Original Article

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Comparative Study of Intrathecal Dexmedetomidine and Buprenorphine as Adjuvant to Bupivacaine in Spinal Anaesthesia: A Randomized Controlled Trail

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Abstract

Background: Various adjuvants were used with local anesthetic in subarachnoid blocks to improve the quality and efficacy. Nowadays, non-opioids like dexmedetomidine had also been introduced in neuraxial blocks and to be investigated as a useful and effective adjuvant.

Aim: The aim of this study is to evaluate and compare intrathecal dexmedetomidine and intrathecal buprenorphine as an adjuvant to 0.5% hyperbaric bupivacaine for lower abdominal surgeries with respect to sensory and motor blockade, hemodynamic changes, and adverse effects.

Materials and Methods: This prospective, randomized, double-blind study was conducted on 60 adult patients of ASA physical status 1 and 2 in the age group of 18-60 years, posted for elective lower abdominal surgeries. The patients were randomly allocated into two groups, namely, Group BB and Group BD of 30 each. Patients in Group BB received 75 mcg of buprenorphine with 0.5% hyperbaric bupivacaine 15 mg intrathecally. Patient in Group BD received 5 mcg of dexmedetomidine with 0.5% bupivacaine 15 mg intrathecally. The following parameters observed were onset and duration of sensory and motor block, time for sensory regression to S1, degree of sedation, hemodynamic stability, and any side effects associated with these drugs. Collected data were analyzed using appropriate statistics.

Results: There was no significant difference between groups regarding demographic characteristics and type of surgery. The onsets of sensory and motor blockades were not statistically significant. The duration of sensory blockade was prolonged in dexmedetomidine group (51%) compared to buprenorphine group. Sensory regression to S1 was also got prolonged in dexmedetomidine group. The sedation level was higher in Group BD (dexmedetomidine) compared to Group BB (buprenorphine).

Conclusion: Dexmedetomidine as an intrathecal adjuvant with 0.5% hyperbaric bupivacaine prolong the sensory and motor blockade with fewer side effects, and the degree of sedation is better.

Key words: α2 adrenergic agonist, Buprenorphine, Dexmedetomidine, Lower abdominal surgery

INTRODUCTION

In subarachnoid blocks, local anesthetics are widely used in combination with adjuvants nowadays to shorten the onset of action, increase the quality of block, increase the duration of anesthesia and analgesia, and decrease the dose of local anesthetics.[1] Dexmedetomidine is a highly selective α2 agonist and introduced in clinical practice in 1999.

The food and drug administration approved dexmedetomidine use for short-term sedation and analgesia in intensive care unit. Although Food and Drug Administration has not approved dexmedetomidine as an adjuvant in neuraxial blocks. In neuraxial anesthesia, dexmedetomidine mediates its analgesia effects via spinal α2 receptors by depressing the release of C-fiber neurotransmitters and hyperpolarization of post-synaptic dorsal neuron.[2] Buprenorphine is an opioid used frequently

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as an adjuvant in spinal anesthesia for post-operative pain relief.[3-5] It exhibits analgesic property both at spinal and supraspinal levels.[6,7] It has been used for various surgeries at different doses for the past decades. It has consistently proven to prolong the duration of anesthesia.[3,4,8]

The aim of this study is to evaluate and compare intrathecal dexmedetomidine and intrathecal buprenorphine as an adjuvant to 0.5% hyperbaric bupivacaine for lower abdominal surgeries with respect to sensory and motor blockade, hemodynamic changes, and adverse effects.

MATERIALS AND METHODS

After approved by the Institutional and Ethical Committee, this study was conducted in 60 ASA I and II patients undergoing elective lower abdominal surgeries under subarachnoid block, and all patients are explained about the procedure and written informed consent was obtained in the age group of 18-60 years.

The detailed pre-anesthetic check-up was done on all patients and relevant hematological, biochemical, and radiological investigations were carried out for all patients as per surgical requirements. Patients with known contraindication for spinal anesthesia or with coagulation disorders or on anticoagulation therapy or with cardiac illness were excluded from this study.

The patients were randomly allocated into two groups to each using closed cover technique. BD group patient received 3 ml 0.5% bupivacaine (15 mg) and dexmedetomidine (5 μg) in 0.5 ml normal saline. BB group patient received 3 ml or 0.5% bupivacaine (15 mg) and 0.5 ml by buprenorphine (75 μg). A total volume of the injected solution was 3.5 ml in both groups.

Anesthetic Procedures

In the operating room, appropriate equipment for the airway management and emergency drugs were kept ready. Non-invasive blood pressure monitor, pulse oximeter, and electrocardiogram (ECG) leads were connected to the patient. Pre-operative baseline systolic and diastolic blood pressure recorded and intravenous line were secured. Patients are preloaded with 10 ml/kg of ringer lactate infusion 10 min before the subarachnoid block.

On sitting position, the skin over the back was prepared with antiseptic solution and draped with sterile towel. After skin infiltration lignocaine 2%, 26G Quinke needle was inserted at L3-4 intervertebral space after confirmation of free flow of cerebrospinal fluid, the prepared solution was injected. The patients were made lie after the injection immediately and time was noted.

The follow-up parameters noted are as follows: (a) Time of injection of subarachnoid block, (b) time of onset and duration of the block, (c) time of onset and duration of motor block, (d) degree of sedation, (e) time for surgery regression to S1 dermatome and (f) duration of surgical procedure, and (g) systolic and diastolic blood pressure, mean arterial blood pressure, pulse rate and oxygen saturation were recorded at 0, 3, and 5 min and there after every 5 min up to 45 min of the procedure.

Adverse Effects

1. Hypotension was said to have occurred mean arterial pressure fell less than 60 mmHg
2. Bradycardia was defined if heart rate <50/min
3. Respiratory depression if respiratory rate <8/min or SpO_2 <90%
4. Any discomfort such as nausea, vomiting shivering, pruritus, and ECG changes was noted
5. Vomiting was planned to manage with ondansetron 4 mg intravenously
6. Ramsay sedation score was used to assess the degree sedation
7. In completion of surgery patient was shifted to post-anesthesia care unit for observation
8. Injection diclofenac sodium 75 mg was given as rescue analgesic when patient complained of pain in post-operative period.

RESULTS

Patient demographic data that includes age, sex and duration of surgery between groups were comparable (Tables 1 and 2, Graphs 1 and 2). The time of onset of surgery block was slower in group PB (3.47 ± 0.507) when compared with Group BD (2.57 ± 0.504), and the p value was statistically not significant (0.629 > 0.05) (Table 3 and Graph 3). The average time taken for the onset of motor block was 3.38 min in Group BB and 4.13 min in Group BD. It was statistically not significant ($P = 0.775 > 0.05$) (Table 4 and Graph 4).

The mean duration of sensory block was shorter in Group BB (332 ± 18.81 min) when compared with Group BD (502.13 ± 12.27 min) and statistically significant ($P < 0.05$). The mean duration of sensory block in Group BD is 51% longer than Group BB (Table 5 and Graph 5). The mean duration of motor block was shorter in Group BB (298.63 ± 35.79 min) when compared with Group BD (432.33 ± 12.74 min) and statistically significant ($P < 0.05$). The mean duration of motor block in Group BD is about approximately 44% longer than Group BB (Table 6 and Graph 6). The time of surgery regression to S1 was shorter in Group BB (272.27 ± 15.39 min) when compared

Table 1: Age distribution

Age group	*N* (%)	
	Group BB	Group BD
Age in years		
Below 30	6 (20)	8 (26.7)
31-40	9 (30)	6 (20)
41-50	6 (20)	9 (30)
Above 50	9 (30)	7 (23.3)
Total	30 (100)	30 (100)
Range	19-60 years	18-60 years
Mean±SD	42.33±12.88	40.57±13.22
P value	0.875 not significant	

SD: Standard deviation

Table 2: Sex distribution

Sex	*N* (%)	
	Group BB	Group BD
Male	25 (83.3)	23 (76.7)
Female	5 (16.7)	7 (23.3)
Total	30 (100)	30 (100)
P value	0.752 not significant	

Table 3: Time of onset of sensory block

Parameter	Time of on set of sensory block (in minutes)	
	Group BB	Group BD
Range	3-4	2-3
Mean±SD	3.47±0.507	2.57±0.504
P value	0.629 not Significant	

SD: Standard deviation

Table 4: Time of onset of motor block

Parameter	Time of onset of motor block (in minutes)	
	Group BB	Group BD
Range	3-5	3-5
Mean±SD	3.83±0.817	4.13±0.78
P value	0.775 not significant	

SD: Standard deviation

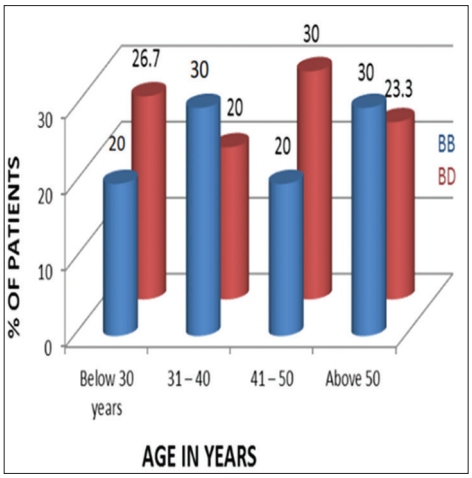
Table 5: Duration of sensory block

Parameter	Duration of sensory block (in minutes)	
	Group BB	Group BD
Range	303-360	480-520
Mean±SD	332±18.81	502.13±12.27
P value	0.005 significant	

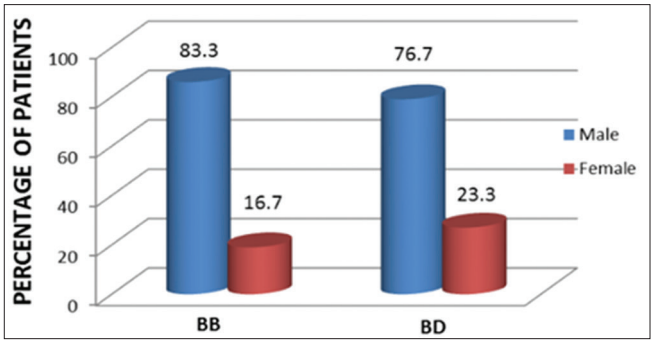
SD: Standard deviation

to with Group BD (398.1 ± 6.50 min) and statistically significant ($P < 0.05$) (Table 7 and Graph 7).

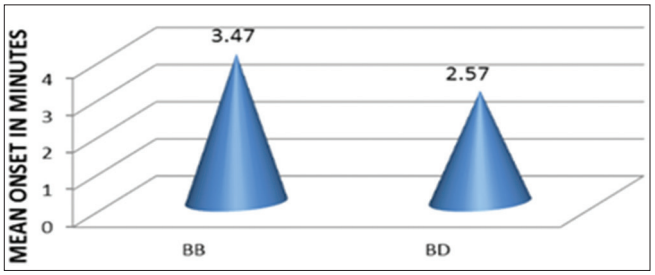
The mean arterial pressure was monitored from preoperative basal to 45 min of procedure (11 intervals) none of the intervals had statistically significant (Table 8 and Graph 8). Heart rate was recorded in 11 intervals out of which only 2 intervals (0 and 3rd min) was statistically significant (Table 9 and Graph 9). The degree of sedation by using Ramsay sedation scale, better in Group BD when compared to Group BB (Table 10).



Graph 1: Age distribution



Graph 2: Sex distribution



Graph 3: Onset of sensory block

DISCUSSION

It has been found recently that prolonged duration of action of buprenorphine is due to its local anesthetic action.[9] The lesser side effects in the post-operative period were due to its high lipid solubility.[10]

Table 6: Duration of motor block

Parameter	Duration of motor block (in minutes)	
	Group BB	Group BD
Range	293-360	413-460
Mean±SD	298.63±35.79	432.33±12.74
P value	<0.05 significant	

Table 7: Time of sensory regression to S1

Parameter	Time of sensory regression to S1 (in minutes)	
	Group BB	Group BD
Range	250-299	389-409
Mean±SD	272.27±15.39	398.1±6.50
P value	0.001, <0.05 significant	

Table 8: Mean arterial pressure

Time interval (min)	Mean±SD		*P* value
	BB Group	BD Group	
0	81.23±10.45	80.17±10.45	0.963
3	80.57±13.35	80.90±10.47	0.089
5	75.63±14.47	80.33±13.79	0.854
10	78.60±13.71	83.20±12.63	0.897
15	75.07±11.96	78.97±12.75	0.337
20	81.17±13.09	79.53±13.21	0.780
25	79.60±10.83	79.60±10.61	0.958
30	74.50±10.86	76.97±11.53	0.406
35	82.13±12.96	83.47±11.56	0.222
40	77.60±10.93	76.43±11.08	0.663
45	78.43±11.50	77.57±12.10	0.503

SD: Standard deviation

Table 9: Heart rate

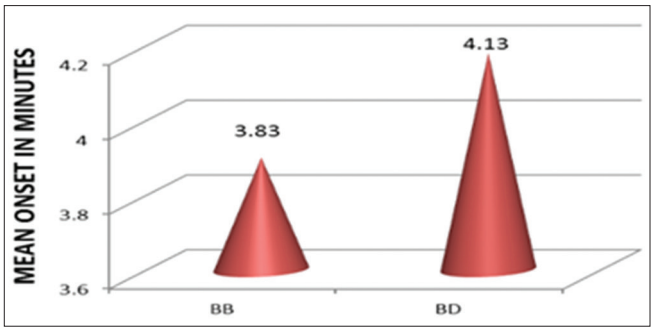
Time interval (min)	Mean±SD		*P* value
	BB Group	BD Group	
0	78.93±12.21	77.43±9.16	0.035*
3	81.47±13.37	74.27±9.13	0.000*
5	80.63±12.79	81.07±11.55	0.360
10	78.37±13.96	80.33±11.89	0.769
15	77.73±15.92	77.80±12.18	0.083
20	79.23±13.13	82.40±13.49	0.806
25	79.77±12.05	78.57±12.43	0.668
30	80.93±12.50	79.87±12.58	0.684
35	79.90±11.72	78.17±11.21	0.584
40	79.70±12.15	80.73±11.36	0.442

SD: Standard deviation

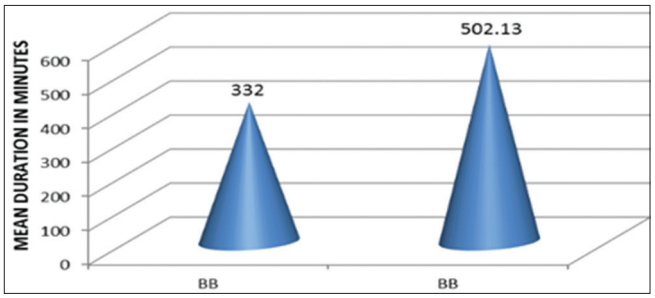
Table 10: SpO_2

Parameter	SpO_2	
	Group BB	Group BD
Range (%)	97-100	97-100
Mean±SD	98.53±1.008	98.43±1.006
P value	0.972 not significant	

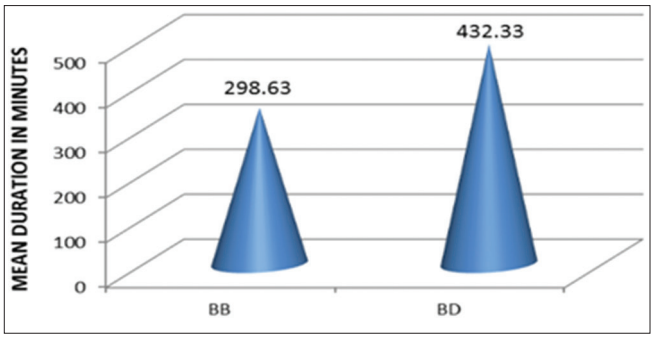
SD: Standard deviation



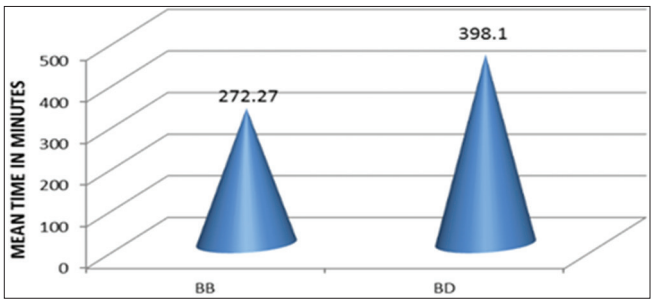
Graph 4: Onset of motor block



Graph 5: Duration of sensory block

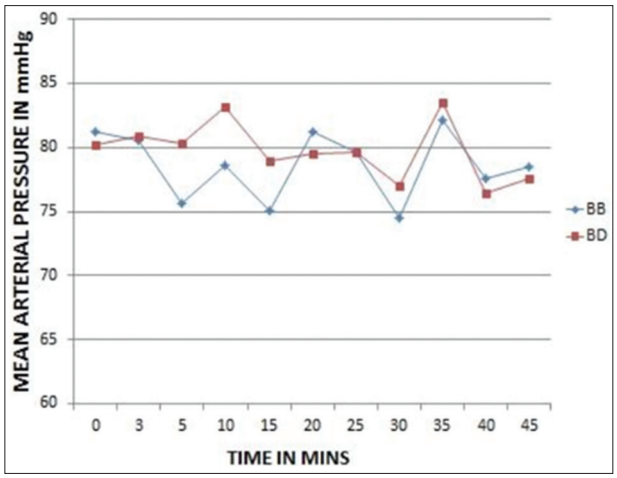


Graph 6: Duration of motor block

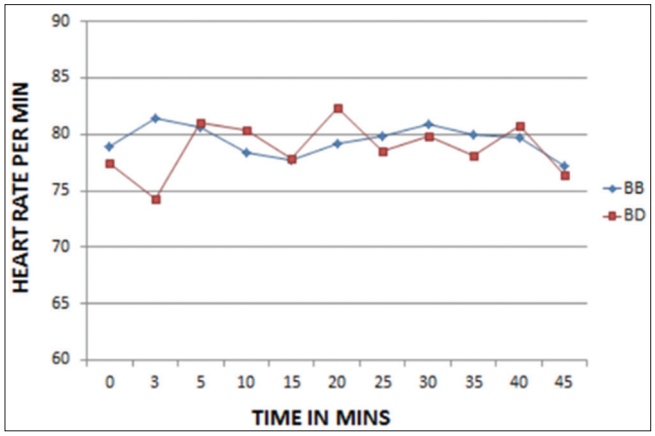


Graph 7: Time of sensory regression to S1

Dexmedetomidine as an additive to intrathecal hyperbaric bupivacaine to prolong the quality and duration of action is not clearly known.[11] It is attributed that it acts by binding to post-synaptic dorsal horn neurons and to the c-fibers in the presynaptic region and decreasing the release of c-fiber neurotransmitters producing hyperpolarization of neurons in the post-synaptic region.[12]



Graph 8: Comparison of mean arterial pressure



Graph 9: Comparison of heart rate

Kanazi *et al.*[13] have used 3 mg of dexmedetomidine and said to have equipotent effect with clonidine. Eid *et al.*[14] studied the effects of dexmedetomidine on dose related manner (10 mcg and 15 mcg) and confirmed the prolongation of duration of analgesia. Many studies have chosen 5 mg of dexmedetomidine as an additive to buprenorphine and proven efficiency.[15] Hence, in our study, we choose 5 µg dexmedetomidine as an additive.

In this study, dexmedetomidine group prolonged duration of analgesia compared to buprenorphine group which has 51% higher than later. Gupta *et al.*[16] had shown similar results. In our study itself, motor blockade in dexmedetomidine group was about 45% prolonged and this could be explained by increased dosage used comparing with Gupta *et al.*,[16] study.

It was noted that 2 cases of bradycardia and nil cases of hypotension in dexmedetomidine group where on 6 cases of bradycardia and 8 cases of hypotension in buprenorphine group and they were managed successfully with the use of atropine 0.6 mg intravenously and ephedrine in incremental doses of 6 mg. Gupta *et al.*[16] study, the incidence of bradycardia was more in dexmedetomidine group. Dexmedetomidine causes bradycardia but the effect is more prominent when administered intravenously with higher doses.[17] The sedation score (Ramsay sedation scale) was higher in patients belonging to dexmedetomidine group as compared to buprenorphine group and it is statistically significant.

LIMITATION

The absence of control group to compare the effect of drugs separately is lacking in our study.

CONCLUSION

Dexmedetomidine as intrathecal adjuvant with 0.5% hyperbaric bupivacaine prolong the sensory and motor blockade with fewer side effects and the degree of sedation is better.

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