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Research Article

EFFECT OF ADDITION OF DEXMEDETOMIDINE TO BUPIVAICAINE VERSUS BUPIVAICAINE ALONE IN SPINAL ANESTHESIA FOR ELECTIVE LOWER LIMB SURGERY

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Abstract
Background : Intrathecal α2 agonists prolong the duration of action of local anaesthetics and reduce the required dose. Dexmedetomidine is a α2 receptor agonist and its α2/α1 selectivity is eight times higher than that of clonidine
Aim : To assess the onset and duration of sensory and motor block as well as operative analgesia of dexmedetomidine given intrathecally with hyperbaric 0.5% bupivacaine or hyperbaric 0.5% bupivacaine alone for spinal anaesthesia Methodology : A prospective randomized study was done comprising of 100 patients who were undergoing elective lower abdominal surgery at Vinayaka Missions KirupanandaVariyar Medical College\ and Hospitals, Salem, Tamil Nadu . Using the sealed envelope method, the patients were randomly allocated into two groups: Group A (n=50) or Group B (n=50). The group A patients received Hyperbaric Bupivacine 0.5% + Normal saline and group B patients were given 0.5% Bupivicaine 15mg + 5mcg Dexmedetomidine. Standard monitoring was continued throughout the operation. Sensory blockade was assessed by using pinprick test on each side of the midclavicular line; motor blockade was assessed based on a modified Bromage scale. Results : The time to sensory block to reach T10 dermatome, the time for highest level of sensory block and the time for onset of motor block were comparatively less among the patients who had received 0.5% Bupivicaine 15mg + 5mcg Dexmedetomidine (group B) than that of the patients who received hyperbaric Bupivacine 0.5% + Normal saline (group A) and this difference was found to be statistically significant. Similarly the time for sensory regression and the time for motor regression to bromage 0 was longer in group B than group A and it was statistically significant ($p<.05$).
Conclusion : Combined use of dexmedetomidine and bupivacaine in spinal anaesthesia prolongs sensory and motor block durations
Keywords: dexmedetomidine, bupivacaine, spinal anaesthesia

INTRODUCTION
Spinal anaesthesia is the most commonly used technique for lower limb surgeries as it is very economical and easy to administer. However, postoperative pain control is a major problem because spinal anaesthesia using only local anaesthetics is associated with relatively short duration of action, and thus early analgesic intervention is needed in the postoperative period. A number of adjuvants, such as clonidine and midazolam, and others have been studied to prolong the effect of spinal anaesthesia [1,2]. A common problem during lower limb surgeries under spinal anaesthesia is visceral pain, nausea, and vomiting[3,4]. While spinal anaesthesia has many advantages, the limited duration of action appears to be one of its drawbacks. Intrathecal α2 agonists prolong the duration of action of local anaesthetics and reduce the required dose. The intrathecal use of clonidine, a partial α2 adrenoceptor agonist, has been shown as an effective

and safe procedure[5,6]. Dexmedetomidine is a α2 receptor agonist and its α2/α1 selectivity is eight times higher than that of clonidine. In animal models, intrathecal dexmedetomidine has been demonstrated to have an analgesic effect[7,8].

Dexmedetomidine, a new highly selective α2-agonist, is under evaluation as a neuraxial adjuvant as it provides stable hemodynamic conditions, good quality of intraoperative and prolonged postoperative analgesia with minimal side effects[4]. Dexmedetomidine has been approved by Food and Drug Administration (FDA) as a short-term sedative for mechanically ventilated intensive care unit (ICU) patients. Based on earlier human studies, it is hypothesized that intrathecal 5 µg dexmedetomidine would produce more postoperative analgesic effect with hyperbaric bupivacaine in spinal anaesthesia with minimal side effects[9,10].

AIM :

To assess the onset and duration of sensory and motor block as well as postoperative analgesia of dexmedetomidine given intrathecally with hyperbaric 0.5% bupivacaine or hyperbaric 0.5% bupivacaine alone for spinal anaesthesia

METHODOLOGY :

A prospective randomized study was done comprising of 100 patients who were undergoing elective lower abdominal surgery at Vinayaka Mission superspeciality Hospitals, Salem, Tamil Nadu . Using the sealed envelope method, the patients were randomly allocated into two groups: Group A (n=50) or Group B (n=50). Patients on drugs such as alpha 2 blockers, calcium channel blockers, beta blockers, or any antiarrythmic drugs were excluded.Patients with body weight more than 120 kgs, height less than150 cms, history of allergy to study drugs, pregnancy , and patients with coagulopathy were excluded from the study.

All the patients were preloaded with 20ml/kg Ringer lactate solution via 18G IV cannula in the dorsum of the hand. Standard anaethesia monitoring was used, including non invasive blood pressure, heart rate, electrocardiography and pulse oximetry. Patients motor power and sensations to cold using alcohol solution were examined. With the patient in sitting position, spinal anaesthesia was performed at the level of L4-L5 through midline approach using 25G quinckie needle with the bevel of the needle pointing upwards. The group A patients received Hyperbaric Bupivacine 0.5% + Normal saline and group B patients were given 0.5% Bupivicaine 15mg + 5mcg Dexmedetomidine. Standard monitoring was continued throughout the operation. Sensory blockade was assessed by using pinprick test on each side of the midclavicular line; motor blockade was assessed based on a modified Bromage scale (5) (0= free movement of legs and feet, 1= just able to flex knees with free movement of feet, 2= unable to flex knees, but with free movement of feet, 3= unable to move legs or feet). The sensory level and Bromage scale were recorded intra-operatively every 5 min for a period of 30 mins, at the end of the surgery and in the Post-Anaesthesia Care Unit (PACU) every 15 min until the patient was discharged from PACU by an anaesthesiologist who was blinded to group allocation.Time of onset of sensory block and time taken to reach T10 dermatome, the highest dermatomal level, planned for surgery and time taken for complete regression of sensory block and time of onset of motor block and time taken to reach Bromage 3 and regression to Bromage 0 times were recorded. A decrease >20% from baseline, or to <90 mmHg in systolic blood pressure, was defined as hypotension and was treated with incremental doses of 5 mg intravenous ephedrine. Bradycardia was defined as heart rate <50 beats/min and was treated with atropine. Intraoperative analgesic

requirement, intraoperative and postoperative nausea and vomiting and other side effects were recorded. The heart rate, blood pressure, oxygen saturation, sedation score, VISUAL ANALOGUE SCALE (VAS) at rest and with movements was recorded during the first hour at 15, 30,45 and 60 minutes and thereafter every hour upto 8 hours, 12 hours and 24 hours after spinal injection. Intergroup comparison of demographic data, durations of sensory and motor blocks, mean arterial pressure, and mean heart rate values were carried out by Student's t test.

RESULTS :

Table 1 : Demographic data among the study subjects

Demographic characters of the patients	Group A Mean (SD)	Group B Mean (SD)	P value
Age in years	40.32 (12)	45.4 (12.7)	0.446
Weight (kgs)	54 (10.1)	56.8 (8.2)	0.519
Height (cms)	169.8 (7.4)	165.6 (8.1)	0.440

P value was calculated using student T test

Table 1 shows the mean and standard deviation of various demographic parameters measured in the patients. Age, weight and height was almost similar in both the groups without showing any statistical significant difference.

Table 2 : Sensory and motor block characteristics among the study patients

Sensory and motor block characteristics	Group A Mean (SD)	Group B Mean (SD)	P value
Time to sensory block to reach T10 dermatome (min)	4.60 (0.70)	2.07 (0.47)	0.034
Highest level of sensory block (T) (min)	5.3 (0.82)	2.96 (0.65)	0.02
Time to onset of complete motor block (Bromage=3) (min)	8.45 (5.3)	6.42 (4.6)	0.046
Time to sensory regression to S1 segment (min)	167 (18.72)	263 (19.78)	0.001
Time to motor block regression to Bromage 0	141.56 (15.29)	229.98 (14.26)	0.001
Duration of analgesia (mins)	221 (19.62)	368 (17.63)	0.0001

P value was calculated using student T test

Table 2 shows the mean and standard deviation of the sensory and motor block characteristics among the two groups of patients. From the table it is inferred that the time for sensory block to reach T10 dermatome, the time for highest level of sensory block and the time for onset of motor block were comparatively less among the patients who had received 0.5% Bupivicaine 15mg + 5mcg Dexmedetomidine (group B) than that of the patients who received hyperbaric Bupivacine 0.5% + Normal saline (group A) and this difference was found to be statistically significant. Similarly the time for sensory regression and the time for motor regression to bromage 0 was longer in group B than group A and it was statistically significant ($p<.05$).

Table 3 : Adverse events occurred in the patients following spinal anesthesia

Adverse effects	Group A (n=50)	Group B (n=50)	P value
Hypotension	2	4	0.265
Bradycardia	3	5	0.378
Nausea	18	21	0.472
Vomiting	7	11	0.621

P value calculated using chisquare test (Fischer exact test)

Table 3 shows the various adverse events occurred among both the groups. It is seen form the table that various adverse events like hypotension, bradycardia, nausea and vomiting had occurred in almost equal numbers in both the groups and there was no significant difference between them ($p>.05$).

DISCUSSION :

In this study, we observed that adding dexmedetomidine to bupivacaine prolonged the sensory and motor block duration in patients subjected to lower limb surgery under spinal anaesthesia. Although the mechanism is unclear, α2 adrenoceptor agonists have been observed to extend the sensory and motor block durations of local anaesthetics. They act by binding to presynaptic C-fibers and postsynaptic dorsal horn neurons. Their analgesic action is a result of depression of the release of C-fibre transmitters and hyperpolarisation of postsynaptic dorsal horn neurons[11]. Local anaesthetic agents act by blocking sodium channels. The prolongation of effect may result from synergism between local anaesthetic and α2 -adrenoceptor agonist, while the prolongation of the motor block of spinal anesthetics may result from the binding of α2 -adrenoceptor agonists to motor neurons in the dorsal horn[12]. Fukushima *et al* administered 2 µg/kg epidural dexmedetomidine for postoperative analgesia in humans but did not report neurologic deficits [13]. Our study has shown that the addition of 5 µg dexmedetomidine with hyperbaric bupivacaine significantly prolongs both sensory and motor block. Al-Ghanem et al had studied the effect of addition of 5 µg dexmedetomidine or 25 µg fentanyl intrathecal to 10 mg isobaric bupivacaine in vaginal hysterectomy and concluded that 5 µg dexmedetomidine produces more prolonged motor and sensory block as compared with 25 µg fentanyl[14].

The onset of sensory and motor block in our study was comparable with the studies done by Shukla et al[15] and Shaikh and Kiran[16] for dexmedetomidine and buprenorphine respectively. Intravenous dexmedetomidine has anti shivering effect [17]. However, we encountered almost equal incidence of shivering in both the groups which implies that intrathecal dexmedetomidine

has no effect on shivering. The duration of analgesia in the dexmedetomidine group in the present study was 368 minutes. This is in agreement with studies done by Shah etal[18] and Gupta etal[19]. Study done by Eid etal[20] showed that the duration of analgesia with dexmedetomidine is proportional to its dose. A meta-analysis of use of dexmedetomidine in regional anaesthesia states that the sensory duration, motor blockade and request for rescue analgesia is prolonged in dexmedetomidine group[21].

In the study of Strebel etal[22] on orthopaedic cases, using clonidine at a dose below 150 µg in combination with isobaric bupivacaine in a dose-dependent fashion was shown to provide significantly prolonged duration of spinal anaesthesia and analgesia without disrupting the haemodynamic stability and inducing sedation. Santiveri etal[23] used 75µg clonidine to prilocaine in patients undergoing transurethral resection of bladder tumours under spinal anaesthesia and reported prolonged sensory and motor blocks along with reduced postoperative analgesic requirement. The quality of anaesthesia was lower in the ropivacaine-only group, the quality of intraoperative analgesia was elevated while motor and sensory block durations were unchanged in the 15µg clonidine group, and sensory block duration was prolonged in the 75µg clonidine group. Kanazi etal[24] reported that the addition of dexmedetomidine (3µg) or clonidine (30µg) to bupivacaine in spinal block shortens the time to onset of motor block and extends the sensory and motor block durations and the results were almost in par with the present study.

Niemi et al[25] reported that the addition of clonidine to bupivacaine in spinal anaesthesia prolonged block duration, but decreased the mean blood pressure and heart rate significantly compared with the control group. Kanazi et al[24] noted that dexmedetomidine or clonidine added to intrathecal bupivacaine did not cause a significant reduction in blood pressure. In the study of Gupta etal[19], hypotension was more severe in the dexmedetomidine group than in the fentanyl group, but no statistically significant difference was determined between the groups. In this study, we observed hypotension and bradycardia almost in equal numbers in both the groups.

CONCLUSION :

Combined use of dexmedetomidine and bupivacaine in spinal anaesthesia prolongs sensory and motor block durations as well as it provides good quality of intraoperative analgesia, hemodynamically stable conditions, minimal side effects, and excellent quality of postoperative analgesia.

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