anesthetic injected perineurally will begin to block motor fibers before it arrives at the centrally located sensory fibers. Bernard, et al. [6] mentioned in their study that each dose of clonidine 30, 50 and 300 µg used along with lidocaine reduced the onset of block when compared to lidocaine used alone in axillary brachial plexus block. Chakraborty, et al. [7] observed a faster onset of sensory and motor blockade in patients who received 30 µg clonidine with bupivacaine as compared to control in supraclavicular block. Chawda PM, et al. [8] compared the effects of 200 µg epinephrine and 90 µg clonidine as adjuvant to bupivacaine and lignocaine in supraclavicular brachial plexus block on onset of sensory and motor blockade. Their study showed faster onset of anesthesia with clonidine 90 µg as adjuvant. Singh S, et al. [9] in their study observed faster onset of sensory and motor block in patients who received bupivacaine plus 150 µg clonidine as compared to control in supraclavicular block. These studies suggest that addition of clonidine in various doses, to local anesthetics in brachial plexus block hastens the onset of sensory and motor blockade. Eleedjam, et al. [10] and Chawda PM, et al. [8] did comparison of clonidine and epinephrine added to bupivacaine for brachial plexus block and concluded that hemodynamic parameters remained stable in all patients. Singelyn, et al. [11] conducted their study using mepivacaine plus increasing doses of clonidine (0.1, 0.2, 0.3, 0.4, 0.5, 1 and 1.5 µg/kg) and demonstrated stable hemodynamic parameters throughout study period in the clonidine group up to a dose of 0.5 µg/kg. Chakraborty, et al. [7] study also demonstrated no change in hemodynamic parameter with bupivacaine plus clonidine (30 µg) v/s bupivacaine alone. The duration of surgery was comparable in both groups in our study. The mean duration of surgery was 83.8 ± 22.32 min in Group A and 95.2 ± 21.9 min in Group B (P > 0.05). Singh et al and Eleedjam, et al. [10] used clonidine 150 µg with bupivacaine in supraclavicular brachial plexus block and concluded that clonidine produces prolongation of motor and sensory block. Our study goes parallel to the findings of above studies. Singelyn et al. [11] in their study used local anesthetic and increasing doses of clonidine as adjuvant to axillary brachial plexus block and concluded that clonidine in a minimum dose of 0.5 µg/kg prolongs motor and sensory block. Similar observations were made by Buttner, et al. [12] with clonidine in doses of 120 µg and 270 µg. Erlacher, et al. [13], Mjahed, et al. [14], Chakraboty, et al. [7], all observed prolongation of sensory and motor blockade with different doses of clonidine (up to 150 µg) used with local anesthetics in brachial plexus block. El Saied, et al. [15] in their study compared ropivacaine and ropivacaine + clonidine 150 µg for axillary brachial plexus block and observed increase in sensory block from 489 min to 628 min with a mean difference of 138 min and motor block from 552 min to 721 min with mean difference of 170 min. Intensity of postoperative pain was evaluated using VAS. Visual Analogue Scale (VAS, described by Aitkin) is easiest and most commonly used tool for assessment of pain. The duration of postoperative analgesia was
significantly more in Group B. (P < 0.05). Various studies evaluating the effects of addition of clonidine into brachial plexus block have been published; most of them reported prolongation of postoperative analgesia with clonidine; duration of which depends on dose of clonidine, type of local anesthetic used and technique of brachial plexus block performed. Eledjam, et al. [10] in their study demonstrated that clonidine 150 µg with bupivacaine (Group I) produced prolonged analgesia compared to bupivacaine with epinephrine 200 µg (Group II). Duration of analgesia was significantly more (994.2 ± 34.2 min (Group I) v/s 728.3 ± 35.8 (Group II) P < 0.001). Singelyn, et al. [11] in their study concluded that the minimum dose of clonidine required to prolong significantly the duration of analgesia after brachial plexus block with mepivacaine 1% with epinephrine, is 0.1 µg/kg (duration of analgesia 351 ± 12 min v/s 260 ± 40 min (control Group I). Increased duration of analgesia was obtained with increasing dose of clonidine (0.2, 0.3, 0.4, 0.5, 1 µg/kg, 1.5µg/kg) Chawda PM, et al. [8] compared clonidine 90 µg and epinephrine 200 µg with bupivacaine in brachial plexus block and concluded that clonidine is better option for prolongation of analgesia. Chakraboty, et al. [7] in their study used bupivacaine with clonidine v/s bupivacaine alone and demonstrated significant prolongation of postoperative analgesia after supraclavicular block (495.4 ± 38.18 min (clonidine group) v/s. 194.2 ± 28.74 min (control group). Similar observations were made by Singh et al with clonidine (150 µg) + bupivacaine for supraclavicular brachial plexus block. Bernard, et al. [5] in their study used lidocaine plus clonidine in three different doses 30, 50, 300 µg and concluded that clonidine produced a dose dependent prolongation of analgesia after axillary brachial plexus block. Damien Murphy, et al. [16] evaluated 6 studies which used clonidine as adjuvant to local anesthetics in brachial plexus block and demonstrated that 5 of the 6 studies had supportive evidence of analgesic benefits of clonidine in brachial plexus block. In our study no major side effects were noted in Group B intra-operatively except a decrease in heart rate to less than 60/min. in one patient which did not require any treatment. Trivedi, et al. [17], Eledjam, et al. [10], Erlacher, et al. [13], Singh, et al. [9], in their studies demonstrated that clonidine (150 µg) when used for brachial plexus block (axillary or supraclavicular approach) was not associated with any side effects. Chakraboty, et al. [7] and Chawda PM, et al. [8] in their study used bupivacaine with clonidine 30 µg and 90 µg respectively in supraclavicular brachial plexus block and concluded that clonidine at these doses was associated with sedation as the only adverse effect. Buttner, et al. [12] and Bernard, et al. [6] in their study used clonidine 240 µg and 300 µg respectively as adjuvant in axillary brachial plexus block and concluded that at these high doses side effects like hypotension and bradycardia occurs. So most of the studies supported our observation that clonidine at 150 µg was not associated with any major side effects other than sedation.

**Conclusion**

In conclusion, when clonidine 150 µg is added to local anesthetic solution in supraclavicular brachial plexus block, it provides rapid onset of block, better analgesia, good hemodynamic stability and profound and longer analgesia without any adverse effects. In nutshell clonidine is a good adjuvant to local anesthetic agent for brachial plexus block via supraclavicular approach for various upper limb surgeries.

**References**


