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Effects of Clonidine Versus Dexmedetomidine with Intrathecal Hyperbaric 0.5% Bupivacaine in Patients Posted for Elective Lower Abdominal Surgeries

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Abstract

Aims: To study and compare the efficacy of intrathecal dexmedetomidine 5 µg *versus* intrathecal clonidine 50 µg as an adjuvant to 0.5% bupivacaine heavy 12.5 mg for spinal anesthesia. *Materials and Methods:* The present study is prospective, controlled double blind comparative clinical study on spinal block characteristics in patients scheduled for elective lower abdominal surgeries was undertaken to evaluate the efficacy and the safety of dexmedetomidine or clonidine as adjuvant to intrathecal hyperbaric 0.5% bupivacaine. Ninety patients were randomly divided into three groups, each group consisting of thirty patients (n = 30). *Results:* Dexmedetomidine group and clonidine group there is an early onset of both sensory and motor blockade and a higher level of sensory blockade compared to control group and duration of sensory, motor blockade and duration of analgesia are significantly prolonged in the dexmedetomidine group and clonidine group compared to the control group. There was a small percentage of patients who developed significant fall *n* blood pressure and heart rate which were easily managed without any deleterious effect. Seven patients each in dexmedetomidine group and clonidine group and two patients in control group developed hypotension requiring treatment. Five patients in dexmedetomidine group, four patients in clonidine group and one patient in control group developed bradycardia requiring treatment. More number of patients in the dexmedetomidine group and clonidine group were sedated and easily arousable. *Conclusion:* Dexmedetomidine is a better neuraxial adjuvant compared to clonidine for providing early onset of sensory and motor blockade, adequate sedation and prolonged postoperative analgesia.

Keywords: Dexmedetomidine; Clonidine; Postoperative analgesia.

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Introduction

Regional anesthesia is the preferred technique for most of lower abdomen and lower limb surgeries. It allows the patient to remain awake, minimizes or completely avoids the problem associated with

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airway management. With spinal anesthesia, the technique is simple to perform, the onset of anesthesia is more rapid, avoids polypharmacy, allowing the surgical incision to be made sooner and also provides postoperative analgesia.

Hyperbaric bupivacaine 0.5% is extensively used in India for spinal anesthesia. Though the duration of action of bupivacaine is prolonged, it will not produce prolonged postoperative analgesia. Hence, another adjuvant is required for producing prolonged postoperative analgesia. The discovery of opioid receptors and endorphins in spinal and supraspinal regions soon led to the use of spinal opiates. Morphine was the first opioid administered intrathecally to augment neuraxial blocks. Opioid analgesic drugs produce intense, prolonged analgesic action without gross autonomic changes, loss of motor power or impairment of sensation other than pain when injected into subarachnoid or epidural space. Morphine can produce serious side effects like late and unpredictable respiratory depression, postoperative nausea and vomiting, pruritus and urinary retention.[1]

Clonidine has been shown to result in prolongation of the sensory blockade and reduction in the amount or concentration of local anesthetic required to produce postoperative analgesia. Clonidine also has the ability to prolong the motor blockade produced by bupivacaine. Large doses of intrathecal clonidine (as much as 450 µg) without local anesthetics provide sedation and intense and long lasting postoperative analgesia, are inadequate for surgical anesthesia and for this reason, clonidine has been used as an adjuvant to local anesthetics rather than used alone.[2]

While clonidine has been used as an adjuvant to local anesthetic agents for intrathecal purposes with successful results, there are only a few studies available for dexmedetomidine for such studies. Till recently dexmedetomidine was not available in India though it is being used in other countries since many years. Since, it has been recently introduced in India and not many studies have been done in India regarding its use as an adjuvant to local anesthetic agents for intrathecal purpose hence, there is a need to study its effectiveness for spinal anesthesia.

Hence, we have undertaken this study to evaluate and compare the effects of adding clonidine versus dexmedetomidine with intrathecal hyperbaric 0.5% bupivacaine in patients scheduled for elective lower abdominal surgeries.

Materials and Methods

The study entitled "Prospective, controlled double blind comparative clinical study of effect of adding 5 µg dexmedetomidine *versus* 50 µg clonidine to intrathecal 12.5 mg of 0.5% hyperbaric bupivacaine on spinal block characteristics in patients scheduled for elective lower abdominal surgeries" was undertaken in Alluri Sitarama Raju Academy of Medical Sciences, during the period November 2013 to July 2015. The study was undertaken after obtaining ethical committee clearance as well as informed consent from all patients.

Ninety patients in the age group between 20 years and 60 years of either sex belonging to ASA Grade-I and Grade-II posted for elective lower abdominal surgeries without any comorbid disease were grouped randomly into Three Groups (n = 30). Randomization was done using simple sealed envelope technique:

Group B (Control Group): Received 12.5 mg of 0.5% hyperbaric bupivacaine with 0.5 ml normal saline.

Group C (Clonidine Group): Received 12.5 mg of 0.5% hyperbaric bupivacaine with 50 µg clonidine.

Group D (Dexmedetomidine Group): Received 12.5 mg of 0.5% hyperbaric bupivacaine with 5 µg dexmedetomidine.

The doses of dexmedetomidine and clonidine were chosen according to a 1:10 ratio found to be equipotent and would produce similar effects on the characteristics of bupivacaine spinal anesthesia.

Inclusion criteria:

Adult patients of either sex, aged between 20 and 60 years, belonging to ASA Grade I and II without any comorbid disease scheduled for elective lower abdominal surgeries.

Exclusion criteria:

Age group less than 20 years and more than 60 years, Patients belonging to ASA Grade III, IV and V, Pregnant females, Patients posted for emergency surgeries, Patients with morbid obesity, Patients having any absolute contraindication for spinal anesthesia like raised intracranial pressure, severe hypovolemia, bleeding diathesis and local infection. and Patients with comorbid diseases.

Preoperative assessment was done for each patient and written informed consent was taken. Patients were kept NPO for solids 6 hrs and clear fluids 2 hrs before surgery. Patients were premedicated on the night before surgery with

Tablet Ranitidine 150 mg and Tablet Alprazolam 0.5 mg. Intravenous line was obtained with 18 guage cannula and preloaded with Ringer lactate 500 ml half an hour before anesthesia. Monitoring was done using multiparameter monitor having pulse oximetry, ECG and NIBP. Patients were placed in flexed lateral position. Under aseptic precautions spinal block was performed at level of L3-L4 through a midline approach using 25G Quincke spinal needle and study drug was injected with operative table kept flat. Patients were turned to supine posture immediately and supplemental oxygen given. The test drugs were prepared by the senior anesthesiologist who was not involved in the study, Clonidine (Cloneon; 150 µg/ml) was diluted to 1.5 ml with normal saline and 0.5 ml (50 µg) of it was added to 2.5 ml of 0.5% hyperbaric bupivcaine. Dexmedetomidine (Dexem 50 µg/0.5 ml) 0.5 ml was diluted to 5 ml with normal saline and 0.5 ml of this was added to 2.5 ml of 0.5% hyperbaric bupivacaine. The observer and the patient were blinded for the study drug.

The following parameters were noted:

- Onset of sensory blockade and motor blockade;
- Maximum level of sensory blockade attained and the time taken for the same was noted;
- Maximum level of motor blockade attained and the time taken for the same was noted;
- Two segments sensory regression time was noted;
- Total duration of analgesia was noted by VAS score;
- Total duration of sensory blockade and motor blockade was noted;
- Sensory blockade was tested using pinprick method with a blunt tipped 27G needle at every minute for first 5 mins and every 5 mins for next 15 mins and every 10 mins for next 30 mins and every 15 mins till the end of surgery and there after every 30 mins until sensory block is resolved;
- Quality of motor blockade was assessed by modified Bromage scale;
- Level of sedation noted by Ramsay Sedation Scale;
- Total duration of surgery and if any side effects were noted.

Hemodynamic monitoring was done during the block every 5 mins for first 15 mins and every 10 mins for next 30 mins and once in 15 mins till the end of surgery and postoperatively every hourly employing multi parameter monitor which displays Heart Rate (HR), Systolic Blood Pressure (SBP) Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), ECG and SpO_2 hourly.

Statistical Analysis:

Results are expressed as the means and standard deviations, medians and ranges, or numbers and percentages. The comparison of normally distributed continuous variables between the groups was performed using one-way analysis of variance (ANOVA) and, if appropriate, followed by the Bonferroni test for post hoc analysis. Nominal categorical data between study groups were compared using the Chi-squared test or Fisher's exact test as appropriate. Ordinal categorical variables and nonnormal distribution continuous variables were Dexmedetomidine or clonidine for supplementation of spinal bupivacaine compared using the Mann-Whitney *U*-test. $p < 0.05$ was considered to be significant.

Table 1: Demographic distribution in present study

	Groups					
	Group B		Group C		Group D	
	No. of Pts.	%	No. of Pts.	%	No. of Pts.	%
Age in years						
20–30	16	53.3	14	46.7	18	60
31–40	9	30	6	20	2	6.7
41–50	5	16.7	6	20	8	26.7
51–60	0	0	4	13.3	2	6.7
Total	30	100	30	100	30	100
Mean ± SD	31.17 ± 9.752		36.60 ± 11.082		33.07 ± 11.585	
Gender						
Male	15	50.0%	20	66.7%	24	80.0%
Female	15	50.0%	10	33.3%	6	20.0%
Total	30	100.0%	30	100.0%	30	100.0%

Results

There is no significant difference in the age of patients between the groups. All the three groups were similar with respect to age distribution (p > 0.05), shown in Table 1.

There is no significant difference in the sex distribution of the patients between the groups. (p > 0.05). 50%, 66.7%, 80% of the patients in Group B, Group C and Group D respectively are males and 50%%, 33.3% and 20% of the patients in Group B, Group C and Group D respectively are females.

There is no significant difference in the height and weight of patients between the groups (p > 0.05) (Table 2).

There is no significant difference in the type of surgical procedure in patients between the groups (p > 0.05). 73.3%, 53.3%, 63.3% of the patients in Group B, Group C and Group D respectively have undergone appendicectomy and 53.3%, 46.7% and 37.7% of the patients in Group B, Group C and Group D respectively have undergone inguinal hernia repair, (Fig. 1).

The mean duration of surgery is 53 ± 6.51 mins in Group B (Control Group), 57.66 ± 12.84 mins in Group C (Clonidine Group) and 51.166 ± 7.15 mins in Group D (Dexmedetomidine Group). There is no significant difference between mean duration of surgery between the groups (p > 0.05), Fig. 2).

There is no statistically significant difference between the groups (p = 0.24).

The mean time of onset of sensory blockade, mean time taken for attaining the maximum sensory blockade and taken for regression of sensory block by two segments is a statistically highly significant

Table 2: Height and weight distribution in present study

	Groups		
	Group B (n = 30)	**Group C (n = 30)**	**Group D (n = 30)**
Height in cm			
Mean	159.4 cm	161.03 cm	161.6 cm
Std. Deviation	4.76 cm	6.18 cm	5.14 cm
Minimum	152 cm	150 cm	150 cm
Maximum	168 cm	170 cm	170 cm
Weight in kg			
Mean	60.9 kg	61.33 kg	60.7 kg
Std. Deviation	4.62 kg	5.53 kg	5.74 kg
Minimum	50 kg	50 kg	50 kg
Maximum	68 kg	70 kg	70 kg

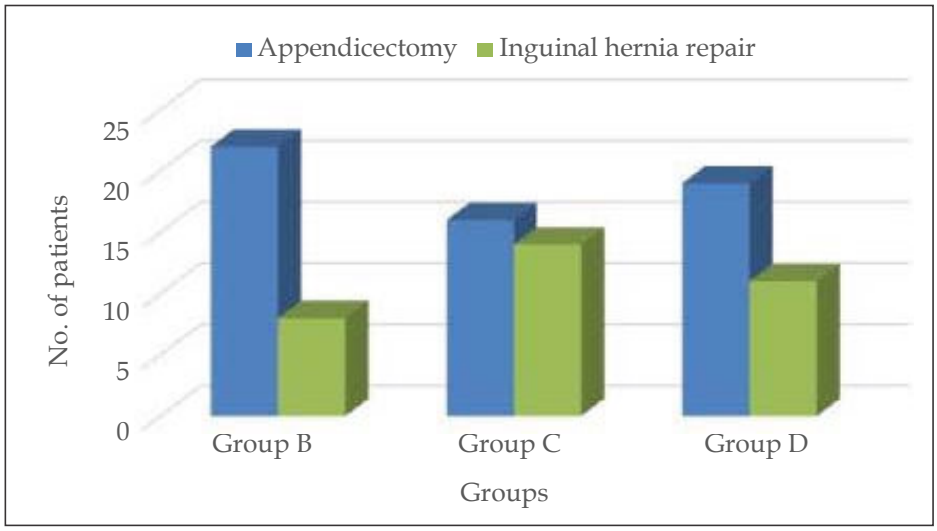

Fig. 1: Types of surgical procedures

difference when Group B was compared with Group C and with Group D (p = 0.000) and there is statistically significant difference between Group C and Group D (p = 0.024), Table 4).

The mean duration of analgesia is a statistically highly significant difference between Group B and Group C (p = 0.000) and between Group B and Group D (p = 0.000) and between Group C and Group D (p = 0.001), (Fig. 3).

The mean time taken for the onset of motor blockade, time taken for attaining maximum motor blockade and duration

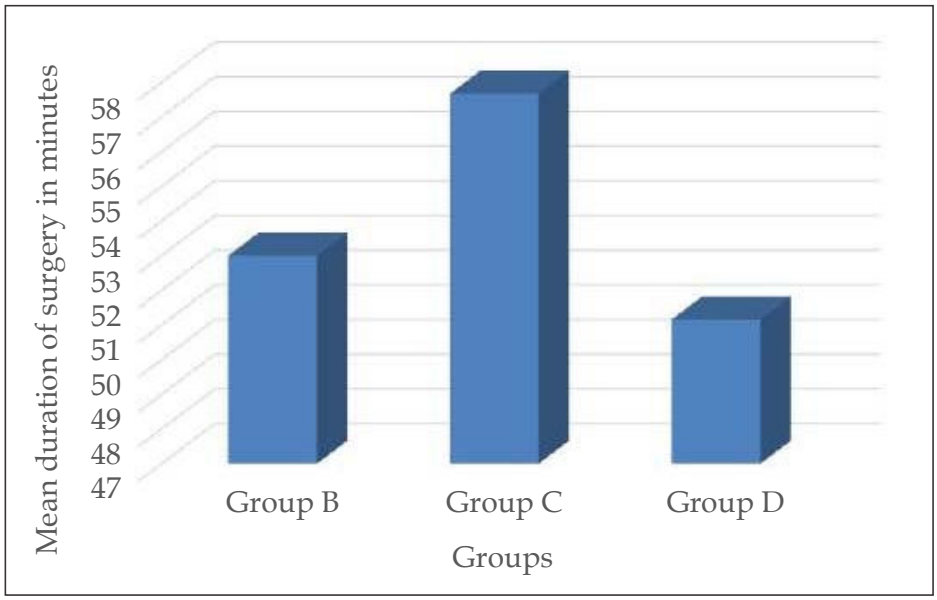

Fig. 2: Mean duration of surgery

Table 3: Maximum level of sensory block attained

Maximum level of sensory block attained	Groups					
	Group B		Group C		Group D	
	No. of Pts.	%	No. of Pts.	%	No. of Pts.	%
T4	2	6.70%	8	26.70%	12	40%
T5	4	13.30%	5	16.70%	2	6.70%
T6	24	80.00%	17	56.70%	16	53.30%
Total	30	100%	30	100%	30	100%

Table 4: Time taken for sensory onset in mins

	Group B	Group C	Group D	p - value B *vs* C	p - value B *vs* D	p - value C *vs* D
Time taken for sensory onset in mins						
Mean ± SD	2.80 ± .664	1.43 ± .504	1.13 ± .346			
Minimum	2	1	1	0.000	0.000	0.024
Maximum	4	2	2			
Time taken for maximum sensory block in mins						
Mean ± SD	7.4 ± 1.102	5.9 ± 0.803	5.2 ± 0.714			
Minimum	6	5	4	0.000	0.000	0.001
Maximum	9	7	7			
Duration of two segment sensory reg in mins						
Mean ± SD	79.46 ± 10.16	136.33 ± 10.90	136.33 ± 11.59			
Minimum	60	120	120	0.000	0.000	1.000
Maximum	95	155	150			

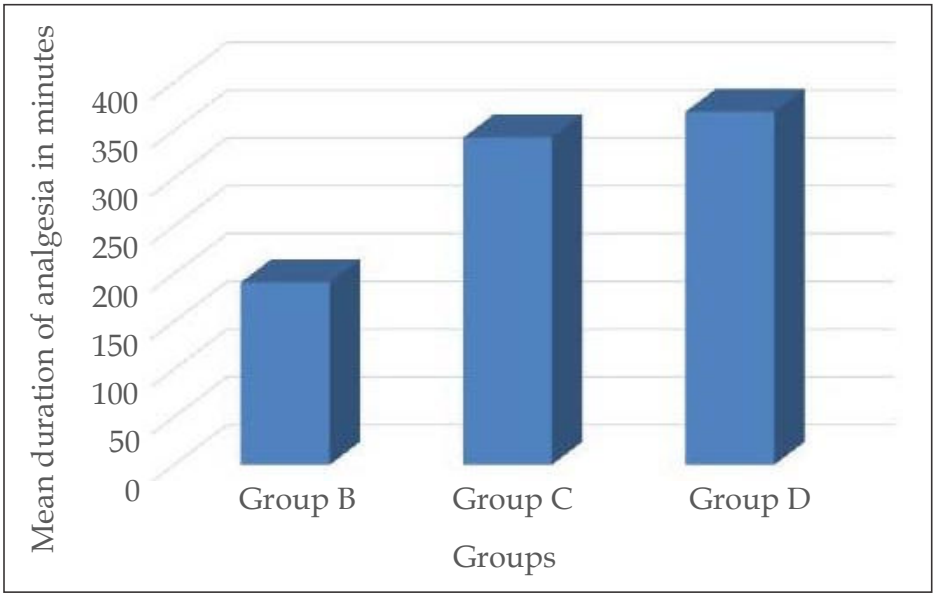

Fig. 3: Mean duration of analgesia

Table 5: Time taken for onset of motor blockade

	Group B	Group C	Group D	*p* - value B *vs* C	*p* - value B *vs* D	*p* - value C *vs* D
Time taken for Motor on set in mins						
Mean ± SD	4 ± 0.695	1.63 ± 0.49	1.17 ± 0.379			
Minimum	3	1	1	0.000	0.000	0.000
Maximum	5	2	2			
Time taken for Maximum motor block in mins						
Mean ± SD	6.57 ± 0.935	6.43 ± 1.04	5.5 ± 0.820			
Minimum	5	5	4	0.000	0.000	0.000
Maximum	9	8	7			
Duration of motor block in mins						
Mean ± SD	166.16 ± 20.95	279 ± 24.68	303.66 ± 35.95			
Minimum	135	240	240	0.000	0.000	0.003
Maximum	210	330	360			

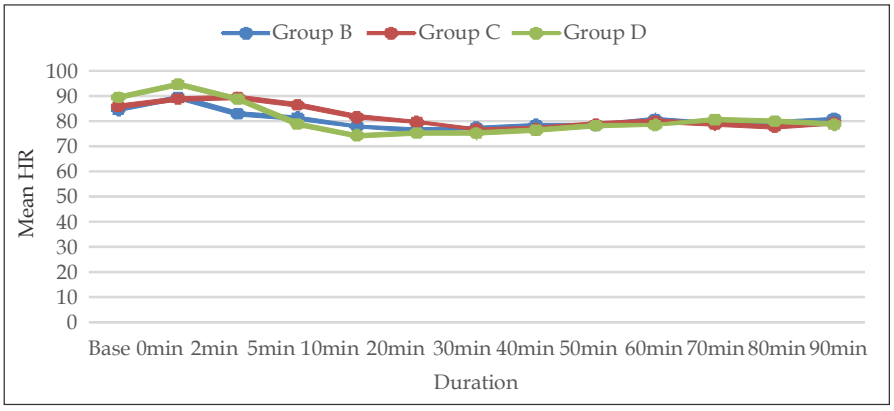

Fig. 4: Mean heart rate at various interval in bpm

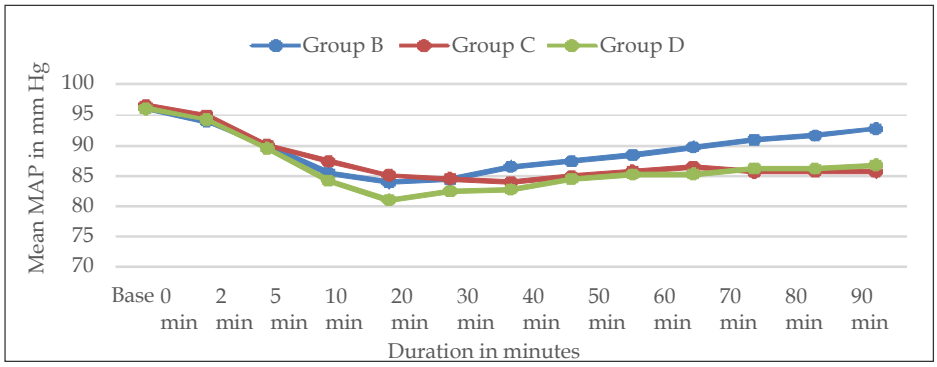

Fig. 5: Mean MAP at various intervals in mm Hg

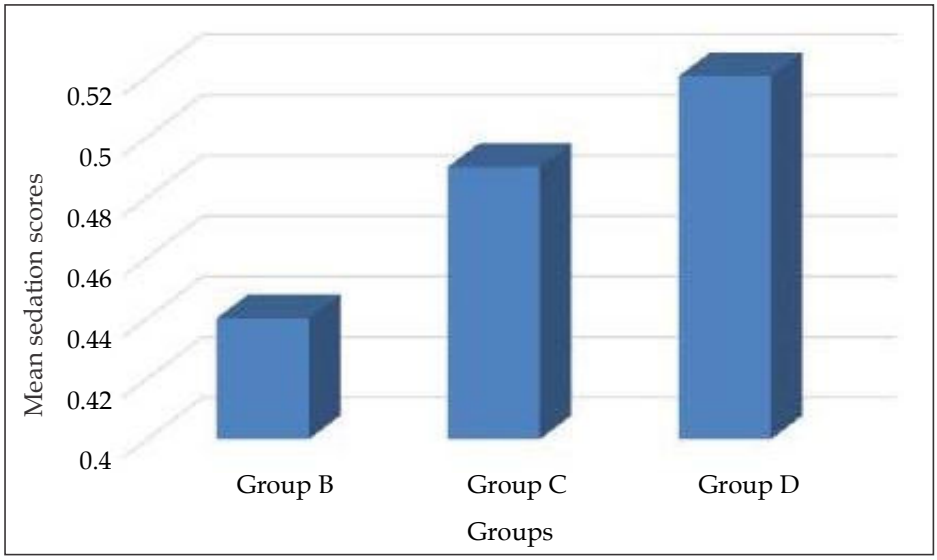

Fig. 6: Mean sedation scores

of motor blockis statistically highly significant difference between Group B and Group C and between Group Band Group D and between Group C and Group D. (p = 0.000), (Table 5).

The mean heart rate from basal to 90th minutes recording is statistically not significant between the groups, (Fig. 4).

The mean MAP from basal to 90th minutes recording is statistically not significant between Group C and Group D, (Fig. 5).

The mean sedation score is 0.4 ± 0.49 in Group B, 0.50 ± 0.682 in Group C, 0.53 ± 0.681 in Group D. There is a statistically highly significant difference between Group B and Group C and between Group B and Group D (p = 0.000). There is statistically no significant difference between Group C and Group D (p = 0.850), (Fig. 6).

In Group B, 2 out of 30 patients, in Group C, 7 out of 30 patients and in Group D, 7 out of 30 patients developed hypotension, which is statistically not significant (p > 0.05). All the patients who developed hypotension could be easily treated with intravenous fluids and vasopressor. In Group B (Control Group) 1 out of 30 patients, in Group C (Clonidine Group), 4 out of 30 patients and in Group D (Dexmedetomidine Group) 5 out of 30 patients developed bradycardia, which is statistically not significant (p > 0.05). All the patients who developed bradycardia were treated by single dose of 0.6 mg of atropine.

Discussion

In present study, demographic data comparing age, sex, height, weight shows no statistical difference

among the groups. Various authors have used different doses of clonidine for intrathecal blockade starting from 15 µg to 300 µg along with local anesthetics and doses of dexmedetomidine starting from 3 µg to 15 µg along with local anesthetics. More number of studies have used 5 µg as the dose hence, we selected a 5 µg of preservative free dexmedetomidine for our study.[2,3]

Asano T et al. showed that binding affinity to spinal alpha-2 receptors of dexmedetomidine compared with clonidine is approximately 1:10.[3] Hence, in our study we selected 10 times the dose of dexmedetomidine as clonidine that is 50 µg.

In our study, the mean time taken for onset of sensory block is 2.8 ± 0.6 mins in the Control Group, 1.43 ± 0.5 mins in the Clonidine Group and 1.13 ± 0.346 mins in the Dexmedetomidine Group. There is a statistically highly significant decrease in the onset of sensory blockade in Clonidine Group and in the Dexmedetomidine Group compared to the Control Group.

In a study, conducted by Saxena H et al. authors observed the onset of sensory blockade to be 6.57 ± 0.49 mins in control group and 2.58 ± 0.33 mins, 2.54 ± 0.34 mins and 2.09 ± 0.89 mins in clonidine group (15 µg, 30 µg and 37.5 µg respectively) and in this study there was a significant reduction in the onset time which concurs with our study.[2] But compared to our study the onset time of sensory block is higher and this could be possibly due to the dose of clonidine used being less than compared to our study in which we used 50 µg.

In a study, conducted by Al-Mustafa MM et al. in which dexmedetomidine was added to spinal isobaric bupivacaine for urological procedures authors observed the onset of sensory blockade to be 9.5 ± 3 mins in control group and 6.3 ± 2.7 mins and 4.7 ± 2 mins in dexmedetomidine group (5 µg and 10 µg respectively) and in this study there was a significant reduction in the onset time of sensory block.[4] But compared to our study the onset time of sensory block is higher and this could be possibly due to the isobaric bupivacaine used compared to our study in which we used hyperbaric bupivacaine.

In a study, conducted by Shukla D et al. authors observed that the duration of onset of sensory blockade in dexmedetomidine group was 2.27 ± 1.09 mins and in control group was 4.14 ± 1.06 mins which showed significant reduction in the onset time of sensory blockade.[5]

The mean time taken for maximum sensory blockade in the present study is 7.4 ± 1.1 mins in the control group, 5.9 ± 0.8 mins in the clonidine group and 5.2 ± 0.71 mins in dexmedetomidine group. There is a statistically significant decrease in the mean time taken for the maximum sensory blockade in the clonidine group and dexmedetomidine group compared to the control group.

In a study, conducted by Saxena H et al. authors observed the mean time to achieve maximum sensory level was 6.8 ± 1.20 mins, 7.4 ± 1.31 mins and 6.7 ± 1.12 mins in clonidine groups (15 µg, 30 µg, 37.5 µg respectively) which is more than our study in clonidine group and this may be due to less mass of clonidine used in the study.[2]

In our study, the maximum level of sensory blockade achieved was T4. Two out of 30 patients in control group, 8 out of 30 patients in clonidine group and 12 out of 30 patients in dexmedetomidine group had T4 level of sensory blockade. There is no statistical significant difference in the maximum level of sensory blockade in the clonidine group and dexmedetomidine group compared to the control group. In studies conducted by Kanazi GE et al., Al-Ghanem SM et al. in dexmedetomidine group the maximum level of sensory blockade achieved was T4 and there was no statistically significant difference in the maximum level of sensory blockade which concurs with our study.[6,7]

The time taken for regression of sensory block by two segments in the present study is 79.46 ± 10.1 mins in the control group, 136.33 ± 10.90 mins in the clonidine group and 136.33 ± 11.590 mins in dexmedetomidine group. There is a statistically significant increase in the mean time taken for regression of sensory block by two segments in clonidine group and dexmedetomidine group compared to the control group.

In a study, conducted by Kanazi GE et al. authors observed the time taken for regression of sensory block by two segments to be 80 ± 28 mins in control group, 101 ± 37 mins in clonidine group and 122 ± 37 mins in dexmedetomidine group, which shows a significant prolongation of two segment regression compared to the control group and it compares with our study.[6] Our study is also consistent with the study done by Sethi BS et al. in clonidine group where it was 136 mins in control group and 218 mins in clonidine group and study done by Eid HEA et al. in dexmedetomidine group where it was 76.9 ± 26.8 mins in control group, 103 ± 28.7 mins in D1 (10 ug) group and 200.6 ± 30.9 mins in D2 (15 ug) group.[8,9] Here authors observed a statistically significant increase in the mean time taken for regression of sensory block by two segments.

The time taken for sensory block to regress to S1 in the present study is 203.33 ± 42.41 mins in the control group, 365.0 ± 24.6 mins in the clonidine group and 396.16 ± 30.61 mins in the dexmedetomidine group. There is a statistically significant increase in the mean time taken for regression of sensory block to S1 in clonidine group and dexmedetomidine group compared to the control group.

This compares with the study conducted by Kanazi GE et al. where the time taken for regression of sensory block to S1 to be 190 ± 48 mins in control group, 272 ± 38 mins in clonidine group and 303 ± 75 mins in dexmedetomidine group which is less than the value in our study.[6] This could be due to the less doses of clonidine and dexmedetomidine used. In a study, conducted by Al-Ghanem SM et al. the mean time taken for regression of sensory block to S1 in dexmedetomidine group was 274.8 ± 73.4 mins compared to fentanyl Group F (179.5 ± 47.4 mins) and in the study conducted by Al-Mustafa MM et al. it was 338.9 ± 44.8 mins in D10 (10 ug) group and 277.1 ± 23.2 mins in D5 (5 ug) group compared to 165.5 ± 32.9 mins in control group and in study conducted by Eid HEA et al.[4,6,7] It was 320 ± 65.8 mins in D1 (10 ug) group and 408.7 ± 68 mins in D2 (15 ug) group compared to 238 ± 57 mins in control group. Authors observed a statistically significant increase in the mean time taken for regression of sensory block to S1 dermatome in dexmedetomidine groups which concurs with our study.

The mean duration of analgesia in our study is 191 ± 22.9 mins in control group, 342.33 ± 28.12 mins in clonidine group and 369.33 ± 34.13 mins in dexmedetomidine group. There is a statistically highly significant increase in the duration of analgesia in dexmedetomidineand clonidine group compared to the control group.

Our study concurs with the study conducted by Grandhe RP et al., where authors observed the mean duration of analgesia of 228 ± 42 mins in the control group and 378 ± 48 mins when using clonidine of 1 µg/kg with a mean weight of 60.6 ± 19.4 kg.

In our study, the mean time for onset of motor block is 4 ± 0.69 mins in control group, 1.63 ± 0.49 mins in clonidine group and 1.17 ± 0.379 mins in dexmedetomidine group. There is a statistically highly significant decrease in the mean time for onset of motor blockade in the dexmedetomidine group and clonidine group compared to the control group.

In a study, conducted by Al-Mustafa MM et al., the duration of onset of motor blockade in Group D10 (10 µg) was 10.4 ± 3.4 mins, Group D5 (5 µg) was 13.0 ± 3.4 mins and Group N (Control Group) was 18.0 ± 3.3 mins and in a study conducted by Shukla D et al. it was 3.96 ± 0.92 mins in Group D and 4.81 ± 1.03 mins in control group which showed a significant decrease in the mean time for onset of motor blockade.[4,5] In the study, done by Saxena H et al. in the clonidine group authors observed a significant decrease in the mean time for onset of motor blockade which was 7.41 ± 0.55 mins in control group and 2.67 ± 0.45 mins, 2.30 ± 0.45 mins, 2.20 ± 0.50 mins in clonidine group (15 µg, 30 µg, 37.5 µg respectively) which concurs with our study.[2]

The mean time taken for maximum motor blockade in our study is 6.57 ± 0.9 mins in control group, 6.43 ± 1.04 mins in clonidine group and 5.5 ± 0.820 mins in dexmedetomidine group. There is a statistically significant decrease in the time taken for maximum motor blockade in dexmedetomidine and clonidine groups compared to the control group. But the grade of motor blockade in the study groups did not differ. All the groups had a motor blockade of Bromage Grade 3.

This compares with the study conducted by Kanazi GE et al. where the time taken for maximum motor blockade is significantly shorter in dexmedetomidine group (13.2 ± 5.6 mins) compared to the control group (20.7 ± 10.3 mins).[6] This is consistent with the studies done by Sethi BS et al. and Saxena H et al. who observed the complete motor blockade of the lower extremity in all patients in clonidine group.[2,8] In a study, conducted by Dobrydnjov I et al. authors found a better quality of block in all the three clonidine groups, where no supplementation with general anesthesia for relaxation request from surgeons was needed intraoperatively.[10]

In our study, the mean duration of motor blockade was 166.16 ± 20.95 mins in control group, 279 ± 24.68 mins in clonidine group and 303.66 ± 35.95 mins in dexmedetomidine group. There is a statistically highly significant increase in the duration of motor blockade in dexmedetomidine group and clonidine group compared to the control group.

This compares with study conducted by Kanazi GE et al. where the mean duration of motor blockade is 163 ± 47 mins in control group, 216 ± 35 mins in clonidine group and 250 ± 76 mins in dexmedetomidine group which is less than the value in our study.[6] This could be due to the less doses of clonidine and dexmedetomidine used. Our study almost concurs with the study

conducted by Kaabachi O et al. who observed the mean duration of motor blockade to be 252 ± 79 mins when using clonidine of 1 μg/kg.[11]

In the control group, we observed a maximum fall in MAP of 12.2 mm Hg from basal MAP at 10th min, in the clonidine group it was 12.56 mm Hg at 30th min and in the dexmedetomidine group it was 14.96 mm Hg at 30th min. There was no statistically significant difference in any of the three groups regarding fall in MAP. However, it was found that there was a delay in maximum fall in MAP in the clonidine group and the dexmedetomidine group compared to the control group.

Two patients in control group, seven patients in clonidine group and seven patients in dexmedetomidine group developed hypotension and were easily managed with intravenous fluids and vasopressor.

In a study, conducted by Sethi BS et al. authors observed lowest MAP (70 mm Hg) in clonidine group (1 μg/kg, mean weight 57.93 ± 4.75 kg) which is less than that in our study (76.05 ± 2.54 mm Hg). In a study, conducted by Grandhe RP et al. the incidence of hypotension (fall in MAP of > 20% of preinduction value) was 10/15 patients in clonidine group (clonidine 1 μg/kg, mean weight 60.6 ± 19.4 kg) and 8/15 patient in clonidine group (clonidine 1.5 μg/kg, mean weight 62.7 ± 18 kg).[8,12] In a study, conducted by Al-Ghanem SM et al. authors observed that the hypotension (fall in MAP of > 30% of preinduction value) was mild to moderate in both dexmedetomidine and fentanyl group.[7] 4/38 patients in dexmedetomidine group and 9/38 patient in fentanyl group had hypotension but it did not reach a significant difference.

Hemodynamic disturbances resulting from intrathecal Alpha 2 agonists depends upon other factors like segmental site of injection, patient position, preloading and baricity of local anesthetic employed.

In the control group, we observed a maximum decrease in the mean heart rate of 7.8 bpm from basal value at 20th min, in the clonidine group it was 9.26 bpm at 30th min and in the dexmedetomidine group it was 15.33 bpm at 10th min. There was no statistically significant difference in any of the three groups regarding decrease in the mean heart rate. However, it was found that there was a delay in maximum decrease in the mean heart rate in the clonidine group compared to the dexmedetomidine group and the control group. Five patients in dexmedetomidine group, four patients in clonidine group and one patient in control group had bradycardia which is statistically not significant. Bradycardia was easily reversed with 0.6 mg intravenous atropine in all the patients. In a study, conducted by Kaabachi O et al. the authors observed the incidence of bradycardia to be 30% in clonidine (2 μg/kg) group which is higher compared to our study and this may probably due to larger dose of clonidine (2 μg/kg) used compared to our study (17.77%).[12]

In our study, sedation is assessed using a sedation scale according to the study done by Al-Ghanem SM et al. at the end of surgery.[7] In our study, in the dexmedetomidine group 10% of patients had Grade 2 sedation, 33.33% had Grade 1 sedation and remaining 56.7% had Grade 0 sedation and in the clonidine group 36% of patients had Grade 2 sedation, 30% had Grade 1 sedation and remaining 60% had Grade 0 sedation compared to 40% of patients in control group having Grade 1 sedation and 60% having Grade 0 sedation. No patients in control group had Grade 2 sedation and there was a statistical significance in mean sedation scores between control group and clonidine group and between control group and dexmedetomidine group. There was no statistical significance between clonidine group and dexmedetomidine group.

In our study, we did not observe any evidence of respiratory depression, episodes of nausea, vomiting, shivering in any of the groups. None of the patients came back to us with backache, buttock pain or leg pain or any neurological deficit. This was conformed with most of the studies.

Conclusion

Dexmedetomidine and clonidine when used intrathecally along with Bupivacaine significantly prolonged the duration of analgesia and there was also clinically significant difference between clonidine and dexmedetomidine on spinal block characteristics, intrathecal dexmedetomidine was better than clonidine with regards to onset and duration of both sensory and motor blockade as well as duration of analgesia. Hence, dexmedetomidine is a better neuraxial adjuvant compared to clonidine for providing early onset of sensory and motor blockade, adequate sedation and prolonged postoperative analgesia.

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