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Original Article

Dexmedetomidine 5 Mcg and Clonidine 75 Mcg Comparison when Added to 12.5 Mg of 0.5% Heavy Bupivacaine for Spinal Anaesthesia in Lower Abdominal Surgeries

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Abstract

Background: Spinal anesthesia is commonly used for lower abdominal surgeries. Various adjuvants have been used to prolong the analgesic effect of bupivacaine.

Materials and Methods: A total of 50 adult patients of American Society of Anesthesiology Grade I-II were divided into two groups randomly in a group of 25 each. Groups clonidine (A), and dexmedetomidine (B) had given hyperbaric bupivacaine 0.5% 2.5 ml intrathecally with clonidine 75 μg and dexmedetomidine 5 μg, respectively.

Results: Motor block was delayed with Group A as compared to Group B. The difference was statistically insignificant (192.13 ± 97.04 s in Group B vs. 172.85 ± 67.85 s in Group A, *P* = 0.001). Onset of sensory block was delayed with Group B as compared to Group A (83 ± 32.42 s in Group A vs. 115 ± 39.35 s in Group B. Regression time of sensory block was 374.34 ± 44.54 min for Group A as compared to 302.5 ± 29.18 min for Group B. Regression time to reach Bromage 1 was 317 ± 32 min for Group A as compared to 220 ± 48 min for Group B patients remained hemodynamically stable in both dexmedetomidine and clonidine groups.

Conclusion: Dexmedetomidine is better in terms of longer duration of action though both clonidine and dexmedetomidine prolonged the duration of sensory and motor block of bupivacaine.

Key words: Adrenoreceptor agonist, Dexmedetomidine, Spinal anesthesia

INTRODUCTION

An adjuvant (from Latin, adjuvare: To aid), is a pharmacological or immunological agent that modifies the effect of other agents, such as a drug or vaccine. Local anesthetics (LA) are the most common agents used for spinal anesthesia but have short duration of action.[1-3] Opioids are one of those commonly used as intrathecal adjuvants to prolong it in the post-operative period without significant motor or autonomic blockade. There are side effects such as nausea, vomiting, urinary retention, pruritus, and delayed respiratory depression. Because of these side effects, further research toward non-opioid analgesics with less serious side effects is been done.[4] α-2 adrenergic agonists like dexmedetomidine is an agonist with an affinity of 8 times greater than clonidine.[5]

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MATERIALS AND METHODS

A randomized double-blinded study was carried out in 50 American Society of Anesthesiology I and II patients age between 20 and 60 years. Approval of Ethics Committee and written informed consent from study participants was

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taken. Patients for lower abdominal surgeries were divided into two groups of twenty each, and they were placed in Group A or Group B.

Patients were shifted to operation theater. After that intravenous (IV) were fluids started. All essential parameters such as systolic and diastolic blood pressure (BP), SpO_2, respiratory rate (RR) and pulse rate (PR) was recorded. The time for intrathecal injection was considered as 0, and the following parameters were observed - onset of sensory blockade was taken as loss of sensation to temperature by spirit swab at L2 level. The onset of motor block was taken as Bromage scale 1. RR, sedation, and any other complications were observed.

The PR, systolic and diastolic BP, SpO_2, and RR were recorded preoperatively, 0 min, 5 min, 15 min, 30 min, 60 min, and 90 min and at the end of surgery. Hypotension was defined as fall in systolic BP >30% from baseline or mean arterial pressure <60 mmHg. This was managed with injection ephedrine 6 mg increments. Bradycardia was defined as heart rate (HR) <50/min, and this was managed with injection atropine 0.01 mg/kg IV respiratory depression defined as RR <8/min and or SpO_2 <85%. This was planned to be managed with bag and mask ventilation or intubation and invasive positive-pressure ventilation if necessary. Blood loss more than the allowable loss was replaced with blood. The occurrence of sedation was assessed using Ramsay sedation scale.

The patient was shifted to the recovery room after completion of surgery; the vital signs were recorded, every 30 min interval. Sensory and motor block assessments were done every 15 min till recovery of pinprick sensation to L1 and Bromage scale of 1, respectively. Patients were shifted to post-operative ward after complete resolution of motor blockade. In the recovery room, pain assessment using visual analog scale (VAS) was done every 15 min. At the end of surgery, the degree of pain was assessed using VAS scale till VAS score >4 was reached. Whenever the patient complained of pain and rescue analgesic injection diclofenac, 75 mg i.m was given. Duration of effective analgesia was defined as time interval between onset of subarachnoid block (SAB) and the time to reach VAS ≥4.

Patients were monitored for 24 h to detect the occurrence of side effects - respiratory depression, nausea, vomiting, dry mouth, and pruritus. Patients were also enquired about the occurrence of Transient neurological symptoms which was described as pain/paresthesia in the buttocks, legs or pain radiating to lower extremities after initial recovery from SAB within 72 h.

Table 1: Demographic and anesthetic parameters between two groups

Parameters	Group A (*n*=25)	Group B (*n*=25)	*P* value
Demographic data			
Age (years)	40.8±9.1	39.5±11.4	0.62
Sex (female/male)	12/18	13/17	1.00
Onset and duration of sensory and motor blockade			
Duration of surgery (min)	116±64.7	161±70	0.66
Onset of sensory blockade (s)	83±32.4	115±39.3	<0.0011
Onset of motor blockade (s)	192.13±97.04	141.7±51.7	0.01
Duration of analgesia (min)	374.34±44.5	302.5±29.1	<0.0001
Duration of motor block (min)	317±32	220±48	<0.0001
Maximum sensory level achieved	T6±1.2	T6±1.2	1.00

RESULTS

Statistical Analysis

All recorded data were entered using MS Excel software and analyzed using SPSS software for determining the statistical significance. The analysis of variance was used to study the significance of mean of various study parameters between the two groups. Student's *t*-test was used to compare the two groups on mean values of various parameters. The $P < 0.05$ is considered statistically significant.

Motor block was delayed with Group A as compared to Group B. The difference was statistically insignificant (192.13 ± 97.04 s in Group B vs. 172.85 ± 67.85 s in

Table 2: PR, RR, and mean arterial BP between two groups

Time (min)	PR			RR			Mean arterial BP	
	Group A	Group B	*P*	Group A	Group B	*P*	Group A	Group B
Pre-operative	81.7±18.6	75.3±10.3	0.10	14±1.0	14±1	>0.05	96.1±9.4	93.4±6.4
0	85.1±20.9	78.2±12	0.12	14±1.4	14±1.1	>0.05	110.2±11.6	92.3±9.6
5	77.6±22.7	67.2±9.8	0.02	14±0.8	14±1.2	>0.05	84.3±10.56	81.4±9.12
15	71.6±17.4	63.3±8.9	0.01	14±0.8	14±1	>0.05	80.1±12.11	77.6±9.23
30	69.7±15.7	61.3±8.0	0.009	13±0.8	14±1	>0.05	80.8±9.7	78.9±10.4
60	66.1±14.2	62.5±7.5	0.16	13±0.6	13±0.9	>0.05	76.4±7.7	80.6±9.67
90	65.6±14.5	62.3±7.1	0.22	13±0.8	13±0.9	>0.05	78.6±8.78	80.2±9.9
EOS	68.9±11.8	63.5±7.4	0.03	13±0.7	13±0.9	>0.05	79.7±11.21	81.3±9.0

PR: Pulse rate, RR: Respiratory rate, BP: Blood pressure

Group A, $P = 0.001$). Onset of sensory block was delayed with Group B as compared to Group A (83 ± 32.42 s in Group A vs. 115 ± 39.35 s in Group B. Regression time of sensory block was 374.34 ± 44.54 min for Group A as compared to 302.5 ± 29.18 min for Group B. Regression time to reach Bromage 1 was 317 ± 32 min for Group A as compared to 220 ± 48 min for Group B patients remained hemodynamically stable in both dexmedetomidine and clonidine groups (Tables 1 and 2).

DISCUSSION

Dexmedetomidine and clonidine are alpha-2 adrenoceptor agonist agents initially prescribed for hypertension and IV sedation. Gradually, the role of these two agents extended beyond wards to operation theater for the provision of intraoperative and post-operative analgesia and sedation. Although there are sufficient studies on addition of clonidine to LA both epidurally and intrathecally, intrathecal and epidural characteristics of dexmedetomidine have been studied mainly in animals, and there is a scarcity of literature about intrathecal use of dexmedetomidine in humans.

When we compared the dexmedetomidine and clonidine with each other, we found that onset of motor block was delayed with dexmedetomidine as compared to clonidine. The onset of sensory block was delayed with clonidine as compared to dexmedetomidine. Dexmedetomidine produced a significantly longer duration of sensory and motor block as compared to clonidine. Regression time of sensory block was 374.34 ± 44.54 min for dexmedetomidine as compared to 302.5 ± 29.18 min for clonidine. Regression time to reach Bromage 1 was 317 ± 32 min for dexmedetomidine as compared to 220 ± 48 min for clonidine. When we searched the literature, we found that very few authors have compared intrathecal dexmedetomidine to clonidine.

Singh and Shukla[6] compared the effects of intrathecal clonidine and dexmedetomidine on sensory analgesia and motor block of hyperbaric bupivacaine. Regression time of sensory block to S1 dermatome was significantly higher. Regression time to reach Bromage 1 was significantly high in group dexmedetomidine and clonidine groups as compared to bupivacaine. They concluded that though both clonidine and dexmedetomidine prolonged the duration of sensory and motor block of bupivacaine, dexmedetomidine is better in terms of longer duration of action.

Bajwa *et al.*,[7] compared dexmedetomidine and clonidine in epidural anesthesia. Dexmedetomidine to ropivacaine as an adjuvant resulted in an earlier onset of sensory analgesia at T10 level as compared to the addition of clonidine dexmedetomidine not only provided a higher dermatomal spread but also helped in achieving the maximum sensory anesthetic level in a shorter period compared to clonidine modified Bromage scale 3 was achieved earlier in patients who were administered dexmedetomidine as adjuvant. Kanazi *et al.*,[8] studied the effect of low-dose dexmedetomidine or clonidine on hyperbaric bupivacaine they opined that dexmedetomidine (3 mcg) or clonidine (30 mcg) when added to intrathecal Bupivacaine, produces a similar prolongation in the duration of the motor and sensory block with preserved hemodynamic stability and lack of sedation.

In this study, patients remained hemodynamically stable in both dexmedetomidine and clonidine groups. Patients in clonidine group had a greater fall in HR than in dexmedetomidine groups, and the difference was statistically significant. There was no much fall in BP and HR when compared to the baseline values.

Al-Mustafa *et al.*,[9] added dexmedetomidine to spinal bupivacaine for urological procedures. They opined that dexmedetomidine has dose-dependent effect on onset and regression of sensory and motor block. Al-Ghanem *et al.*,[10] evaluated the onset and duration of sensory and motor block as well as operative analgesia and adverse effects of dexmedetomidine (5 μg) or fentanyl (25 μg) given intrathecally with plain 0.5% bupivacaine (10 mg) for spinal anesthesia. Patients in dexmedetomidine Group (D) had significant longer sensory and motor block as compared to patients in fentanyl Group (F). Hypotension was mild to moderate in both groups except one patient in Group F, who had a BP <90 mmHg and required 36 mg ephedrine to restore his BP. They concluded that 10 mg plain bupivacaine supplemented with 5 μg. Dexmedetomidine produces prolonged motor and sensory block compared with fentanyl.

CONCLUSION

On the basis of the observations made during this study and their analysis, the following conclusion was drawn: Addition of dexmedetomidine 2 μg to 0.5% heavy bupivacaine intrathecally produced the faster onset of sensory blockade, longer duration of analgesia and motor blockade and better hemodynamic stability than clonidine 50 μg.

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