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Randomized Control Trial Using Bupivacaine in Spinal Anaesthesia with and without Intravenous Dexmedetomidine in Lower Abdominal Surgeries

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Abstract

Introduction: Commonly we use 0.5% hyperbaric bupivacaine in spinal anaesthesia. Adjuvants to spinal anaesthesia have been used to improve quality of analgesia and in prolongation of anaesthetic duration. Dexmedetomidine has been studied and shown to have synergism with bupivacaine and other local anesthetics. Recently, in a few studies, intravenous (IV) dexmedetomidine has been shown to improve analgesic quality. In this study we aimed at finding the efficacy of (IV) dexmedetomidine in improving the analgesia quality and duration of subarachnoid blockade in our hospital scenario. *Materials and Methods:* Ninety patients were divided into two groups of 45 each. In Group A - 3.5 mL of 0.5% hyperbaric bupivacaine was used for spinal anesthesia. In Group B - 3.5 mL 0.5% hyperbaric bupivacaine used for spinal anaesthesia, thirty minutes later a loading dose of IV dexmedetomidine 1 mcg/Kg was infused over 30 min followed by maintenance dose of 0.3 mcg/kg/hr IV dexmedetomidine infused till the end of surgery. In Group A, isotonic saline was used instead of dexmedetomidine preparation. Duration of motor block, sensory block, analgesia, hemodynamic changes, sedation levels, complications and side effects were noted and compared between the study groups in patients undergoing lower abdominal surgeries. *Results:* The duration of motor block in Group A was 149.38 ± 21.32 minutes vs.189.13 ± 31.18 minutes in Group B ($p< 0.05$), duration of sensory block in Group A was 166.79 ± 33.12 minutes vs. 248.13 ± 48.32 minutes in Group B ($p< 0.05$), and duration of analgesia in Group A was 198.69 ± 41.38 minutes vs. 298.57 ± 34.65 minutes in Group B ($p< 0.05$). *Conclusion:* Use of IV dexmedetomidine improves analgesia quality and prolongs anaesthesia duration in subarachnoid block with 0.5% hyperbaric bupivacaine without any hemodynamic instability and with optimum sedation.

Keywords: Spinal anaesthesia; Bupivacaine; Dexmedetomidine; Postoperative analgesia.

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Introduction

Spinal anaesthesia is a very common procedure carried out in the operation theatre and is accomplished by injecting local anaesthetic solution into the cerebrospinal fluid in the region of lower lumbar intervertebral spaces which creates an intense sensory, motor and sympathetic block and provides excellent operating conditions for surgeries below the umbilicus. Spinal anaesthesia also provides good operating conditions for lower abdominal and lower limb surgeries [1]. However; one of the major limitations is the anesthesia duration in subarachnoid block. To

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overcome this, many additives and adjuvants have been tried. Intrathecal adjuvants like morphine, fentanyl, sufentanyl, neostigmine, ketamine, midazolam, magnesium sulphate, clonidine and dexmedetomidine, have been used to improve analgesia quality and anaesthesia duration in spinal anaesthesia [2-13].

Use of IV dexmedetomidine premedication in general anaesthesia has been shown to provide sedation preoperatively, reduces intraoperative inhalational anaesthetic requirements, intraoperative and postoperative analgesia with good hemodynamic stability [14]. In central nervous system highest number of alpha2 adrenoreceptor receptors are present in locus ceruleus, presynaptic activation of theses in locus ceruleus leads to inhibition of noradrenaline release resulting hypnotic and sedative effects. In the spinal cord, activation of $alpha_2$ adrenoreceptor receptors at substantia gelatinosa leads to inhibition of nociception and release of substance P. [15].

Materials and Methods

Ethical committee approval was taken. After written informed consent, ninety patients between 20 and 60 years of age, of ASA Class I and II, scheduled for elective lower abdominal surgery were enrolled in the study. Computer based randomization was done. Investigator and the patient were blinded to the study. Infection at the site of spinal anaesthesia, patients with uncontrolled hypertension and diabetes, any neurological or psychiatric diseases and patients with bleeding or coagulation disorders were excluded from the study.

Preoperatively all study patients were advised 8 hours nil by mouth. All patients received Tab. Ranitidine 150 mg orally on the night before surgery at 10 pm with a sip of water as premedication. The patients were transferred to the operation theatre at 8.30 AM. Intravenous access was achieved with 18G cannula. All patients were preloaded with Ringer's Lactate 10 mL/Kg, 15 minutes prior to the surgery. In operating theatre, standard monitoring viz. oxygen saturation (SpO_2) heart rate (HR), non – invasive blood pressure (NIBP), electrocardiogram (ECG) were attached and baseline hemodynamic parameters were recorded. Under aseptic precautions, using 25G Quincke spinal needle, subarachnoid block was performed at L_3–L_4 inter-space in the midline with 0.5% hyperbaric bupivacaine (Neon Pharmaceuticals, India) was administered at the rate of 0.2 mL/sec.

Group A received 3.5 mL of 0.5% hyperbaric bupivacaine and normal saline infusion. Group B received 3.5 mL 0.5 % hyperbaric bupivacaine for spinal anaesthesia, thirty minutes later loading dose of dexmedetomidine 1 mcg/Kg was infused over 30 min followed by maintenance dose of 0.3 mcg/kg/hr IV dexmedetomidine infused till the end of surgery (AKAS Syringe Pump). SpO_2, HR, Systolic blood pressure (SBP), Diastolic blood pressure (DBP) and Mean arterial pressure (MAP) were recorded preoperatively and after performing the subarachnoid block, every 5 minutes till the end of surgery.

Modified Bromage Scale was used to assess level of motor block. Time taken for regression of motor block to Modified Bromage Scale 1 was considered. Using pinprick bilaterally at mid – clavicular line, time of onset of sensory block, level of sensory block and sensory block duration were recorded. Time taken to reach L5/S1 dermatome was considered as recovery time for sensory block. Postoperatively, the Modified Bromage Scale and the sensory level were recorded every 15 minutes till the patients were discharged from the postanaesthesia care unit. The level of pain was assessed by The Visual Analog Scale (VAS). VAS greater than 4 was considered as cut off point to treat pain. IV Paracetamol 1 gram was considered for rescue analgesia [16,17]. Level of sedation was assessed by The Ramsay Sedation Score. Score greater than 4 was considered as excessive sedation.

Any decrease in MAP of 20% from the baseline was treated with bolus dose of 6 mg IV ephedrine and infusion of intravenous fluids. HR less than 50/min was treated with IV 0.6 mg atropine. The baseline, intraoperative and postoperative hemodynamic changes at various time intervals were compared between the study groups using *Chi square test* and *unpaired t test*. Data validation and analysis was carried out by SPSS Version 11.0. All the p values < 0.05 were considered significant statistically.

Results

The study groups were comparable in terms of demographic data (Table 1), baseline hemodynamic parameters (Table 2) and mean duration of surgery (Table 3). Both the duration of motor block and sensory block were prolonged in Group B compared to Group A ($p < 0.001$) (Table 4). The two segment regression in Group A was 87.9 ± 9.64 minutes whereas in Group B it was 119.0 ± 11.79 minutes ($p < 0.001$) (Table 5). The time taken for rescue

analgesia was prolonged in Group B compared to Group A ($p < 0.001$) (Table 6).

In Group A, the mean sedation score was 2 at the beginning of postoperative period and 1 at the end of 90 minutes whereas in Group B, the mean sedation score was 2.18 at the beginning of postoperative period and 2.08 at 90 minutes. The Ramsay sedation score was higher in Group B ($p < 0.05$). However, respiratory depression was not observed in any of the patients of either group.

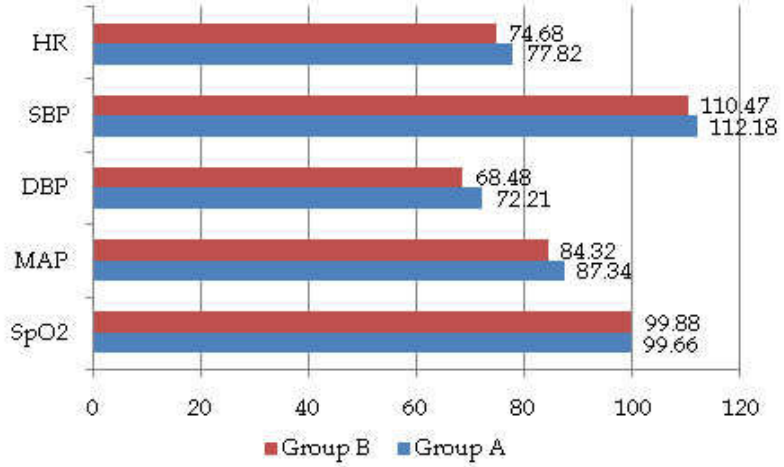
In Group A, the VAS score was 2.13 at the beginning of post – operative period and gradually increased to 4.93 at 90 minutes whereas in Group B, the VAS score was 0.71 at the beginning of post – operative period and 2.97 at 90 minutes. The pain scores were higher in Group A ($p < 0.05$). Hence, it is evident from the above observations that intravenous dexmedetomidine provides adequate sedation and analgesia even in the post – operative period without causing any respiratory depression.

Intraoperatively, 9 patients had bradycardia and hypotension in 13 patients in Group A, whereas in Group B, 4 patients had bradycardia and 4 patients had hypotension. The two groups did not differ significantly with respect to intraoperative hemodynamics at any interval of time and SpO_2 at any interval of time ($p > 0.05$) (Graph 1).

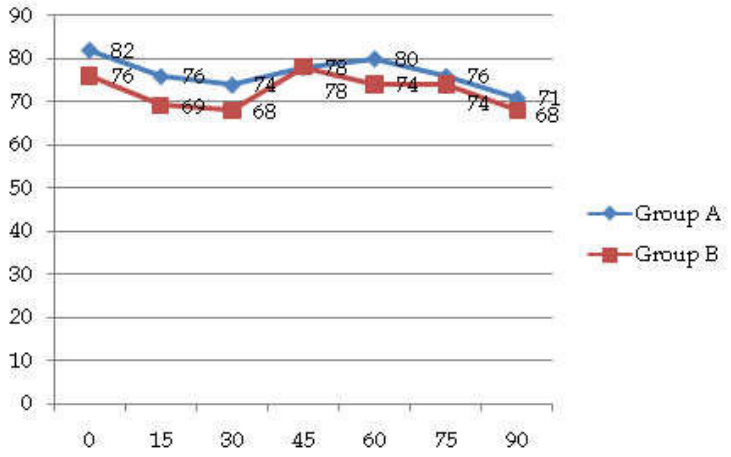
In Group A, 1(2%) patient had vomiting whereas in Group B, none were observed. It was treated with IV Ondansetron 4 mg. In Group A, 1(2%) patient experienced shivering in the postoperative period. It was treated with IV Pheniramine Maleate 45.5 mg whereas in Group B, none was observed.

Table 1: Demographic data

Parameter	Group A	Group B	p value
Age (Years)	45.44 ± 7.60	46.20 ± 8.36	0.658
BMI (kg/m^2)	20.44 ± 1.82	19.98 ± 2.01	0.269
Sex (Male/Female)	14:31	13:32	



Graph 1: Intraoperative Hemodynamics at various intervals



Graph 2: Postoperative HR at various intervals

Table 2: Baseline Hemodynamic Parameters

Parameter	Group A	Group B	p Value
HR	81.88	86.08	<0.05
SBP	129.73	127.07	<0.05
DBP	79.75	81.44	<0.05
MAP	98.33	97.17	<0.05
SpO_2	100	99.88	<0.05

Table 3: Duration of Surgery

Group A	Group B	p value
97.11 ± 24.79	97.44 ± 26.19	0.95

Table 4: Comparison of sensory and motor blockade

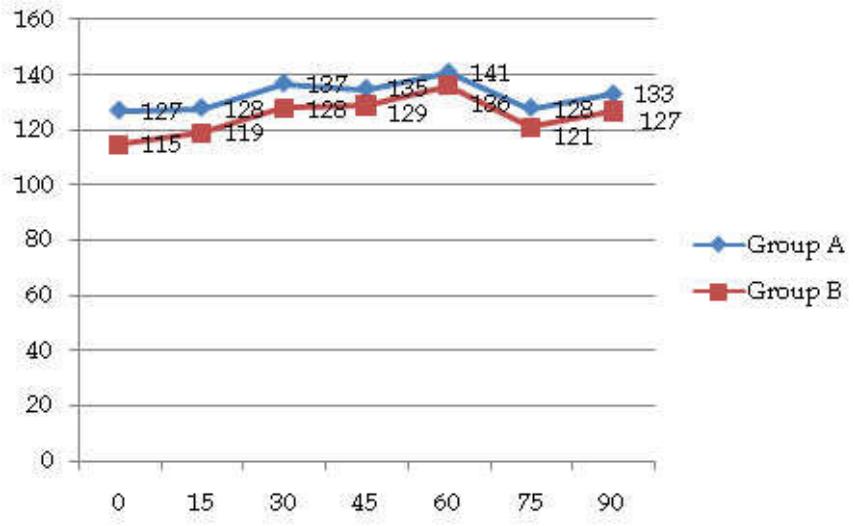
Parameter	Group A	Group B	p value
Sensory Blockade	166.79 ± 33.12	248.13 ± 48.32	< 0.001
Motor Blockade	149.38 ± 21.32	189.13 ± 31.18	< 0.001

Table 5: Two segment regression

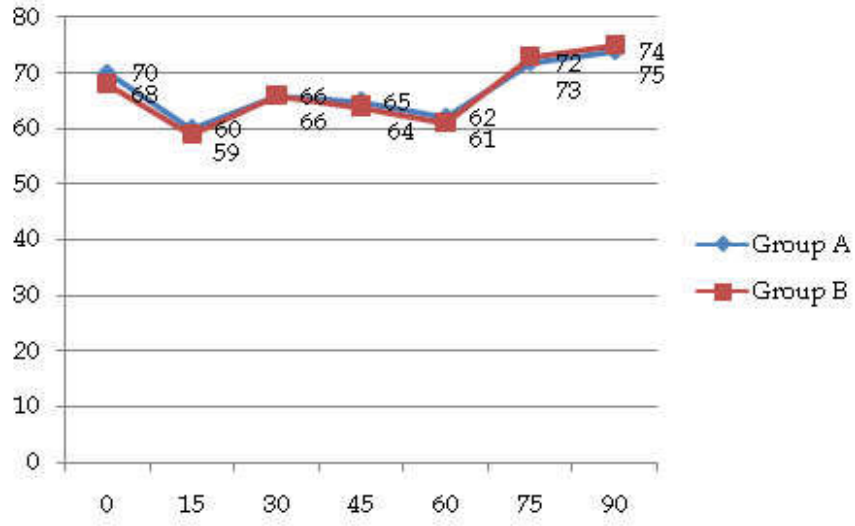
Group A	Group B	p value
87.9 ± 9.64	119.0 ± 11.79	< 0.001

Table 6: Rescue Analgesia

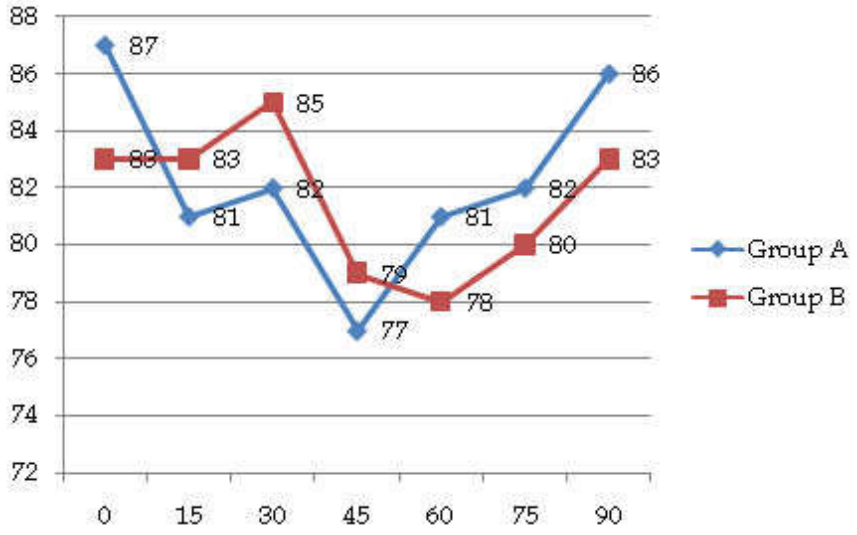
Group A	Group B	p Value
198.69 ± 41.38	298.57 ± 34.65	< 0.001



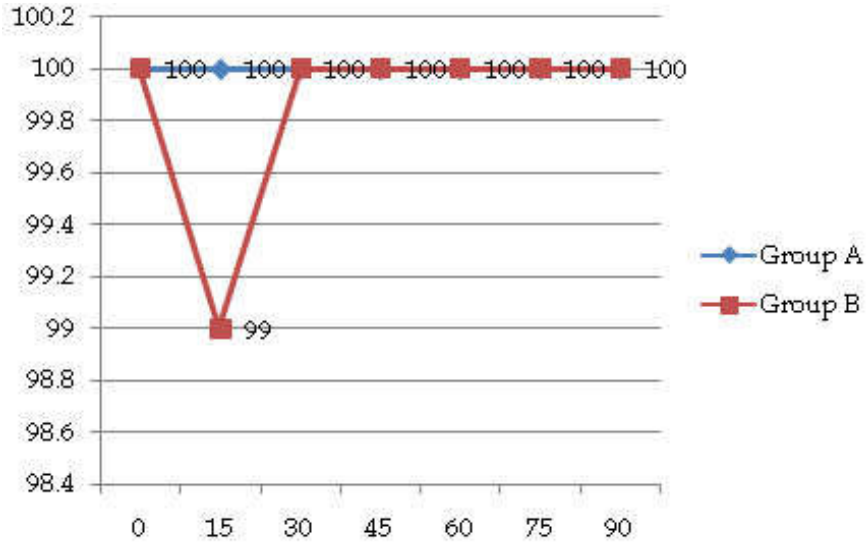
Graph 3: Postoperative SBP at various intervals



Graph 4: Postoperative DBP at various intervals



Graph 5: Postoperative MAP at various intervals



Graph 6: Postoperative SpO_2 at various intervals

Discussion

Dexmedetomidine is a boon in the present day practice of anaesthesia. It has wonderful properties such as hemodynamic stability, sedation, anxiolysis and reduced postoperative requirements of parenteral analgesics owing to its supraspinal action. It reduces the release of neurotransmitters and hyperpolarization of neurons. Neuroprotection is another added benefit. Dexmedetomidine mainly acts on the locus ceruleus by causing disinhibition of nociception. It reduces the release of noradrenaline and blocks its sympathetic activity. In the intensive care it is blessing in disguise owing to its affinity for $alpha_2$ receptors and routinely used by many clinicians.

In a study done by Mahmoud M al Mustafa showed that when dexmedetomidine was administered there was statistically significant prolongation of sensory and motor blockade (p <0.05) [18]. Similarly, SS Harsoor et al. concluded that the duration of effective analgesia was prolonged in subjects who received dexmedetomidine intravenously (p < 0.001) [19]. First analgesic requirement was increased by more than 50% in a clinical study done by Abdallah FW

($p < 0.00001$) [20]. In a clinical study done by Reddy VS showed that the level of sensory blockade level was greater with dexmedetomidine and also the time to first analgesic requirement was higher ($p < 0.0001$) [21]. The 2 segment regression of sensory blockade was increased by 42.33 minutes in a clinical study by Jyotsana Kubre and the duration of analgesia was increased by 70.50 minutes in the group of subjects who received dexmedetomidine ($p < 0.05$) [22]. The duration of analgesia was prolonged levobupivacaine was administered caudally along with concurrent dexmedetomidine intravenously in the study done by Yao Y [23]. In a clinical study conducted by Vatsalya T concluded that dexmedetomidine shortened the time taken to reach dermatome level of T10 along with significant prolongation the duration of regression of sensory and motor blockade. Analgesia was prolonged significantly by 34.38 minutes ($p = 0.0001$). The profile of side effects and its treatment were comparable with incidences of bradycardia and hypotension [24]. In a study done by Kavya UR showed that sensory block was significantly higher by 83.4 minutes. Motor blockade recovery also showed significant prolongation by 88.2 minutes. Subjects who received dexmedetomidine were adequately sedated and were easily arousable [25]. Hemodynamic responses are in direction relation to dose and speed of infusion of dexmedetomidine. Transient hypertension with reflex bradycardia is often followed by hypotension when dexmedetomidine is infused rapidly.

Conclusion

Dexmedetomidine when infused in the loading dose of 1 mcg/kg followed by 0.3 mcg/kg/hr prolongs the action of hyperbaric bupivacaine in subarachnoid block along with improved quality of analgesia, adequate sedation and hemodynamic stability.

Conflicts of interest: None

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