

TO COMPARE AND EVALUATE THE EFFECT OF DEXMEDETOMIDINE AS AN ADJUVANT TO LOCAL ANAESTHETIC AGENTS IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK FOR ELECTIVE FOREARM SURGERY

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ABSTRACT

A variety of receptor mediated nociception on peripheral sensory axons and the peripheral administration of appropriate drugs (adjuncts) may have analgesic benefit without the disadvantage of systemic adverse effects and it may also allow reduction in the total dose of local anaesthetic used. Recent studies suggest that α_2 agonists when combined with local anaesthetics extends the duration of regional anaesthesia. Thus, in the present study, we investigated the effects of adding dexmedetomidine 50 μ g to a 30 mL of local anaesthetic solution in supraclavicular brachial plexus block with respect to onset and duration of motor and sensory block and duration of analgesia.

METHODS

Sixty patients scheduled for elective forearm surgery were divided into two equal groups in a randomised double blind fashion. In group C (n=30), 20 mL of 0.5% bupivacaine+10 mL of 2% lignocaine+0.5 mL of normal saline and in group D (n=30) 20 mL of 0.5% bupivacaine+10 mL of 2% lignocaine+50 μ g dexmedetomidine were given for supraclavicular brachial plexus block using peripheral nerve stimulator. Onset and duration of sensory and motor block were assessed along with total duration of analgesia. Demographic and haemodynamic data were subjected to student's t-test and for statistical analysis of onset time and duration of sensory and motor blocks and total duration of analgesia, unpaired t-test was applied. P-value <0.05 was considered as statistically significant and P-value <0.001 as highly significant.

RESULTS

Dexmedetomidine added as an adjuvant to local anaesthetic agents for supraclavicular block shortens onset time and significantly prolongs the duration of sensory and motor blocks and duration of analgesia.

KEYWORDS

Dexmedetomidine, Bupivacaine, Lignocaine, Supraclavicular Block.

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INTRODUCTION

Upper limb surgeries are mostly performed under regional anaesthesia i.e. brachial plexus block. The first percutaneous supraclavicular block was performed in 1911 by German surgeon Diedrich Kulenkampff.¹ Brachial plexus blocks not only provide intraoperative anaesthesia, but also ensures analgesia in the postoperative period without any side effects.

In an attempt to produce ideal adjuvant for the brachial plexus blockade, various agents such as dexamethasone, neostigmine, tramadol,^{2,3} midazolam, opioids (fentanyl⁴ and sufentanyl³) and α_2 agonists (clonidine³ and dexmedetomidine) were added to local anaesthetics to achieve quick, dense, and prolonged block, but the results are either inconclusive or associated with side effects.

Dexmedetomidine is highly selective (eight times more selective than clonidine), specific and potent α_2 adrenergic

agonist⁵ having analgesic,⁶ sedative, antihypertensive, and anaesthetic sparing effects when used in systemic route. Addition of dexmedetomidine to local anaesthetics may also prove to prolong the duration of anaesthesia and analgesia.

The results from various previous studies (Esmoğlu et al⁷ Kenan Kaygusuz et al⁸ Khade, Amit R. et al⁹) regarding addition of dexmedetomidine as an adjuvant to peripheral nerve block are variable.

Observations from above studies inspired us to conduct a study with the aim to evaluate the effect of addition of 50 μ g dexmedetomidine to local anaesthetics in supraclavicular brachial plexus block for forearm orthopaedic surgeries. The characteristics of block, which were observed included the onset of sensory and motor blocks, duration of sensory and motor blocks, and total duration of analgesia.

MATERIAL AND METHODS

After obtaining institutional ethics committee approval and written informed consent, 60 adult patients of either sex, aged between 18 to 60 years, weighing between 50 to 70 kg, belonging to ASA class I and II posted for elective forearm surgeries under supraclavicular brachial plexus block were randomly allocated to two equal groups (n=30 in each group) using computer generated random number list.

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Patients in Group C (Control group) received inj. bupivacaine 0.5% 20 mL+inj. lignocaine 2% 10 mL+inj. normal saline 0.5 mL for the supraclavicular brachial plexus block.

Patients in Group D (Dexmedetomidine group) received inj. bupivacaine 0.5% 20 mL+inj. Lignocaine 2% 10 mL+inj. dexmedetomidine 50 micrograms for the same block.

Patients who refused to take part in the study, presented with any known hypersensitivity to local anaesthetic agents and dexmedetomidine, severe renal, hepatic, or cardiopulmonary abnormality, local skin site infection, neurological, psychiatric, neuromuscular disorder, bleeding disorder, pregnant women, and lactating mother were excluded from study.

Preanaesthetic evaluation was done on the day before surgery and routine examination was carried out assessing general condition of the patient, airway examination of the patient, cardiovascular system, and respiratory system of the patient.

After arrival of patients in operation theatre, intravenous line was secured with an 18 gauge intravenous cannula. The patients were connected to multipara monitor and baseline vitals were recorded. After proper explanation of technique, the patients were placed in the dorsal recumbent position with the head turned away from the site of injection. Under all aseptic precautions, skin infiltration was done with lignocaine 2% at the site of block prior to block placement. All supraclavicular brachial plexus blocks were performed as described by Winnie.¹⁰ using 22G, 50 mm insulated blunt needle [Locoplex (Vygon) needles with extension tubing] and Plexygon nerve stimulator. After an appropriate response was localised with a current <0.5 mA, the drug (according to the group) was injected in 3 mL increments after a negative aspiration test with repeat aspirations every 3 mL. Assessments were carried out initially at 5 min interval thereafter at each minute till the achievement of complete block until 30 minutes. After 30 minutes if the block was considered to be adequate, surgeons were allowed to start the surgery. If the block was considered to be inadequate for surgery, the patients were given general anaesthesia and such patients were not included in the study.

ASSESSMENT

Assessment of sensory block was done by atraumatic pinprick test using 3-point scale: 0=normal sensation, 1=loss of sensation of pinprick (analgesia), and 2=loss of sensation of touch (anaesthesia).

It was done at 5 minutes, thereafter at each minute in the dermatomal areas corresponding to median nerve, radial nerve, ulnar nerve, and musculocutaneous nerve till complete sensory block was achieved. Complete sensory block was considered when there was complete loss of sensation in all the dermatomal areas.

Assessment of motor block was carried out at 5 minutes, thereafter at each minute till complete motor blockade. It was determined by thumb abduction (radial nerve), thumb adduction (ulnar nerve), thumb opposition (median nerve), and flexion of elbow (musculocutaneous nerve) according to the modified Bromage scale on a 3-point scale:

Grade 0: Normal motor function with full flexion and extension of elbow, wrist, and fingers.

Grade 1: Decreased motor strength with ability to move the fingers only.

Grade 2: Complete motor block with inability to move the fingers.

Sedation Score was Assessed by Modified Wilson Sedation Scale,¹¹ which has Scoring from 1 to 4

Score 1: Fully awake and oriented and follows verbal command.

Score 2: Drowsy, eyes closed, but arousable only to commands.

Score 3: Eyes closed, but arousable to mild physical stimulation (Ear lobe tug).

Score 4: Eyes closed and unarousable to mild physical stimulation.

Patient’s heart rate, blood pressure, electrocardiogram, respiratory rate, and SpO2 were monitored and recorded at regular intervals throughout the period of study. Total duration of analgesia was also recorded during the course of study. Patients were monitored for any signs of cardiovascular or central nervous system toxicity (changes in HR/BP/rhythm/signs of CNS stimulation) throughout the study. Any hypersensitivity reaction for the drugs, evidence of pneumothorax, and other adverse events were also monitored. In the postoperative period, the time was noted when the patient first complained of pain at the operative site and hence the total duration of analgesia was calculated.

The results were presented as mean±standard deviation (SD) for parametric data and as percentage for nonparametric data. Demographic and haemodynamic data were subjected to Student’s t-test and for statistical analysis of onset time and duration of sensory and motor blocks and total duration of analgesia, unpaired t-test was applied, and reconfirmed with the Wilcoxon W and Mann-Whitney U tests. P-value <0.05 was considered as statistically significant and P-value <0.001 as highly significant. Any adverse effects were analysed using the chi-square/Fischer’s exact test. The data were analysed by using Microsoft Excel 2010 for construction of graph and SPSS version 14 software for data analysis.

OBSERVATIONS AND RESULTS

Demographic Parameter

Statistically, there was no significant difference in the demographic profile of patients in either group. Both groups were comparable in terms of age, body weight, or male/female (M/F) ratio.

Parameters	Group C (Control)	Group D (Dexmedetomidine)
Age (yrs.) (mean±SD)	37.6±11.87	36.3±12.58
Sex	Male	22(73.3%)
	Female	8(26.7%)
Wt. (kg.)	65±10	66±12

Table 1: Showing Demographic Parameters

Onset Time of Sensory and Motor Block

Onset of sensory block was faster in group D (7.015+/-1.04 min.) as compared to group C (8.0+/-1.02 min.) and the difference was statistically significant (p<0.05) while onset of

motor block was faster in dexmedetomidine group (14.5±1.76 min.) as compared to control group (17±1.44 min.) and the difference was highly significant (p= <0.001).

Parameter	Group C (Control)	Group D (Dex-medetomidine)	P-value	Remark
Onset Time for Sensory Block (Min.)	8.0±1.02	7.015±1.04	<0.05	Significant
Onset Time for Motor Block (Min.)	17±1.44	14.5±1.76	<0.001	Highly Significant

Table 2: Showing Onset Time of Sensory and Motor Block

Duration of Sensory and Motor Block and Duration of Analgesia

Duration of sensory block was longer in dexmedetomidine group (756±42.7 min.) as compared to control group (390±20.6 min.) and the difference was highly significant (p<0.001) and duration of motor block was also found to be longer in dexmedetomidine group (639±35.1 min.) as compared to control group (313±9.04 min.) and this difference was also highly significant (p<0.001). The total duration of analgesia was found to be significantly longer in group D (979.67±33.53 min.) as compared to group C (487.66±15.85 min.). This difference was clinically and statistically significant (<0.001).

Parameter	Group C (Control)	Group D (Dex-medetomidine)	P-value	Remark
Sensory Block (Min.)	390±20.6	756±42.7	<0.001	Highly Significant
Motor Block (Min.)	313±9.04	639±35.1	<0.001	Highly Significant
Total Duration of Analgesia (Min.)	487.66±15.85	979.67±33.53	<0.001	Highly Significant

Table 3: Showing Duration of Sensory and Motor Block and Duration of Analgesia

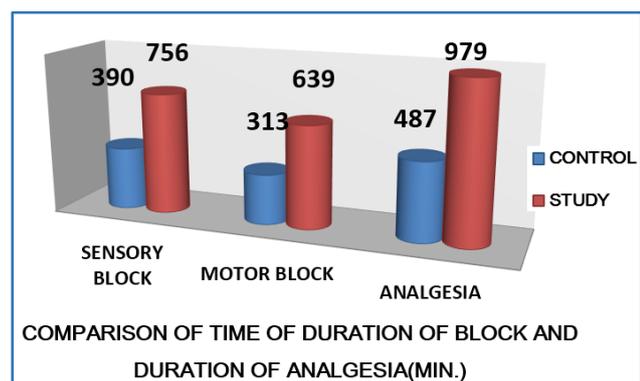


Fig. 1: Showing Comparison of Time of Duration of Block and Duration of Analgesia (Minutes)

Heart Rate

We clinically noticed lower pulse rate in dexmedetomidine group as compared to control group and this difference was also statistically significant (p-value<0.05) at 5, 10, 20, 30, 40 min. and at the end of surgery.

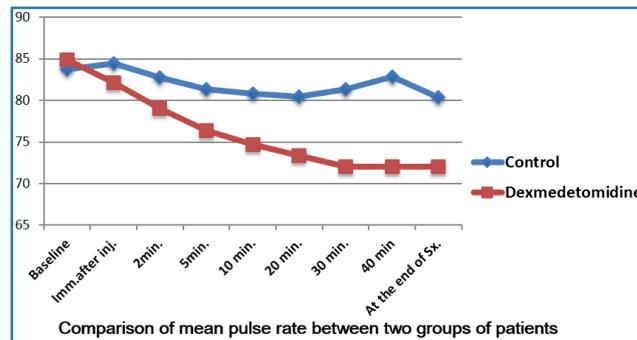


Fig. 2: Showing Comparison of Mean Pulse Rate between Two Groups of Patients

Blood Pressure

S.B.P., D.B.P., MAP were found to be lower in dexmedetomidine group as compared to control group. Difference in S.B.P. was statistically significant (p-value <0.05) at 5, 10, 20, 30, 40 min. and at the end of surgery while difference in D.B.P. and M.A.P. was statistically significant at 20, 30, 40 min. and at the end of surgery.

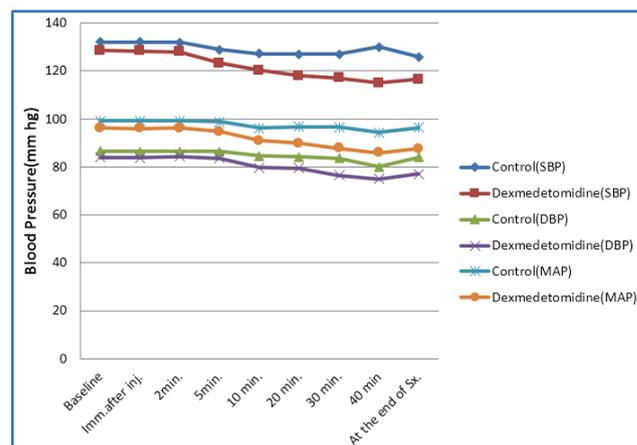


Fig. 3: Showing Comparison of S.B.P., D.B.P., and M.A.P. between Two Groups of Patients

Side Effects

In our study, while comparing the groups, intraoperative side effects were statistically insignificant. Incidence of hypotension was ≈6% (2/30) and that of bradycardia was ≈3% (1/30) in dexmedetomidine group, which was statistically not significant. Mean sedation score was 2.3 in dexmedetomidine group as compared to 1 in group C. Other side effects like nausea, vomiting, hypoxaemia, respiratory depression were not observed in either group.

Parameters	Group C	Group D
Hypotension	0	2
Bradycardia	0	1
Nausea and Vomiting	0	0
Respiratory Depression	0	0

Table 4: Showing Side Effects

DISCUSSION

The aim of proposed study was to evaluate whether additional anaesthetic and analgesic effects could be obtained after

administration of α_2 adrenoceptor agonist, dexmedetomidine, for brachial plexus block.

Dexmedetomidine, the pharmacologically active D-isomer of medetomidine is a highly specific and selective α_2 adrenoceptor agonist with $\alpha_2:\alpha_1$ selectivity ratio of 1620:1 as compared to 220:1 for clonidine, thus decreasing the unwanted side effects of α_1 receptors. High selectivity of dexmedetomidine to α_2 -A receptors mediates analgesia, sedation, and anxiolysis. Several mechanisms of action have been suggested to explain the analgesic effect of α_2 -adrenoceptor agonists in peripheral nerve blocks. Some of these include vasoconstriction around the injection site, direct suppression of impulse propagation through neurons as a result of a complex interaction with axonal ion channels or receptors, local release of enkephalin-like substance, a decrease in localised inflammatory mediators and an increase in anti-inflammatory cytokines through an α_2 -adrenoceptor mediated mechanism. It also has supraspinal analgesic action via noradrenergic neurons by hyperpolarisation. It inhibits norepinephrine release in descending medullospinal tract.

In present study, we used dexmedetomidine in the dose of 50 microgram. This was based on the observations of the previous studies conducted by Rancourt et al¹² and Yoginee Satishrao Patki et al¹³ etc.

The average age of the patients in group C was 37.6 ± 11.87 years and that in group D was 36.3 ± 12.58 years. Both groups were comparable and there was no statistically significant difference between the two groups in terms of age, gender, and weight. (p value >0.05). Both groups predominantly had male patients.

Onset of sensory block was 8.0 ± 1.02 min. in group C while it was 7.015 ± 1.04 min. in group D. This difference was statistically significant (p value <0.05). These findings correlate well with the studies of Amit R Khade et al,⁹ Esmaoglu et al,⁷ Obayah and Colleagues,¹⁴ Kenan Kaygusuz et al,⁸ Amay S Ammar and Mahmoud,¹⁵ Sandhya Agarwal et al.¹⁶ In Kenan Kaygusuz et al study, onset of sensory blockade was 7.75 ± 2.22 min. in patients who received levobupivacaine and dexmedetomidine (100 microgram) as compared with patients received levobupivacaine alone (10.75 ± 2.55 min.).⁸ In Sandhya Agarwal et al¹⁶ study, onset of sensory blockade was significantly earlier in dexmedetomidine (100 micrograms) group (13.20 ± 1.84 min. v/s 19.04 ± 3.19 min.).

Onset of motor block was 17 ± 1.44 min in group C while it was 14.5 ± 1.76 min in group D. This difference was statistically significant (p value <0.001). Similarly, Marhofer et al¹⁷ Ammar and Mahmoud¹⁵ Sandhya Agarwal et al¹⁶ in their studies found that motor block onset was hastened by the use of dexmedetomidine as an adjuvant in brachial plexus block with local anaesthetics. Sandhya Agarwal et al¹⁶ noted that the onset of motor block with dexmedetomidine (100 micrograms) was 16.3 ± 1.7 min. while plain bupivacaine having onset of motor blockade 22.7 ± 2.8 minutes. Ammar and Mahmoud¹⁵ found that onset of motor block was earlier in dexmedetomidine group (15.3 min vs. 22.2 min.).

In present study, mean of total duration of sensory block in group C was 390 ± 20.6 minutes and in group D was 756 ± 42.7 minutes. This difference was statistically significant (p value <0.001). Mean duration of motor block in group C was 313 ± 9.04 minutes and in group D was 639 ± 35.1 minutes. This difference was statistically significant (p value <0.001). These findings support to observations of various earlier studies of

Esmaoglu et al,⁷ Marhofer et al,¹⁷ Rancourt et al,¹² Rachana Gandhi et al,¹⁸ Kenan Kaygusuz et al,⁸ Ammar and Mahmoud,¹⁵ Sandhya Agarwal et al.¹⁶

In present study, mean duration of analgesia in group C was 487.66 ± 15.85 minutes while it was 979.67 ± 33.53 minutes in group D. Significant statistical difference was observed between the two groups with respect to mean duration of analgesia (p value <0.001).

Esmaoglu et al⁷ observed statistically significant (p value <0.05) longer duration of postoperative analgesia in dexmedetomidine group as compared with plain levobupivacaine. Obayah and colleague¹⁴ observed that the time for first analgesia was 22 hrs. in dexmedetomidine group compared with 14.2 hrs. in plain bupivacaine group. Similar results were observed by Rachana Gandhi et al¹⁸ where the total duration of postoperative analgesia was longer with dexmedetomidine (732.4 ± 98.1 min.) as compared with bupivacaine (194.8 ± 60.4 min). Kenan Kaygusuz et al⁸ observed that the postoperative duration of analgesia was 1279.54 ± 138.42 min in dexmedetomidine group as compared with levobupivacaine (736.80 ± 45.31 min.). Ammar and Mahmoud¹⁵ observed that total duration of postoperative analgesia was longer with dexmedetomidine (403 min.) as compared to bupivacaine (233 min.).

In group D, patients had decreased heart rate and reduced mean arterial pressure, which maybe explained with systemic absorption of dexmedetomidine. Presynaptic activation of α_2 adrenoceptor in central nervous system inhibits release of norepinephrine terminating prolongation of pain signals and their postsynaptic activation inhibits sympathetic activity thereby reduces heart rate and blood pressure. Baroreceptor reflex and heart rate response to pressor agent is well preserved with use of dexmedetomidine confirming haemodynamic stability.

No sedation was required in most of the patients in group D. This can be explained on the basis of alpha 2 agonist produce sedation by central action by inhibition of substance P release in the nociceptive pathway at the level of dorsal root neuron and by activation of alpha 2 adrenoceptors in locus coeruleus.

Respiratory rate and oxygen saturation were comparable in both the groups and there was no statistically significant difference (p-value >0.05).

In our study, intraoperative side effects were not statistically significant in both the groups. Incidence of hypotension was $\approx 6\%$ (2/30) and that of bradycardia was $\approx 3\%$ (1/30) in dexmedetomidine group, which was statistically not significant. Other side effects like nausea, vomiting, hypoxaemia, and respiratory depression were not observed in either group. Rancourt et al,¹² Kenan Kaygusuz et al,⁸ Sarita Swami et al,¹⁹ Sandhya Agarwal et al,¹⁶ observed similar results in their studies.

CONCLUSION

Thus, it can be concluded from the present study that 50 μ g of dexmedetomidine when used in combination with local anaesthetics solution in brachial plexus block gives early onset of sensory and motor block. It significantly prolongs the duration of analgesia to 979 minutes as compared to control group (487 minutes). There was also less requirements of analgesics in first 24-hour postoperatively. The addition dexmedetomidine provides added advantages like conscious

sedation and haemodynamic stability with minimal side effects.

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