ORIGINAL RESEARCH

A study on the anaesthetic and analgesic effects of intravenous dexmedetomidine as premedication for spinal Anaesthesia

¹Dr. Ayushi Ramawat, ²Dr. Sameer Goyal, ³Dr. Sonu, ⁴Dr. Prakash Chandra Audichya, ⁵Dr. Vijay Kumar, ⁶Dr. Krishan Gopal Jangir

¹Post-Graduate Resident, ²Professor, ³Senior Resident, ⁴Professor and Head, ⁵Associate Professor, ⁶Assistant Professor, Department of Anaesthesia, Pacific Medical College and Hospital, Udaipur, Rajasthan, India

Corresponding Author

Dr. Ayushi Ramawat

Post-Graduate Resident, Department of Anaesthesia, Pacific Medical College and Hospital, Udaipur, Rajasthan, India

Email: dr.ayushiramawat@gmail.com

Received: 19 March, 2023 Accepted: 22 April, 2023

ABSTRACT

Background: Spinal anaesthesia is a popular technique for infraumblical surgeries. Dexmedetomidine is a highly potent novel α_2 -adrenoceptor agonist also used as adjunct in spinal anaesthesia. Aim and Objectives: The aim of the present study is to assess the effects of intravenous Dexmedetomidine as premedication on the onset, level and duration of sensory and motor blockade, analgesia and sedation in patients undergoing infraumblical surgeries under Bupivacaine (hyperbaric, 0.5%, 3mL) spinal anaesthesia. Material and Methods: A total of 100 patients undergoing spinal anaesthesia were randomly divided into two groups, Group D (n=50) received inj. Dexmedetomidine 0.5mcg/kg diluted in 10ml of sterile water and Group C (n=50) received 10ml of normal saline intravenously over 10 mins half an hour before surgery. **Result:** The sensory block duration was significantly higher in group D (137.6) than in group C (74.2), p=0.000, and the motor block in group D (178) was significantly higher as well than that in group C (129.6), p=0.000. The time of request for rescue analgesia in the postoperative period was significantly extended in group D (236.24±45.91) when compared to group C (145.80±22.50), p=0.000. The Ramsay sedation score of the patients in group D was significantly high-82% of the patients had a sedation score of 3 whereas all the patients in group C had Ramsay sedation score of 2 only. Conclusion: We concluded that the use of intravenous Dexmedetomidine as premedication prolonged the duration of sensory and motor block while maintaining a stable hemodynamic profile and providing adequate arousable sedation.

Keywords: Anaesthetic and analgesic, intravenous dexmedetomidine, premedication, spinal Anaesthesia

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

INTRODUCTION

Spinal anaesthesia is a popular technique used for a number of infraumbilical surgical procedures due to benefits of a quicker onset time and a well-recognized and approved sensory and motor blockage.

Bupivacaine is the most commonly used local anesthetic agent for spinal anaesthesia. Many adjuncts have been used to extend the duration of spinal analgesia such as epinephrine, phenylephrine, adenosine, magnesium sulphate, and clonidine etc. Utilizing these adjuncts may have benefits such as fewer local anaesthetic dose requirements, which helps prevent the negative effects of local anaesthetics, delayed perioperative pain onset, decreased analgesic requirements.

Dexmedetomidine α₂-receptor agonists used for procedural sedation and ICU sedation due to its quicker recovery time. Dexmedetomidine has an inhibitory action on the locus coeruleus (A6 group) present at the brain stem. The prolongation of spinal anaesthesia is indicative of the supraspinal action of Dexmedetomidine when given intravenously^[1].It helps reduce the opioid dose requirement and decrease the respiratory and addictive side-effects therewith^[2]. Studies have shown a quicker onset of blockade and significantly longer duration with intrathecal Dexmedetomidine as an adjunct with bupivacaine for regional anaesthesia with stable hemodynamic profile^[3]. Only few studies have been conducted to study the effects of intravenous Dexmedetomidine during spinal anaesthesia. This study was conducted compare the effects intravenous of Dexmedetomidine patients posted on infraumblical surgeries under spinal anaesthesia.

AIMS AND OBJECTIVES

To evaluate the effects of intravenous Dexmedetomidine as premedication on the onset, level and duration of sensory and motor blockade, postoperative analgesia, sedation level and perioperative hemodynamic stability in patients posted for infraumbilical surgeries under Bupivacaine (hyperbaric, 0.5%, 3mL) spinal anaesthesia.

MATERIALS AND METHODS

This was a comparative, prospective, randomized double blind controlled study carried out in the patients posted for infraumblical surgeries.

SAMPLE SIZE

Sample size was calculated using the formula $n = \underbrace{(Z_\alpha + Z_{1-\beta})^2 \ P(1-P)}_{E^2}$

TOTAL POPULATION

100 patients.

Patients were allocated into two groups of 50 each using computer generated random number.

Group D(Study group)-Bupivacaine plus Dexmedetomidine group 0.5mcg/kg.

Group C (Control group)-Bupivacaine plus Normal Saline group.

METHODOLOGY

This study was conducted at a tertiary care service hospital between January 2021 to December 2022 after approval from the institutional ethical committee. ASA grade 1 and 2 patients of either sex, aged between 20-60 years of age, weighing 50-70kgs, posted for elective lower abdominal or lower limb Surgeries of less than three hours duration under spinal anaesthesia were enrolled for the study after obtaining written informed consent. Patients of ASA Grade III and IV, localized skin infection, coagulation disorder, head injury, refusal to spinal anaesthesia were excluded in our study.

All the patients were visited on the previous day of the surgery and explained in detail about the anaesthetic procedure, reassured and written informed consent taken. The patients were kept nil per oral 8 hrs prior to surgery. On arrival to the premedication room 18G venous cannula was secured and all the patients were preloaded with 500ml of Ringer lactated solution. All standard monitoring like electrocephalography (ECG), non-invasive blood pressure (NIBP) and pulse oximeter (SPO₂) and capnography were attached and baseline parameters recorded.

Premedication for patients in Group D, included Inj. Ranitidine 50mg iv, Inj. Metoclopramide 10mg iv and Inj. Dexmedetomidine, 0.5mcg/kg which was diluted to make up a total of 10ml with sterile water and infused intravenously over 10 mins whereas patients

in Group C received 10ml of normal saline intravenously over 10 mins with the aid of an infusion pump half an hour before surgery. The patients were positioned lateral decubitus and dural puncture performed at L3-L4 interspace through midline approach with 25 G Quincke needle, 15mg (3mL) of 0.5% Hyperbaric Bupivacaine was injected intrathecally and were immediately positioned supine for the surgery.

The heart rate (HR), mean arterial blood pressure (MAP), peripheral oxygen saturation(SpO₂), end tidal CO₂ (ETCO₂), respiratory rate(RR) were recorded before premedication, 2 mins after premedication, before and after intrathecal block every 2 minutes till ten minutes and then every 15 minutes till end of surgery. Fall in MAP below 20% of baseline or a systolic pressure less than 90 mm Hg was considered as hypotension for the purpose of the study and was treated with incremental doses of Inj. Ephedrine and a bolus administration of 250 ml RL over 10 minutes. Heart rate below 50 bpm was considered bradycardia and was treated with 0.6mg Inj. Atropine iv.

SENSORY BLOCKADE

The highest level of Sensory blockade was assessed using cold iced tube in the midaxillary plane. Time of onset from intrathecal drug injection to achieved highest sensory dermatome level was noted.

MOTOR BLOCKADE

Motor blockade was assessed by Modified Bromage scale^[4]

Modified Bromage scale 0 - No paralysis.

Modified Bromage scale 1 - Unable to raise extended leg.

Modified Bromage scale 2 - Unable to flex knee. Modified Bromage scale 3 - Unable to flex ankle.

SEDATION

Sedation was assessed using Ramsay sedation score^[5]at 10, 30, 50, 70, 90, 110 and 120 min.

TIME FOR FIRST RESCUE ANALGESIA

Postoperative pain was measured using Visual Analog Scale(VAS). InjectionTramadol 50 mg iv was given for rescue analgesia if the VAS score was more than 3.

STATISTICAL ANALYSIS

Statistical Package for Social Sciences (SPSS 16.0) is used for the statistical analysis of the present study. Independent t test is applied to find the statistical significant between the two groups. p value (p<0.05) is considered statistically significant 95% confidence interval. The datas are expressed in number, percentage, mean and standard deviation.

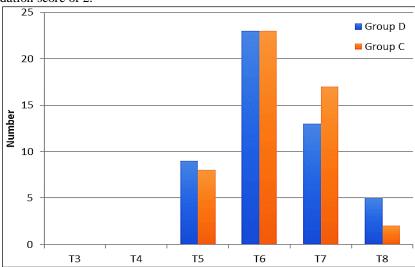
RESULTS

All the patients in group C and D were comparable demographically (age, sex, weight, height) (table 1).

Table 1: Demographic data

Parameters	Group $D(n = 50)$	Group $C(n = 50)$	P value
Age in years (mean+S.D.)	34.32 <u>+</u> 10.25	34.98 <u>+</u> 7.55	0.71
Weight in kg(mean+S.D.)	61.78 <u>+</u> 6.08	65.58 <u>+</u> 3.63	0.06
Height in cm(mean±S.D.)	161.66 <u>+</u> 4.77	163.46 <u>+</u> 4.82	0.064
Age (M/F)	36/14	32/18	

The sensory block duration was significantly higher in group D (137.6) than in group C (74.2), p=0.000, and the motor block in group D (178) was significantly higher as well than that in group C (129.6), p=0.000 (Graph 1). The time of request for rescue analgesia in the postoperative period was significantly extended in group D (236.24 \pm 45.91) when compared to group C (145.80 \pm 22.50), p=0.000. The Ramsay sedation score of the patients in group D was significantly high-82% of the patients had a sedation score of 3 whereas all the patients in group C had Ramsay sedation score of 2.

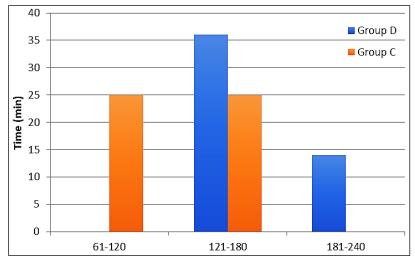


Graph 1: Level of Sensory Block

Table 2: Time for two dermatome regression of sensory blockade

der matome regression or sensory brockade							
Category	N T	Duration of sensory block in min		4			
	I	Mean	SD	ι	P		
Group D	50	137.6	21.14	20.27	0.000		
Group C	50	74.2	6.09	20.37	0.000		

In group D 58% of patients had sensory block lasting for 121-180 minutes, 38% had for 61-120 minutes and for 4% the sensory block lasted for up to 180 minutes. Whereas in group C, all the patients had sensory block lasting for not more than 120 minutes before the initiation of dermatome level regression. From the observations of the mean values, sensory block duration was significantly higher in group D (137.6) than in group C (74.2), p=0.00. (Table 2)



Graph 2: Duration of Motor Nerve Block

DISCUSSION

In this study, the mean baseline heart rate was 78.40±10.06 in group D and 77.58±10.11 in group C. Grewal A *et al.* ^[6] conducted a similar study in which the lowest heart rate noted was 61.7±6.2 in Dexmedetomidine group and 63.1±7.1 in normal saline group, which was statistically comparable. The incidence of bradycardia requiring management with atropine was relatively higher in group D (23.3%) than in group P (20.0%); but this difference was statistically not significant (p=0.754).

The fall in MAP was steeper in group C up to 90 minutes following spinal anaesthesia, lowest reading was at 60 minutes-81.96±7.84and that in group D was 83.70±8.13, p=0.02 which makes the difference statistically significant. At 135 minutes the mean MAP of group D was 80.86±6.91 and that in group C was 91.62±8.69 which indicates a steep rise in MAP in group C, p=0.000. In our study the need for ephedrine following fall in blood pressure had comparable levels in both the groups.

In our study majority of the patients attained a highest level of block at T6 in both the groups. Kaya FN et al. [7] in their study recorded the highest level of sensory significantly higher (p < 0.001)Dexmedetomidine group of patients (T4.6±0.6) than in saline group of patients (T6.4±0.8). The motor block duration was significantly prolonged in group D than in the control group. Kanazi GE et al. [8] demonstrated that combination of 12 mg of bupivacaine with a low dose (3 mcg) Dexmedetomidine or 30 mcg of clonidine administered intrathecal significantly reduced the onset of motor block and extended both motor and sensory block when compared with bupivacaine alone. Mustafa MM et al. [9] concluded that intravenous Dexmedetomidine prolonged the sensory and motor blocks of bupivacaine spinal analgesia with excellent sedation and hemodynamic stability. Considering the present as well as the earlier studies it can be concluded that the effect of Dexmedetomidine on duration of spinal and motor blockade is independent on route of administration Dexmedetomidine. Choudhary AK et al. [10]concluded in a study done in 2022 that IV infusion of dexmedetomidine 1 $\mu g/kg$ body weight prior to SAB can be recommended to achieve better sensory blockade and adequate hemodynamic stability and sedation.

In our study, 50% of patients in group D requested their first analgesia between 181-240 minutes after surgeryand 40% requested it between 241-300 minutes, whereas in group C, 70% of patients needed rescue analgesia between 121-180 minutes and 16.67% between 181-240 minutes. In fewer than 240 minutes, every patient in group C needed rescue analgesia, whereas 6.67% of patients in group D didn't ask for it until 300 minutes had passed, p=0.001. These results are consistent with those of a related study that was done to by Vimal H Patel *et al.* prove

that intravenous Dexmedetomidine and not midazolam prolongs bupivacaine spinal anesthesia.

CONCLUSION

We concluded that intravenous Dexmedetomidine offer a clinical advantage as an adjunct to bupivacaine spinal anaesthesia, where a longer duration of anaesthesia is called for without the use of higher dose of local anaesthetic and to reduce the analgesic requirement. Furthermore, it maintained a steady hemodynamic profile throughout the intraoperative period and offered arousable sedation without respiratory depression.

REFERENCES

- Scheinin M, Schwinn D. The locus coeruleus: site of hypnotic actions of α₂-adrenoceptor agonists Anesthesiology.1992;76:873-5.
- Goodman and Gilman. The pharmacological basis of therapeutics.11th Edition. McGraw Hill publishing division (United States of America); c2006. p. 204-214.
- Panzer O, Moitra V, Sladen RN. Pharmacology of Sedative-Analgesic Agents: Dexmedetomidine, Remifentanil, Ketamine, Volatile Anesthetics, and the Role of Peripheral μ Antagonists. Crit. CareClin. 2009;25(3):451-69.
- Ejnar Eriksson. Illustrated Handbook in localanesthesia.2nd Edition. Lloyd-Luke(London UK); c1979. p. 13.
- AitkenheadAR,SmithG.Text book of Anesthesia. 3rd Edition. Churchill livingstone(United States of America); c1996. p. 435-444.
- Grewal A. Dexmedetomidine: New avenues. J Anaesthesiol. ClinPharmacol. 2011;27:297-302.
- Kaya FN, YavascaogluB, Turker G, Yildirim A, Gurbet A, Mogol EB, et al. Intravenous dexmedetomidine, but not midazolam, prolongs bupivacaine spinal anesthesia. Can J Anaesth. 2010;57(1):39-45.
- Kanazi GE, Aouad MT, Jabbour-Khoury SI, Al Jazzar MD, AlameddineMM, Al-Yaman R, et al. Effect of low-dose dexmedetomidine or clonidine on the characteristics of bupivacaine spinal block. Acta Anaesthesiol Scand.2006;50(2):222-7.
- 9. Al-Mustafa MM, Badran IZ, Abu-Ali HM, Al-Barazangi BA, Massad IM, Al-Ghanem SM. Intravenous dexmedetomidine prolongs bupivacaine spinal analgesia. Middle East J Anesthesiol. 2009;20(2):225-31.
- Choudhary AK, Prasad MK, Keshri R, Choudhary S. Effects of IV dexmedetomidine as a pre-medication on clinical profile of bupivacaine spinal Anaesthesia in lower abdominal surgeries: a randomized clinical study. Pan Afr. Med J. 2022 Jan;41:74.
- Vimal H Patel, Harsha R Patel. A comparison between Dexmedetomidine an midazolam infusion on characteristic of spinal anaesthesia. IJMSPH. 2016;5:906-910.