Original Research Article

Indian Journal of Anesthesia and Analgesia
Volume 7 Number 5, September – October 2020
DOI: http://dx.doi.org/10.21088/ijaa.2349.8471.7520.40

Comparative Study to Evaluate Intravenous Dexmedetomidine as Bolus Versus Infusion with Spinal Anaesthesia for Infra-Umbilical Surgeries

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How to cite this article:
Shah Parth M, Juhi Mattoo, Thakur Dinesh. Comparative Study to Evaluate Intravenous Dexmedetomidine as Bolus Versus Infusion with Spinal Anaesthesia for Infra-Umbilical Surgeries. Indian J Anesth Analg. 2020;7(5):1149–1154.

Abstract

Context: Dexmetedomidine, an alpha 2 agonist as adjuvant to spinal anesthesia for prolongation of sensory, motor block, postoperative analgesia.

Aims: We studied effectiveness of intravenous dexmedetomidine in prolonging duration of subarachnoid block when administered as bolus and bolus plus infusion dose.

Methods and Material: 100 ASA 1 and 2 patients scheduled to undergo elective surgeries under SAB were randomly allocated into two groups. After SAB,Group B received 1 µg/kg of dexmedetomidine bolus over 10 min and group Ireceived 0.5 mcg/kg over 10 mins followed by 0.5 mcg/kg over next 60 mins.Time of onset, duration of sensor and motor blockage, hemodynamic stability and sedation score were observed in both group patients.

Statistical analysis used: Results were evaluated by applying paired t test and P value using SPSS statistical software.

Results: Time to onset of sensory blockade (T10) in group B was 4.3 ±1.02 mins and group I - 5.2±1.6 mins. Time of onset of motor blockade in group B and I was 4.6 ± 1.01 and 5.5 ± 1.4 mins respectively. Two segment regression time was prolonged in group I – 124 ± 11.51 mins as compared to group B 110± 12.2 mins. Time to achieve complete sensory recovery in group B was 211 ± 10.2 min and group I was 240 ± 9.24 min. Time to achieve complete motor recovery in group B and I was 196 ± 9.6 and 219 ± 6.2 min respectively.

Conclusions: Both dosage regimens of dexmedetomidine can be used for prolongation of spinal anaesthesia with bupivacine. Time of onset of block is faster in bolus group however bolus plus infusion dosage provides more prolonged sensory and motor regression of block.

Keywords: Intravenous dexmedetomidine; Subarachnoid block; Bolus; Bolus plus infusion.

Keymessages: Intravenous Dexmedetomidine can be Used as an Adjuvant for Prolongation of Spinal Anaesthesia in Bolus as Well as Bolus and Infusion Doses.

Introduction

Subarachnoid block is regional anaesthetic technique used frequently to produce intense sensory and motor blockade for infraumbilical and lower limb surgeries. Several drugs such as opioids and alpha-2 agonists can be used as adjuvants to prolong sensory and motor blockade1 so as to provide sedation and postoperative analgesia to patients.

Dexmedetomidine is a highly selective alpha-2 agonist with relatively high α2/α1 activity (1620:1)

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as compared to clonidine (220:1).[2] It possesses advantages of sedative, analgesic properties with lack of respiratory depression makes it a preferable and suitable adjunct in various clinical anaesthesia.[3,4]

It was introduced for short-timeintensive care unit sedation in 1999. Since, thenit is rapidly emerging drug now-a-days as anadjuvant to regional and general anesthesia, MAC, premedication for prolonged postoperative sedation andanalgesia.[5]

Apart from conventional technique of adding adjuvants intrathecally, we carried out study with new approach gaining importance of usingintravenous dexmedetomidine to prolong duration and intensity of SAB and to provide sedation during the perioperative period. Thereby desirable goals of postoperative analgesia and intraoperative sedation are achieved.

The primary aim of this study was to assess the quality of subarachnoid block on different iv dosage of dexmedetomidine as bolus and infusion in elective surgeries.

Materials and Methods

After approval from the Institutional Ethics Committee and obtaining written and informed consent from the patients wecarried out this randomized prospective comparative double blind study.

100 adult patients of either sex, aged between 18 and 65 years belonging to American Society of Anaesthesiologists Physical Status (ASA-PS) 1 or 2 scheduled to undergo electiveinfraumbilical or lower limb surgery under spinal anesthesia were enrolled. Patients who refused to give consent, belongingto ASA 3–4, emergency surgeries, patients with co-agulopathis,bleeding diathesis, allergic to local anaesthetic agents or otherdrugs were excluded from the study. A detailed preoperative evaluation was performed prior to surgery, and were kept Nil by mouth overnight.

On the day of surgery, 18G IV line was secured in thenondominant hand and iv fluid was administered- ringer lactatesolution at 100 mL/h.

Patient was shifted to the opertation theatre. Multiparamonitoring system was established that included 5-electrode electrocardiogram monitoring Lead II and V 5, noninvasive blood pressure, and pulse oximetry and baseline vitals recorded.– pulse, BP, SP02, RR .Drug preparation and administration was done intravenously by one of the two anesthesiologists not involved in data collection. Another anesthesiologist who was kept blinded about the study drug administered performed subarachnoid block and recorded the sensory and motor effects of spinal anesthesia.

Patients were randomly allocated into one of the two groups (50 patients in each group) using a computer-generated random number table.

Under all aseptic and antiseptic precautions: Intrathecal bupivacaine (2.5 mL of 0.5% bupivacaine heavy) was administered in both groups at L 3 –L 4 or L 4 –L 5 interspace using a23 standard wire gauge Quincke-Babcock spinal needle after confirming free flow of clear cerebrospinal fluid . The time ofintrathecal drug injection was noted as time "0" and the patientturned supine. Immediately after spinal anaesthesia with intrathecal hyperbaric bupivacaine patients were given dexmedetomidine intravenous as per group allocation.

Group B: received bolus 20 mL of 0.9% NaCl containing 1 µg/kg of dexmedetomidine (rounded to nearest 10 micrograms) intravenously over 10 min followed by 20 mL of 0.9% NaCl overnext 60 min.

Group I: received intravenous dexmedetomidine in atotal dose of 1 µg/kg (rounded to nearest 10 micrograms). Initially half of this dose (0.5 µg/kg) diluted in 20 mL of 0.9%NaCl was administered over the first 10 min, followed by theremaining half dose (0.5 µg/kg) diluted in 20 mL of 0.9% NaClover the next 60 min.

Onset of sensory blockade at T 10 was noted. In addition, 2-segment regression time (defined as recovery of sensory blockby two segments from the highest sensory level achieved inthat patient) and sensory recovery (defined as recovery at S2 – S4 dermatomes) were also noted.

Motorblock was assessed by modified bromage scale:

0 No paralysis, able to flex hips/knees/ ankles.

1 Able to move knees, unable to raise extendedlegs

2 Able to flex knees, unable to flex knees

3 Unable to move any part of the lower limb.

Motor blockade wasperiodically assessed till a modified Bromage score of 3 (inability to flex hip, knee, and ankle) was obtained. This timewas denoted as onset of motor blockade. Attainment of amodified Bromage score of 0 (ability to flex hip, knee, and ankle) was noted to herald recovery from motor blockade.

Sedation score was noted on a 6-point Ramsay sedation score Ramsay Sedation Scale (RSS):

1. Patient fully awake and oriented.
2. Patient cooperative, drowsy and tranquil.
3. Patient asleep but responds to oralcommands.
4. Asleep, but responds to light glabellar tap.
5. Asleep, sluggish response to light glabellartap.
6. Asleep, no response.

Hemodynamic parameters - blood pressure, heart rate, respiratory rate, and oxygensaturation were monitored throughout the surgery.anynotable Adverse effects such as nausea, vomiting, andpruritus were noted.

Results

There was no significant statistical difference inall patients in Group B and Group I with respect todemographic profile that included patients age, sex,height, weight, ASA physical status and durationof surgery, (Table 1).

Table 1: Demographic Data.

Parameter	Group B (N= 50)	Group I (N= 50)	P Value
Age (Years)	38.2±14.5	45.1±10.4	0.0931 (Not Significant)
Weight (Kg)	68.2±11.1	65.1±12.4	0.1909 (Not Significant)
Height (Cm)	168±10.2	169.4±8.8	0.4642 (Not Significant)
Sex M:f	46:4	44:6	
Asa 1 :2	32:18	28:22	

The time to onset of sensory blockade (at t10)in group B was 4.3 ±1.02 mins and group I - 5.2±1.6 mins with p value 0.0011 . the time of onset of motor blockade (BROMAGE 3) in group B and I was 4.6 ± 1.01 mins and 5.5 ± 1.4 mins respectively with P value 0.0004 (Table 2).

Table 2: Spinal Anaesthesia Parameters.

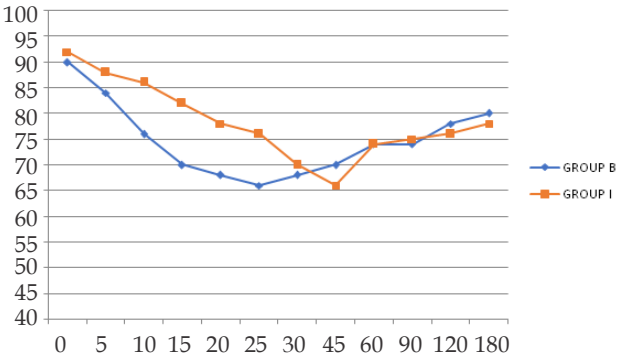
Parameter	Group B (N= 50)	Group I (N= 50)	P Value
Time To Onset Of Sensory Blockade (At T10) Min	4.3 ± 1.02	5.2 ± 1.6	0.0011 (Significant)
Time To Onset Of Motor Blockade (Bromage 3) Min	4.6 ± 1.01	5.5 ± 1.4	0.0004 (Significant)
2 Segment Regression (Min)	110 ± 12.2	124 ± 11.51	0.0001 (Significant)
Complete Sensory Recovery	211 ± 10.2	240 ± 9.24	0.0001 (Significant)
Complete Motor Recovery	196 ± 9.6	219 ± 6.2	0.0001 (Significant)
Time To Reach Ramsay Sedation Score Of 3	6.3 ± 2.3	6.6 ± 2.6	0.5425 (Significant)

The two segment regression time was prolonged in group I – 124 ± 11.51 mins as compared to group B 110 ± 12.2 mins and the difference is significant p value 0.0001 (Table 2).

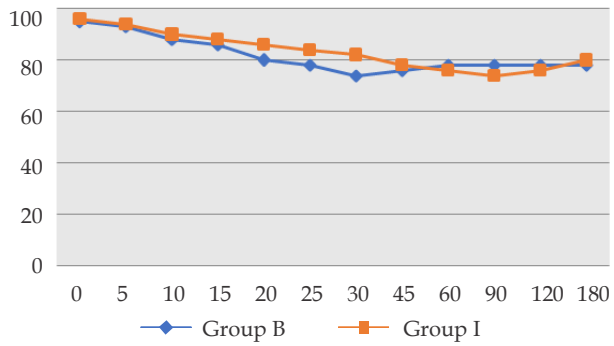
Time to achieve complete sensory recovery in group B was 211 ± 10.2 min and group I was 240 ± 9.24 min with p value 0.0001 (highly significant). Time to achieve complete motor recovery in group B and group I was 196 ± 9.6 min and 219 ± 6.2 min respectively and its p value was 0.0001 (highly significant) (Table 2).

Time to reach Ramsay Sedation of score 3 in group B and I was nearly same Group B : 6.3 ± 2.3 mins and group I was 6.6 ± 2.6 mins. (Table 2).

Heart rate and Blood Pressure were continuously monitored and graph plotted (GRAPH 1 and 2) and the decrease in heart rate was more evident for intital transitory period in group B as compared to group I.



Graph 1: Heart Rate.



Graph 2: Mean Arterial Pressure (Map).

Discussion

We conducted a study to observe the effect of intravenous dexmedetomidine given as a bolus and bolus plus infusion immediately after spinalanaesthesia with intrathecal hyperbaric bupivacaine.

The demographic data that included age, height, weight, ASA physical status (duration of surgery) were comparable among the two groups and there was no significant difference between them (Table 1).

The time of onset of sensory blockage in group B was 4.3 ± 1.02 mins faster than group I – 5.2 ± 1.6 mins with p value 0.0011. the time of onset of motor blockade in group B and I was 4.6 ± 1.01 mins and 5.5 ± 1.4 mins respectively with P value 0.0004 ,both these p values reflect a significant difference between them. (Table 2).

The above faster onset of action in group B might be due to the fact that bolus dose administration has more effect in fastening the time to reach the T10 level.

Thomas et all in his study entitled "Comparison of different regimens of intravenous dexmedetomidine on duration of subarachnoid block" compared patients receiving dexmedetomidine and divided them among 3 groups. He concluded quick time of onset to achieve sensory level of T10 in patients recvng bolus doses of dexmedetomidine. These observations were similar to our study.[6]

However Upadhyaya R. Kavya in their research entitled: "effect of intravenous dexmedetomidine administered as bolus or as bolus plus infusion on subarachnoid anaesthesia with hyperbaric bupivacaine" observed that the time of onset of sensory blockade was almost similar in both bolus and infusion groups of dexmedetomidine.[7]

SS Harsoors's study "effect of supplementation of low dose iv dexmedetomidine on characteristics of hyperbaric bupivacaine " also support our result as in their study that dexmedetomidine when given as bolus plus infusion hastened the onset of sensory blockade.[4]

The alpha receptors activation induced inhibition of nociceptive impulse transmission may be leading to faster onset of sensory blockade as compared to motor blockade.

The two segment regression time was prolonged in group I – 124 ± 11.51 mins as compared to group B 110 ± 12.2 and the difference is significant p value 0.0001. This may be due to the longer duration of action of group I where the drug was administered as an infusion followed by bolus dosage.

Our findings were similar to TriptiVatsalya's study" comparison of intravenous bolus and infusion of dexmedetomidine on characteristics of subarachnoid block" where patients receiving intravenous dexmedetomidine as infusion showed prolonged 2 segment regression time as compared to bolus dosage. Thomas et all concluded the same in his study.[8]

In our study time to achieve complete sensory recovery in group B was 211 ± 10.2 min and group I was 240 ± 9.24 mins with p value 0.0001 (highly significant). Time to achieve complete motor recovery in group B and group I was 196 ± 9.6 and 219 ± 6.2 respectively and its p value was 0.0001 (highly significant). Hence in our study bolus plus infusion dosage (group I) of intravenous dexmedetomidine prolongs the sensory and motor blockade of spinal anaesthesia as compared to bolus administration only.

SS Harsoor has results similar to our study thus strengthen our results and observations.[4]

Other researchers also carried out comparative study and observed the effect of dexmedetomidine infusion on spinal anaesthesia with ropivacaine and concluded that dexmedetomidine bolus of 1 microgram/kg followed by 0.4 mcg/kg/hr prolonged sensory and motor regression.[9]

Study entitled "Intravenous dexmedetomidine prolongs bupivacaine spinal analgesia " by Mustafa et all included comparison of loading plus infusion dosage of dexmedetomidine with normal saline (placebo) resulted in prolongation of spinal anaesthesia with sensory and motor regression.[10]

Dexmedetomidine provides good sedation with wide safety margins and doesnot cause much respiratory depressiom. Moreover sedation produced by it is different from other sedatives as patients receiving dexmedetomidine remain co-operative and are easily arousable. This is termed as "co-operative arousable sedation" and is easily distinguished from sedation caused by drugs-propofol and midazolam that act by inhibiting GABA.[11]

Here we assessed sedation with Ramsay Sedation Score (RSS) and observed that patients of both groups took nearly similar times to reach RSS score of 3 with no statistical significant difference.

However the duration for which RSS was maintained 3 was greater in group bolus plus infusion (group I) than group bolus. (group B) that

was maintained throughout in group I and for 60 mins in group B.

In our study the decrease in Heart Rate was more evident for initial transitory period in group B than group I. However only 1 patient required atropine in group B. There was hypotension and fall in Mean Arterial Pressure (MAP) in both groups intraoperatively but clinically was not that significant.

The above bradycardia and hypotension can be explained by the fact that dexmedetomidine doesnot have any direct effect on heart. It causes dose dependent increase in coronary Vascular Resistence and o2 extraction in coronary circulation but supply/demand ratio remains unaltered.[12]

Dexmedetomidine causes biphasic cardioversion response after administration. Pharmcaokinetics describe that bolus dose of 1 mcg/kg leads to transient rise in BP and reflex decrease in HR. This initial response occurs due to direct B-adenoreceptor stimulation of vascular smooth muscle. However this response is attenuated when it is administered as slow infusion over 10 mins. Hence we in our study administered dose over 10 mins which resulted in stabilization of Heart rate and BP 10–15% below the baseline parameters.

Jyotsna Kubre et all in their study of 0.5 mcg/kg dexmedetomidine loading over 10 mins concluded no difference in MAP in both groups and only 2 patients required atropine and ephidrene to treat bradycardia and hypotension.[13]

Mustafa et al, Tekin et al and Whizar Lugo et all reported no significant difference in Mean Arterial Pressure in dexmedetomidine group in their studied.[10,14,15]

Conclusion

Administration of intravenous dexmedetomidine as an adjunct to spinal anaesthesia in a dose of 1 mcg/kg aver 10 mins and 0.5 mcg/kg over 10 mins followed by 0.5 mcg/kg over next 60 mins both prolong the sensory and motor blockade produced by 12.5 mg of intrathecal hyperbaric bupivacaine.

However time of onset of bolus dosage of 1 mcg/kg was faster than bolus plus infusion divided dosage of 0.5 mcg/kg. however the bolus plus infusion dosage had prolonged sensory and motor regression of blockade as compared to bolus dosage regimen solely.

Both dosage regimens had hemodynamic stability in parameters within tolerant and treatable limits with arousable sedation and Ramsay Sedation Score of ≥ 3 was maintained.

Hence we conclude that Intravenous Dexmedetomidine in above mentioned 2 dosage regimens can be used as a adjuvant for prolongation of spinal anaesthesia without any notable adverse events.

Acknowledgement:

Conflict of Interest: nil

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