ORIGINAL RESEARCH

Comparison Between Two Different Doses of Intrathecal Dexmedetomidine Along With Bupivacaine in Lower Limb Surgeries

Verma Reetu[1], Gupta Deepesh[2], Agarwal Aditya[3]

ABSTRACT

Introduction: The newer trends in regional anaesthesia for lower limb surgeries advocate use of lower dose of local anaesthetic alongwith adjuvants like opioids or α_2-agonists to prolong analgesia. The present study compared effects of two different doses of dexmedetomidine added to hyperbaric Bupivacaine on duration of sensory and motor block for lower limb surgeries.

Materials and Methods: A randomized double blind prospective controlled study involving 100 patients aged 20-50 yrs of ASA grade I, II scheduled for receiving subarachnoid block for lower limb surgeries was conducted. The patients were divided into two groups of 50 each. Each group was given 2.5 ml of hyperbaric Bupivacaine 0.5%. In addition Group-I was given 3 mcg dexmedetomidine in 0.5 ml of normal saline and Group-II was given 5 mcg of dexmedetomidine in 0.5 ml of normal saline. The parameters assessed were time of onset and regression of sensory & motor blockade, intraoperative hemodynamic parameters and complications. Results between two groups were compared using unpaired t-test. P-value <0.05 was considered significant.

Results: The time for sensory block to reach T10 dermatome and motor block to reach Bromage score-3 was significantly less in group-II. Also, the mean time of sensory regression to reach S1 dermatome and motor block regression to Bromage-0 was significantly prolonged in group-II.

Conclusion: Dexmedetomidine 5 µg can be safely used as an adjuvant to intrathecal Bupivacaine for early onset and prolonged duration of action in lower limb surgeries.

Keywords: Dexmedetomidine, lower limb surgeries, Intrathecal Bupivacaine.

How to cite this article: Verma Reetu, Gupta Deepesh, Agarwal Aditya. Comparison between two different doses of intrathecal dexmedetomidine alongwith Bupivacaine in lower limb surgeries. International Journal of Contemporary Medical Research 2015;2(3):658-662

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Source of Support: Nil

Conflict of Interest: None

INTRODUCTION

Spinal anesthesia is the most common anaesthetic technique for lower limb surgeries because it is economical, safe and easy to administer. However, the duration of action of local anaesthetics used for spinal anesthesia is shorter warranting early postoperative analgesic intervention. Many adjutants like opioids, midazolam, clonidine, dexmedetomidine etc. have been used to prolong the effect of spinal anaesthesia.[1,2] Earlier human studies hypothesize that intrathecal dexmedetomidine added to local anaesthetic would produce prolonged postoperative analgesia with minimal side effects.[3-5] This study was done to compare two different doses of dexmedetomidine added to intrathecal hyperbaric bupivacaine regarding onset, regression time and duration of sensory and motor blocks and effects on hemodynamics and sedation.

MATERIAL AND METHOD

This randomised prospective controlled study was conducted after approval from institutional ethics committee and written informed consent from all the patients. 100 patients of both the sexes between 20-50 years of age belonging to ASA I & II physical status scheduled for lower limb orthopaedic surgery were included in the study and were divided into two groups of 50 each. Exclusion criteria: Patient's refusal, patients using adrenergic receptor blockers or calcium channel blockers, coagulopathy, any contraindication of spinal anesthesia.

Patients were randomly divided into two groups using sealed envelope method. No patient was given any premedication prior to surgery. The parameters monitored were non invasive blood pressure, pulse rate, SpO2 and

ECG.

Spinal anesthesia was given in sitting position after local infiltration with 2 ml of lignocaine 2%. A 25 G Quincke tip spinal needle was introduced through L3-L4 interspace under strict a septic precautions and drugs were given as under:

Group-I patients received 2.5 ml of hyperbaric bupivacaine 0.5% + 3 mcg dexmedetomidine in 0.5 ml of normal saline.

Group-II received 2.5 ml of hyperbaric bupivacaine 0.5% + 5 mcg of dexmedetomidine in 0.5 ml of normal saline.

A senior anaesthesiologist prepared the drugs and gave spinal anesthesia. He was not further involved in the study. Hence, both the person collecting the data and the patient were blinded to the drugs used. Injection was given over 10 seconds. Immediately after completion of injection patient was made to lie supine. Supplemental oxygen was given at 3 l/minute via oxygen mask. Patients were monitored for mean blood pressure, pulse rate, respiratory rate and SpO2. Every 5 minutes for first 30 minutes then every 15 minutes thereafter till 180 minutes. Hypotension, defined as fall of systolic blood pressure by >30% of baseline or <90 mm of Hg was treated with intravenous mephentermine 6 mg and i/v fluids as required. Bradycardia defined as heart rate <60/minute was treated with i/v atropine 0.3-0.6 mg. Incidence of side effects such as respiratory depression, nausea and vomiting and sedation was recorded.

Level of sensory block was assessed by loss of pinprick sensation to 23G hypodermic needle every 2 minutes and time for sensory block to reach T10 dermatome was recorded. Onset of motor blockade was assessed by modified Bromage scale and time for attainment of complete motor blockade of lower limbs (Bromage-0) was recorded. Modified Bromage scale was scored as follows:

Bromage-0: Patient able to move hip, knee and ankle.

Bromage-1: Patient unable to move the hip but is able to move the knee and ankle

Bromage-2: Patient unable to move the hip and knee but able to move the ankle.

Bromage-3: patient unable to move the hip, knee and ankle.

When T10 sensory level and Bromage 3 score were attained, surgery was allowed. Meanwhile, assessment was done every 10 minutes till the time of two segment regression of block. Thereafter, assessment was done at 20 minutes interval till the block height decreased to S1 dermatome. Data regarding time to reach T10 dermatome and Bromage -3 and time to S1 level sensory regression, and time to reach Bromage-0 were recorded and compared.

Sedation was assessed by modified Ramsay Sedation Scale.

STATISTICAL ANALYSIS

Statistical analysis was done using computer Statistical software SPSS version 15 (statistical packages for Social sciences, Chicago, IL, USA). Data was expressed as either mean+/- Standard Deviation (SD) or numbers and pecentage.[6] Unpaired t-test was used to test the significance of results of quantitative variables. Chi-square test was used to test the significance of results of qualitative variables. $P<0.05$ was considered statistically significant.

RESULTS

The groups were comparable with respect to age, weight, sex distribution and duration of surgery. The results regarding the characteristics of sensory and motor blocks are summarised in Table-1. The time taken for sensory block to reach T10 dermatome (8.4+/-2.5 and 6.2+/-2.6 minutes respectively) was significantly shorter in group–II. The mean 2-segment regression time was longer in group-II as compared to group-I. Similarly sensory block regression to S1 segment was also shorter in group-II (261.6+/-23.0 and 277+/-23.3 minutes respectively). The time taken for motor block to reach Bromage-3 (15.8+/-3.5 and 13.0+/-3.2 minutes respectively) was significantly shorter in group –II and time for motor block regression to Bromage-0 was significantly more in group-II (236+/-26.3 and 246.4+/-

Characteristics of block	Group-I (n =50)	Group-II (n =50)	P-VALUE
Sensory block to reach T 10 dermatome.	8.4 ± 2.5	6.2 ± 2.6	<0.05
Motor block to reach Bromage 3.	15.8 ± 3.5	13.0 ± 3.2	<0.05
Sensory regression to S1 segment.	261.6 ± 23.0	277.0 ± 23.3	<0.05
Motor block regression to Bromage 0.	236 ± 26.1	246.4 ± 25.8	<0.05

Table-1: Block onset and regression time in minutes

25.8 minutes respectively).

Values are expressed as Mean+/- SD. P-value ≤0.05 is considered significant.

There was no significant difference in rate of complications like bradycardia, hypotension, respiratory depression, nausea and vomiting and sedation between the two groups (Figures-1,2,3,4) & (Table-2)

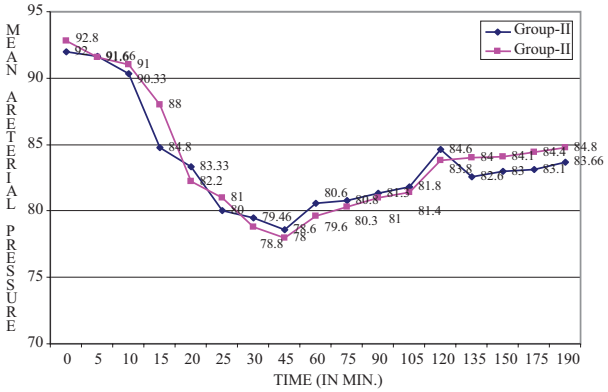

Figure-1: Comparison of Mean arterial pressure.

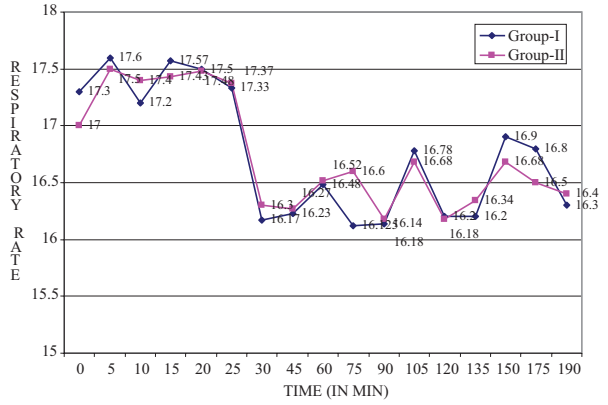

Figure-2 : Comparison of pulse rate between two groups

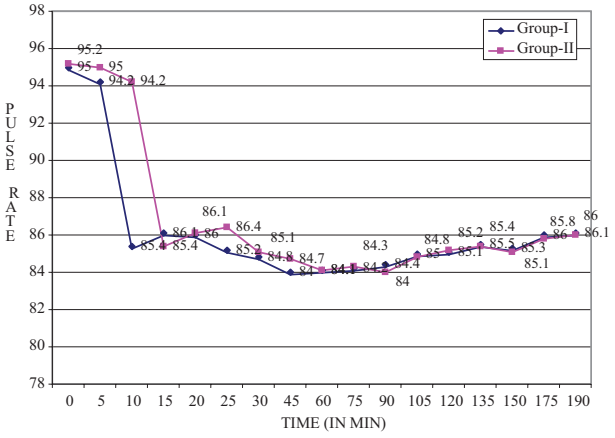

Figure-3: Comparison of Respiratory rate between two groups

DISCUSSION

The most prevalent technique of anaesthesia for lower limb surgeries is subarachnoid block using local anaesthetics. But the main problem is relatively shorter duration of action of these drugs necessitating early analgesic intervention. To prolong the duration of action, a number of adjutants like midazolam, opioids, and clonidine have been studied.[1-3] Our study is aimed to compare two different doses of dexmedetomidine as adjuvant to intrathecal bupivacaine. Many studies on animals such as rats, sheep, rabbits using intrathecal dexmedetomidine in the dose range of 2.5 -100 µg have been conducted without any neurological toxicity or deficit.[7-14] Intrathecal dexmedetomidine has also been used in human beings without any postoperative neurological deficit.[15-17] Kanazi et al used a small intrathecal dose of dexmedetomidine (3 µg) in combination with bupivacaine on humans for spinal anesthesia. The result showed an earlier onset of motor block and a prolongation of duration of sensory block with hemodynamic stability and lack of sedation.[5] A study by Al-Ghanem et al using 5 µg Dexmedetomidine as adjuvant to intrathecal bupivacaine in surgical procedures concluded it to be an alternative especially when long duration of action with minimal side effects and excellent quality of analgesia.[3] Dexmedetomidine, an imidazole compound, is the pharmacologically active dextro isomer of medetomidine that displays selective α-2 adrenoceptor agonism. The mechanism of action

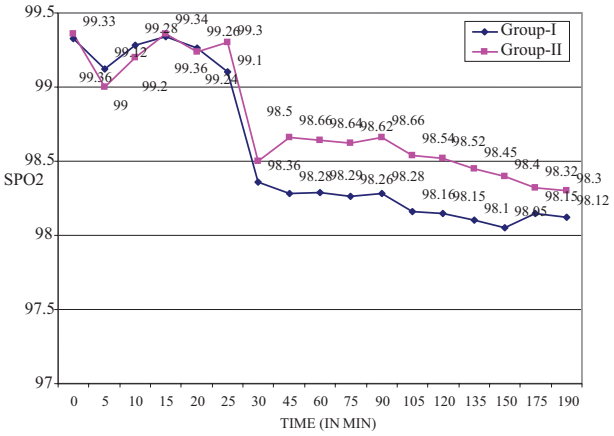

Figure-4: Comparison of SpO2 between two groups.

Side effects	Group-I	Group-II
Bradycardia (<60 BPM)	6(12%)	8(16%)
Hypotension (fall in B.P. >20% of baseline)	5(10%)	6(12%)
Respiratory depression	0	0
Nausea & Vomiting	0	0
Sedation	2	2

Table-2: Side Effects

by which intrathecal α-2 agonists prolong the motor and sensory block of local anesthetics is not clear. The local anesthetics act by blocking sodium ion channels whereas α-2 adrenoceptor agonists act by binding to presynaptic C-fibres and postsynaptic dorsal horn neurons. The analgesic action of intrathecal α-2 agonists is due to depression of release of C-fibre neurotransmitters and by hyperpolarisation of post-synaptic dorsal horn neurons.[18] The synergic effect of different mechanisms of action of Local analgesics and Dexmedetomidine may be responsible for prolonged sensory block as studied by Salgado et al.[16] The prolongation of motor block may be due to binding of intrathecal α-2 agonists to motor neurons in the dorsal horn.

In our study, we aimed to evaluate the role of 3 µg and 5 µg Dexmedetomidine added to heavy bupivacaine 0.5% intrathecally for lower limb surgeries.

In our study, the mean time for sensory block to reach T10 dermatome was significantly shorter in group-II (6.2 +/- 2.6 vs. 8.4+/-2.5) and regression time to reach S1 dermatome was prolonged in group-II. Similar to our results Al Mustafa MM et al showed an earlier onset and late regression of sensory block in Dexmedetomidine 10 µg group compared to Dexmedetomidine 5 µg group. The time for motor block to reach romage-3 was shorter (13+/-3.2 in group-II and regression of motor block to Bromage-0 was prolonged in group-II (246+/-25.8). Similar results were found in studies conducted by Al Mustafa MM and Hala EA Eid.[19] They found a dose dependent prolongation of motor blockade. The prolongation of motor blockade might be due to direct impairment of excitatory amino acid release from spinal interneurons.[19]

Bradycardia was seen in 12% cases in group-I and 16% cases in group-II. The difference was insignificant. Bradycardia is believed to be due to postsynaptic activation of central α-2 adrenoceptors (α2-ARs) which results in sympatholytic effect leading to bradycardia and hypotension.[20]

In our study hypotension was observed in 5 (10%) of group-I patients and 6 (12%) of group-II patients. The difference was insignificant. Kanazi et al showed insignificant effect of Dexmedetomidine on mean blood pressure when added to intrathecal bupivacaine.[5] Al Mustafa and colleagues in their study using 5 µg and 10 µg found a dose dependent but insignificant decrease in mean blood pressure when compared to bupivacaine (control) group.[4]

There was no incidence of nausea and vomiting or respiratory depression in our study.

The mean sedation score in both the groups was 2. The sedative effect of α-2 agonists is due to action on α-2 adrenergic receptors in locus coeruleus.[20,21] The cause of sedation after intrathecal Dexmedetomidine may be due to its systemic absorption and vascular redistribution to higher centres or cephalad migration in CSF.[15] Hence, our results were in agreement with previous studies.

CONCLUSION

From our study we came to the conclusion that 5 µg of dexmedetomidine can be safely used for early onset of sensory motor blocks and prolonged duration of analgesia without any adverse effect.

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