# **ORIGINAL RESEARCH**

# Effect of intravenous dexmedetomidine on post-operative analgesia in patients undergoing orthopedic lower limb surgeries under subarachnoid block, a placebo controlled trial

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Received: 15 February, 2023 Accepted: 21 March, 2023

## **ABSTRACT**

**Background:** Regional anesthesia is the preferred technique for most of abdominal and lower limb surgeries. This study was designed to evaluate the effects of intravenous dexmedetomidine on spinal anesthesia with 0.5% hyperbaric bupivacaine. **Materials & Methods:** The present study consisted of 160 patients of ASA Grade I & II patients planned for orthopedic lower limb surgeries under sub arachnoid block. Patients randomized into two equal groups of 80 patients each. Drugs prepared by the same person throughout the study, involved only in group allocation and drugs administration. Drugs for both groups prepared in two 50 ml syringes- one for loading dose (labelled L) and other for maintenance dose (labelled M). **Results:** The duration of onset of sensory blockade was 7.0 [6.0-7.0] minutes in group I and 6.0 [6.0-7.0] in group II. The duration of onset of motor blockade was 3.0 [3.0-4.0] minutes in both the groups. There was no statistically significant difference in the mean duration of onset of sensory and motor blockade of the subjects belonging to two groups (p value >.05). The sedation score was 2.5 [2.0-3.0] in group I and 3.0 [2.0-3.0] in group II. The duration of recovery from sensory blockade was 210.0 [180.0-210.0] minutes amongst group I subjects whereas it was 165.0 [150.0-180.0] minutes amongst group I subjects whereas it was 180.0 [180.0-210.0] minutes amongst group II subjects. The difference was significant (P< 0.05). There was significant difference in post-operative pain in both group (P< 0.05). **Conclusion:** Intrathecal dexmedetomidine prolongs the effect of subarachnoid anesthesia with arousable sedation.

Key words: dexmedetomidine, subarachnoid anesthesia, post-operative pain

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# INTRODUCTION

Regional anesthesia is the preferred technique for most of abdominal and lower limb surgeries. To prolong the duration of action of bupivacaine, many adjuvants such as epinephrine, phenylephrine, magnesium sulfate, neostigmine, opioids, clonidine have been used through intrathecal route. <sup>1</sup>

The molecular mechanisms of the analgesic action of  $\alpha 2$ -agonists are through activation of inwardly rectifying G1-protein-gated potassium channels, resulting in membrane hyperpolarization thus decreasing the firing rate of excitable cells in the central nervous system. In addition,  $\alpha 2$ -agonists inhibit neurotransmitter release through reduction in

calcium conduction into the cell. These two mechanisms represent two very different ways of affecting analgesia: first, by preventing the nerve from firing and second, by inhibiting propagation of the signal to its neighbor.<sup>2</sup>

ISSN: 2250-3137

Dexmedetomidine possesses anxiolytic, sedative, analgesic, and sympatholytic propertiesadministered by systemic and intrathecal routes and acts at both supraspinal levels and spinal level, laminae VII and VIII of the ventral horns of the spinal cord. It is, highly selective  $\alpha 2$ -adrenergic agonist, also use as premedication during general anesthesia due to its sedative and analgesic effects. Although there are not much clinical data regarding the effects of intravenous

dexmedetomidine on the duration of sensory and motor block of spinal anesthesia but synergistic interaction between intrathecal dexmedetomidine and local anesthetics has been observed in previous studies. <sup>4,5</sup>This study was designed to evaluate the effects of intravenous dexmedetomidine on spinal anesthesia with 0.5% hyperbaric bupivacaine.

# **MATERIALS & METHODS**

The present study consisted of 160 patients of ASA Grade I & II patients planned for orthopedic lower limb surgeries under sub arachnoid block. Exclusion criteria was haemodynamically unstable patients, coagulation disorder, allergy to local anesthetic amides, infectionat the site of lumber puncture, deformed spine and patient not giving consent.

A pre anaesthetic check-up was done. Patients kept nill by mouth for 8 hours prior to the procedure and an i/v line secured. Monitors attached, and vitals recorded. Patients randomized into two equal groups of 80 patients each. Drugs prepared by the same person throughout the study, involved only in group allocation and drugs administration. Drugs for both groups prepared in two 50 ml syringes- one for loading dose (labelled L) and other for maintenance dose (labelled M). Data recording done by another person not aware of the group of patients.

Dexmedetomidine in a dose of 0.7 μg/kg given as a loading dose in recovery room and patients of group (control group) receive normal saline. The study drug premixed to a total volume of 50 ml and administered through infusion pump @ 1ml/min as maintenance, 20 min after loading dose subarachnoid block with 0.5% hyperbaric bupivacaine given.

On arrival to operation-theatre, baseline vital parameters of heart rate, systemic arterial blood pressure, pulse oximetry and electrocardiography were recorded. All patients were pre-hydrated with 10 ml/kg Ringer lactate solution before initiation of the subarachnoid block. Under all aseptic condition, the subarachnoid block was established with an intrathecal injection of 3.5ml 0.5% hyperbaric bupivacaine in a sitting position at the L2-3 or L3-4 interspace through midline approach using a 25 G Quincke spinal needle.

ISSN: 2250-3137

Oxygen 5 l/min via a face mask was given throughout the surgical procedure. The patient and the anesthesiologist were blinded to the treatment group and all recordings were performed by an anesthesiologist, who was blinded to randomization schedule. Sensory block was assessed by pinprick method at 2 min intervals until the maximum level of the block was achieved and at 5 min interval subsequently. The motor blockade was evaluated bilaterally by modified Bromage scale.

Level of sedation was evaluated intra-operatively using Ramsey Sedation Score (RSS). The postoperative analgesia was noted with VAS Score. Total duration of analgesia was defined as time from administration of subarachnoid block until the first complaint of pain. Systemic arterial blood pressure, heart rate, pulse oximetry and electrocardiography were recorded at base line, after subarachnoid block at 3 min interval until 20 min and then at 5 min interval until the end of surgery. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

# **RESULTS**

**Table I: Distribution of patients** 

Gender		Group I	Group II	Total
Male	Number	66	59	125
	Percentage	82.5%	73.8%	78.1%
Female	Number	14	21	35
	Percentage	17.5%	26.3%	21.9%

In both the groups, majority of the subjects were males, the number of male and female subjects did not differ significantly between the groups (p value >.05).

Table II: Distribution of subjects based on age

Age groups		Group I	Group II	P value
18-30 years	Number	32	32	0.580
	Percentage	40.0%	40.0%	
31-40 years	Number	17	22	
	Percentage	21.3%	27.5%	
41-50 years	Number	17	11	
	Percentage	21.3%	13.8%	
51-60 years	Number	14	15	
	Percentage	17.5%	18.8%	

In both the groups, majority of the subjects belonged to the age group of 18-30 years. The number of subjects belonging to different age groups did not differ significant between the groups (p value >.05).

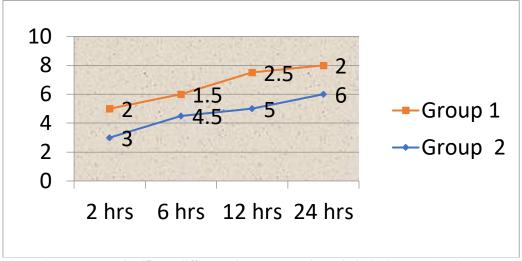
Table III: Assessment of parameters

Parameters	Group I	Group II	P value			
duration of onset of sensory block (minutes)	7.0	6.0	.418			
duration of onset of motor blockade (minutes)	3.0	3.0	.714			
sedation score	2.5	3.0	.636			
duration of recovery from sensory blockade	210.0	165.0	0.001			
duration of recovery from motor blockade	232.5	180.0	0.001			

The duration of onset of sensory blockade was 7.0 [6.0-7.0] minutes in group I and 6.0 [6.0-7.0] in group II. The duration of onset of motor blockade was 3.0 [3.0-4.0] minutes in both the groups. There was no statistically significant difference in the mean duration of onset of sensory and motor blockade of the subjects belonging to two groups (p value >.05). The sedation score was 2.5 [2.0-3.0] in group I and 3.0 [2.0-3.0] in

group II. The duration of recovery from sensory blockade was 210.0 [180.0-210.0] minutes amongst group I subjects whereas it was 165.0 [150.0-180.0] minutes amongst group II subjects. The duration of recovery from motor blockade was 232.5 [210.0-240.0] minutes amongst group I subjects whereas it was 180.0 [180.0-210.0] minutes amongst group II subjects. The difference was significant (P< 0.05).

**Graph I: Post-operative pain** 



Graph I shows that there was significant difference in post-operative pain in both group (P< 0.05).

# DISCUSSION

Subarachnoid block (SAB) with hyperbaric bupivacaine is routinely administered for abdominal and lower limb surgeries. Various agents have been used intrathecally as adjuvants to local anesthetic to increase efficacy and prolong the duration of SAB, among which opioids and a2 agonists are most commonly used. Intravenously administered dexmedetomidine has also been shown to prolong the duration of sensory and motor blockade obtained with subarachnoid block.6 When dexmedetomidine was given intravenously before spinal anesthesia or as a loading dose followed by continuous infusion during surgery, it also lengthened the duration of spinal anesthesia. This study was designed to evaluate the effects of intravenous dexmedetomidine on spinal anesthesia with 0.5% hyperbaric bupivacaine.

The results of this study indicate that infusion of dexmedetomidine hastens the onset of sensory block, though the onset of motor blockade was not affected. Faster onset of the sensory block may be due to alpha-2 receptor activation induced inhibition of nociceptive impulse transmission. Sharma et al<sup>8</sup>in their study seventy-five patients posted for lower limb and

infraumbilical procedures were enrolled for a prospective, randomized, double-blind, placebocontrolled study and divided into three groups: Group B (n = 25) received intravenous 20 mL 0.9% NaCl over 10 min followed by intrathecal 2.4 mL 0.5% bupivacaine + 0.2 mL sterile water; Group BDexIT (n = 25) received intravenous 20 mL 0.9% NaCl over 10 min followed by intrathecal 2.4 0.5% bupivacaine + 0.2 шL (5 dexmedetomidine; Group B Dex IV (n = 25) received intravenous dexmedetomidine 1 µg/kg in 20 mL 0.9% NaCl over 10 min followed by intrathecal 2.4 mL 0.5% bupivacaine + 0.2 mL sterile water. Onset and recovery from motor and sensory blockade, and sedation score were recorded. Although onset of sensory and motor block was similar in the three groups, motor recovery (modified Bromage scale 1) and two-segment sensory regression was prolonged in Group BDexIT > Group BDexIV > Group B (P < 0.001). Patients in Group BDexIT and Group BDexIV were sedated but easily arousable.

We found that in a study by Bayram A et al, the duration of onset of motor blockade was similar in both groups which similar in our result. The onset of sensory blockade in Group II with dexometomidine was 6 min as compare in the study by SS Hasroor et al<sup>9</sup> was just 1.6 minutes.

Sedation Score in our study was higher in the testing drug group than placebo similar to the study by Reddy VS et al.<sup>10</sup> Also, in study of Kumari R et. al.<sup>11</sup> Sedation Score for the drug group was 3.4 which was 3 in our study and placebo group was 2.9 and 2.5 respectively.

The Duration of regression of motor blockade in our study was 232.5 min and sensory blockade was 210 min in Group II which was higher than Group I, similarly in the study by Dinesh CN et al<sup>12</sup> the regression duration for motor & sensory blockade was 220.7 & 137.4 min respectively. The Postoperative pain according to VAS score was lower in Group 2 than Group 1in 24 hours, in the study by Halder S et al.<sup>13</sup> results were similar.

### **CONCLUSION**

Authors found that

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