

ADDITION OF DEXMEDETOMIDINE TO LIGNOCAINE FOR INTRAVENOUS REGIONAL ANESTHESIA IN UPPER EXTREMITY ORTHOPAEDIC SURGERIES

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ABSTRACT

Objective: Objective of this trial was to study time of onset, duration of sensory & motor blockade and quality of anaesthesia by addition of dexmedetomidine to local anaesthetic solution in Intravenous Regional Anaesthesia (IVRA) in upper extremity orthopaedic surgeries.

Methods: This was a prospective, randomized and double blind clinical trial. Ninety American Society of Anaesthesiologists Grade I and II patients of either gender between 18 and 60 years of age scheduled for elective upper extremity orthopedic surgeries lasting for <90 min were included in the study. Patients were randomly allocated to two Groups A and B of 45 each. Group A received 3 mg/kg preservative free lignocaine alone and Group B received 3 mg/kg preservative free lignocaine with dexmedetomidine, 0.5 µg/kg in IVRA.

Result: Onset time of sensory blockade in Group A and B was 5.6±0.93 min and 3.9±0.63 min respectively. Onset time of motor blockade in Group A and Group B was 15.01±4.53 min and 10.74±3.64 min respectively. The difference in onset time of sensory and motor blockade between the two groups was statistically significant (p<0.05). Sensory blockade recovery time after release of tourniquet was 6.9±0.53 min in Group A and 29.21±5.23 min in Group B. Motor blockade recovery time was 4.35±0.76 min for Group A and 12.32±7.23 min for Group B. The difference in sensory and motor blockade recovery time between the two groups was statistically significant (p<0.05).

Conclusion: Dexmedetomidine on addition to lignocaine for IVRA provided rapid onset of sensory and motor blockade, prolonged duration of sensory & motor blockade and reduced tourniquet pain.

Keywords: Intravenous regional anesthesia, Dexmedetomidine, Lignocaine, Sensory, Motor

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INTRODUCTION

Intravenous regional anesthesia (IVRA) is a simple, safe, reliable and effective technique to provide anesthesia for upper extremity surgeries with quicker onset and recovery with minimal expertise. Hence, it is safe and suitable for upper limb surgeries of shorter duration. However, IVRA has certain limitations such as very short post-operative analgesia and tourniquet pain when local anesthetics are used alone. This leads to discomfort to the patient [1]. To overcome these limitations, adjuvants such as opioids, for example fentanyl, morphine, pethidine, tramadol; non-steroidal anti-inflammatory drugs, for example ketorolac, tenoxicam, aspirin and alpha-2 agonists, for example clonidine, dexmedetomidine have been studied in IVRA to hasten the onset and prolong duration of analgesia [1-3].

Lignocaine, an amide local anesthetic prevents transmission of nerve impulses by inhibiting the passage of sodium ions through ion-selective sodium channels in the nerve membranes which slows the rate of depolarization such that the threshold potential is not reached resulting in non-propagation of action potential [4]. Lignocaine is the ideal local anesthetic agent for IVRA with low cardiovascular and central nervous system toxicity as bupivacaine in IVRA is associated with fatal cardiotoxicity whereas chloroprocaine is not used in IVRA after reports of hypersensitivity reactions and thrombophlebitis [5]. Although prilocaine and ropivacaine also can be used, lignocaine is the most commonly used local anesthetic agent in IVRA [6].

Dexmedetomidine, an alpha-2 adrenoreceptor agonist as an adjuvant to local anesthetics leads to a superior quality of anesthesia with lesser intra-operative and post-operative analgesic requirements [7-10].

Dexmedetomidine with lignocaine has been associated with prolongation of the duration of the sensory blockade and post-operative analgesia when used in nerve blocks [11]. Dexmedetomidine depresses nerve action potential especially in C fibers by a mechanism independent of the stimulation of alpha-2 adrenergic receptors [12]. This clinical trial was conducted to study effect of addition of dexmedetomidine to lignocaine on quality of anesthesia and analgesia when given in IVRA.

METHODS

This was a prospective, randomized, double blind and single-center clinical trial conducted at a tertiary care teaching hospital spanning over a period of 1 year. After approval of Institutional Ethics Committee, 90 patients of American Society of Anaesthesiologists (ASA) physical status I and II aged between 18 and 60 years who were scheduled for upper limb orthopedic surgeries lasting for <90 min were included in the study.

Inclusion criteria

ASA Grade I and II willing patients of either sex aged between 18 and 60 years scheduled for elective orthopedic surgeries of upper limb predicted to last for <90 min were included in the study.

Exclusion criteria

Patients with history of allergy to local anesthetics, coagulation disorders, sickle cell disease, Raynaud's disease, scleroderma, local infection, Paget's disease and patients who had contraindication to dexmedetomidine were excluded from the study.

Patients were allocated to two Groups A and B of 45 each by computer generated randomization method. Group A received 3 mg/

kg preservative free lignocaine 0.5% alone diluted to 40 ml volume. Group B received 3 mg/kg preservative free lignocaine, 0.5% with dexmedetomidine, 0.5 µg/kg diluted to 40 ml volume.

Informed consent was obtained from all the patients. All patients were kept fasting overnight. Patients were pre-medicated with intravenous Inj. midazolam, 0.03 mg/kg 45 min before surgery. Resuscitation equipment and drugs were kept ready. The initial pulse rate, blood pressure and peripheral oxygen saturation were recorded and then regularly monitored during the procedure at 5 min interval.

A 22 G cannula was placed intravenously as distal as possible in the arm to be operated on. Venous access was established in the opposite arm for administration of fluid or drugs if required. The double tourniquet was applied on the operative arm with generous layers of padding ensuring that no wrinkles were formed and the tourniquet edges were away from the skin. The arm to be anaesthetized was exsanguinated using Esmarch bandage. If this was not possible, exsanguination was achieved by elevating the arm for 2–3 min while compressing the axillary artery.

The proximal tourniquet was inflated at least 100 mm Hg higher than the patient's systolic blood pressure. Before injection of local anesthetic, radial pulse was palpated and confirmed that it was non-palpable. The local anesthetic was then injected slowly over 90 seconds. Then, the distal tourniquet overlying on part of the anesthetized arm was inflated and the proximal one was deflated. After confirmation of sensory and motor blockade by standardized tests, the surgeons were allowed to proceed.

Vital parameters and signs of drug toxicity if any were monitored regularly during intra-operative period. If patients complained of tourniquet pain or Visual Analog Score (VAS) >3, they were supplemented with Inj Fentanyl, 1 mg/kg IV. The cuff was not deflated for 30 min after local anesthetic injection even if surgery was complete before 30 min and was not kept inflated for more than 90 min. Cuff deflation was performed in cycles with deflation/inflation times of <10 s until the patient no longer exhibited signs of systemic toxicity.

Tourniquet pain was assessed on the basis of the VAS score (0="no pain" and 10="worst pain imaginable") and the degree of sedation was measured by Ramsay sedation scale (1=patient is anxious and agitated or restless or both; 2=patient is cooperative, oriented and tranquil; 3=patient responds to commands only; 4=patient exhibits brisk response to light glabellar tap or loud auditory stimulus; 5=patient exhibits a sluggish response to light glabellar tap or loud auditory stimulus and 6=patient exhibits no response) at 05 min interval after tourniquet application and injection of the anesthetic agent.

Patients were regularly monitored in the post-operative care room at 02 min interval for pulse, blood pressure, respiratory rate, peripheral oxygen saturation and recovery of sensory & motor blockade for 30 min. VAS and Ramsay sedation score was recorded at 05 min interval in the post-operative period for 30 min.

Statistical analysis

Data analysis was done with the help of computer using SPSS software. Range, frequencies, percentages, means, standard deviations, Chi-square and p-values were calculated using this software. P<0.05 was taken to denote significant relationship.

RESULTS

In our study, mean age of Group A was 38.4 years and that of Group B was 37.34 years.

There was no statistically significant difference in mean age of the patients between two groups (p>0.05).

Percentage of male patients was 66.6% in Group A and 60% in Group B. There was no statistically significant difference in gender distribution between the two groups (p>0.05) as shown in Table 1.

The onset time of sensory blockade in Group A was 5.6±0.93 min. This was significantly higher than the sensory blockade onset time in Group B (3.9±0.63 min) with p<0.05 as shown in Table 2.

The onset time of motor blockade in Group A was 15.01±4.53 min. The onset time of motor blockade in Group B was 10.74±3.64 min. It was significantly shortened in Group B (p<0.05) as shown in Table 3.

Duration of surgery was comparable in both the groups with no statistically significant difference as depicted in Table 4.

Sensory blockade recovery time after the release of tourniquet was 6.9±0.53 min in Group A and 29.21±5.23 min in Group B. It was significantly prolonged in Group B (p<0.05) as shown in Table 5.

Motor blockade recovery time was remarkably shorter (4.35±0.76 min) in Group A than that in Group B (12.32±7.23 min) which was statistically significant with p<0.05 as shown in Table 6.

Table 1: Distribution of Gender

Gender	Group A		Group B	
	No.	%	No.	%
Male	30	66.6	27	60
Female	15	33.3	18	40
Total	45	100	45	100
p-value	>0.05			

Table 2: Sensory Blockade Onset Time

Parameters	Sensory Blockade Onset Time (in min)	
	Group A	Group B
Range	4–6	1–4
Mean±SD	5.6±0.93	3.9±0.63
p-value	<0.05	

Table 3: Motor Blockade Onset Time

Parameters	Motor Blockade Onset Time (in min)	
	Group A	Group B
Range	11–27	8–12
Mean±SD	15.01±4.53	10.74±3.64
p-value	<0.05	

Table 4: Duration of Surgery

Parameters	Duration of Surgery (in min)	
	Group A	Group B
Range	41–56	40–55
Mean±SD	46.25±5.65	45.43±4.24
p-value	>0.05	

Table 5: Sensory Blockade Recovery Time after Tourniquet Release

Parameters	Sensory Blockade Recovery Time (in min)	
	Group A	Group B
Range	4–7	11–30
Mean±SD	6.9±0.53	29.21±5.23
p-value	<0.05	

Table 6: Motor Blockade Recovery Time after Tourniquet Release

Parameters	Motor Blockade Recovery Time (in min)	
	Group A	Group B
Range	2-6	6-16
Mean±SD	4.35±0.76	12.32±7.23
p-value	<0.05	

The average VAS score was more than 3 in Group A whereas it was <3 in Group B.

The average Ramsay sedation score was <2 in Group A whereas it was 2 in Group B.

Rescue analgesic supplementation was not required in Group B whereas Inj Fentanyl supplementation 1 mg/kg was given to 40 patients in Group A.

There was no statistically significant difference in pulse rate, blood pressure, respiratory rate and peripheral oxygen saturation between the two groups.

No drug related adverse effects were observed in both the groups.

DISCUSSION

Regional anesthesia offers numerous advantages over conventional general anesthesia (GA) including faster recovery time, fewer side effects, no need of airway manipulation during Surgery and dramatic reduction in post-surgical pain. Patients who receive regional anesthesia instead of GA also suffer from minimal or no post-operative nausea and vomiting and recover quickly after surgery [13]. IVRA reduces nursing time in the post-anesthesia care unit (PACU) and facilitates early hospital discharge when compared with GA and brachial plexus block for the upper extremity surgeries [14]. However, it often does not provide effective post-operative analgesia and incidences of tourniquet pain are reported. To overcome these limitations, adjuvants such as opioids, for example fentanyl, morphine, pethidine, tramadol; non-steroidal anti-inflammatory drugs, for example ketorolac, tenoxicam, aspirin and alpha-2 agonists, for example clonidine, dexmedetomidine have been studied in IVRA with variable success rate[15].

In our study, it was observed that mean onset time of sensory blockade in dexmedetomidine group (Group B) was 3.9±0.63 min as against 5.6±0.93 min in plain lignocaine group (Group A). The onset time of sensory blockade was remarkably shortened in study group (Group B) which was found to be statistically significant (p<0.05). Mean onset time of motor blockade in dexmedetomidine group (Group B) was 10.74±3.64 min as against 15.01±4.53 min in plain lignocaine group (Group A). The onset time of motor blockade was remarkably shortened in study group (Group B) which was found to be statistically significant (p<0.05).

In our study, it was also observed that mean recovery time of sensory blockade in dexmedetomidine group (Group B) was 29.21±5.23 min as against 6.9±0.53 min in plain lignocaine group (Group A). The recovery time of sensory blockade was remarkably prolonged in study group (Group B) which was found to be statistically significant (p<0.05). Mean recovery time of motor blockade in dexmedetomidine group (Group B) was 12.32±7.23 min as against 4.35±0.76 min in plain lignocaine group (Group A). The recovery time of motor blockade was remarkably prolonged in study group (Group B) which was found to be statistically significant (p<0.05).

However, Gupta *et al.* who compared two different doses of dexmedetomidine found it to be superior in terms of onset of sensory

block, onset of motor block and duration of post-operative analgesia when dexmedetomidine was used in the dose of 1 µg/kg instead of 0.5 µg/kg [15]. However, premedication with sedatives and narcotics was deliberately avoided in their study. All patients were premedicated with intravenous midazolam (0.03 mg/kg) 45 min before IVRA in our study.

The average VAS score was more than 3 in Group A whereas it was <3 in Group B. This indicates that dexmedetomidine augmented intra-operative and post-operative analgesia when combined with lignocaine for IVRA.

According to Esmoğlu *et al.* who conducted a study with dexmedetomidine as an adjunct to lignocaine in IVRA for the upper limb surgeries and concluded that addition of 1 µg/kg of dexmedetomidine to lignocaine for IVRA improved quality of anesthesia and post-operative analgesia but had no effect on onset and regression time of motor and sensory blockade [16]. However, Subramanya *et al.* found that the onset of motor and sensory block was rapid when dexmedetomidine 0.5 µg/kg was used along with lignocaine in IVRA. Quality of anesthesia was superior in dexmedetomidine group and also there was prolongation of post-operative analgesia [17].

The mechanism of tourniquet pain remains unclear despite the role of A fibers and unmyelinated C fibers [18]. Dexmedetomidine depresses nerve action potential, especially in C fibers by a mechanism independent of the stimulation of α-2 adrenergic receptors. This mechanism accounts for enhancement of the local anesthetic block achieved by perineural administration of the drug and could be implicated in the results observed in this study [18,19].

In this study, it was found that dexmedetomidine when added to local anesthetic for IVRA reduced tourniquet pain and intra-operative as well as post-operative analgesic requirement. Considering all the above factors, dexmedetomidine in the dose of 0.5 µg/kg can be used as an adjuvant to local anesthetic agent in IVRA for improvement in quality of anesthesia and analgesia.

CONCLUSION

From the data and statistical analysis, it was concluded that dexmedetomidine, 0.5 µg/kg when added to lignocaine for IVRA resulted in quicker onset of sensory and motor blockade, prolonged sensory & motor blockade and reduced tourniquet pain.

AUTHOR CONTRIBUTIONS

1. Dr. Santosh Kumar Singh – Drafting. 2. Dr. Gaurav Mittal – Result and Bibliography Search. 3. Dr. Ashok Rout – Statistical Analysis. 4. Dr. Dewendra J. Gajbhiye – Conception and Design of work. 5. Dr. Pradeep Kedar- Statistical Analysis and interpretation of data.

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