A Comparative Study of Effect of Intrathecal Dexmedetomidine and Clonidine as Adjuvants to 0.5% Hyperbaric Bupivacaine in Spinal Anaesthesia

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ABSTRACT

Background:

This study was conducted to compare the effects of intrathecal clonidine (30 micrograms) with intrathecal dexmedetomidine (5 micrograms) with regard to a) time of onset, peak effect and duration of sensory and motor block; b) hemodynamic effects following administration of the drugs; c) duration of complete and effective analgesia; d) sedation levels and any side effects associated with the drugs used in the study; e) and to find out which of these two is more useful clinically.

Materials & Methods:

After obtaining clearance from the institutional ethics committee and written informed consent from the study participants a hospital based double blind, prospective randomized control study was conducted among 60 patients undergoing elective urological surgeries under spinal anaesthesia, for a period of one year, in the Sri Sathya Sai Institute of Higher Medical Sciences, Prasanthigram, Andhra Pradesh.

Results: In comparison of intra- and post-operative sensory block and motor block assessments in two groups of patients, the difference was found to be statistically significant. In a comparison of VAS scores in two groups of patients, VAS scores were lower in the dexmedetomidine group at 3rd, 5th and 6th hours compared to clonidine which was statistically significant. In comparison

of sedation scores in two groups of patients, a higher sedation score was significantly more associated with group BD. In comparison of heart rate/min in two groups of patients at 30 minutes, the clonidine group had a lower heart rate than the BD group. In a comparison of SBP mm hg in two groups, the systolic blood pressure at 60 minutes was significantly lower in the clonidine group than the dexmedetomidine group.

Conclusion: 5 μg dexmedetomidine seems to be an attractive alternative to 30 μg clonidine as an adjuvant to spinal bupivacaine in surgical procedures.

Keywords: Intrathecal Dexmedetomidine, Clonidine, 0.5% Hyperbaric Bupivacaine, Spinal Anaesthesia

INTRODUCTION

Spinal anaesthesia results from the delivery of the anaesthetic agents into the subarachnoid space. It is one of the simplest regional anaesthetic techniques to perform. Safe practice of spinal anaesthesia includes properly selecting and preparing the patient, accessing the CSF, administering appropriate anaesthetic drugs and adjuvants, managing physiologic side effects and monitoring the patient throughout the procedure as well as in the early recovery process.[1] Spinal anaesthesia with hyperbaric bupivacaine 0.5% is a popular method. The main reasons for the popularity of spinal blocks are that they have well-defined end points, and the anaesthesiologist can produce them reliably with a single injection.[2] The versatility of spinal anaesthesia is afforded by a wide range of local anaesthetics and additives that allow control over the level, the time of onset and the duration of spinal anaesthesia. The distribution of local anaesthetic solutions within the subarachnoid space determines the extent of the neural blockade produced by spinal anaesthesia. August Bier performed the first spinal anaesthetic more than a century ago by injecting cocaine into the cerebrospinal fluid of a patient. For most of the subsequent hundred years, local anaesthetics were the only substances used for neuraxial blockade. This changed with the discovery of opioid receptors in the spinal cord in the 1970s, when intrathecal and epidural opioid administration alone or in combination with local anaesthetics became widespread. Since then driven by the ongoing discovery of multiple spinal transmitters and receptors[3] like cholinergic, opioid, NMDA, GABA, and benzodiazepam receptors[3] has triggered the usage of many diverse groups of pharmacological agents such as neostigmine,[4] opioids, ketamine,[5] midazolam[6] for a synergistic effect with hyperbaric bupivacaine (0.5%) in prolonging the duration of analgesia. However, each drug has its own limitations and a need for an alternative method or drug always exists. Intrathecal clonidine is being extensively evaluated as an alternative to neuraxial opioids for pain control and has been proven to be a potent analgesic. It is used in combination with opioids and local anaesthetics in

labour analgesia and orthopaedic surgery.[7]

However, there is still a dearth of studies on using intrathecal clonidine for postoperative analgesia in lower abdominal surgeries. Clonidine, the prototype of alpha2-agonists, was synthesised in the early 1970s for its use as a nasal decongestant and antihypertensive drug. Clonidine is widely used as an adjunct to anaesthesia and pain medicine.[8] However, it has been little used as a sedative.[9] Dexmedetomidine is a new alpha2-agonist that received FDA approval in 1999 for use as a short-term (less than 24 h) sedative analgesic in the intensive care unit. The use of alpha2-agonists as anaesthetics is not new since many alpha2-agonists are used in veterinary medicine to induce anaesthesia.[10] With dexmedetomidine, there are a number of reasons for the growing and renewed interest in the use of alpha2-adrenoceptors agonists as sedatives: Dexmedetomidine compared to clonidine is a much more selective alpha2-adrenoceptor agonist, which might permit its application in relatively high doses for sedation and analgesia without the unwanted vascular effects from activation of alpha1-receptors. In addition, Dexmedetomidine is a shorter-acting drug than clonidine and has a reversal drug for its sedative effect, atipamezole. These properties render dexmedetomidine suitable for sedation and analgesia during the whole perioperative period: as premedication, as an anaesthetic adjunct for general and regional anaesthesia, and as a postoperative sedative and analgesic.[11]

AIMS AND OBJECTIVES

In this study we wanted to compare the effects of intrathecal clonidine (30 micrograms) with intrathecal dexmedetomidine (5 micrograms) with regards to time of onset, peak effect and duration of sensory and motor block haemodynamic effects following administration of the drugs, duration of complete and effective analgesia, and sedation levels and any side effects associated with the drugs used in the study. We also wanted to find out as to which of these two is more useful clinically.

MATERIALS AND METHODS

After obtaining clearance from the institutional ethics committee and written informed consent from the study participants, a hospital based double blind, prospective randomized control study was conducted among 60 patients undergoing elective urological surgeries under spinal anaesthesia, for a period of one year in the Sri Sathya Sai Institute of Higher Medical Sciences, Prasanthigram, Andhra Pradesh.

Inclusion Criteria

- Patients of male sex.
- Patients with ASA grade I and II.
- Patients aged between 18 and 60 years.
- Patients giving valid informed consent.

Exclusion Criteria

- Patients belonging to ASA grades 3 and 4
- Patients are physically dependent on narcotics, benzodiazepines.
- Contraindications to regional anaesthesia.
- Failure of spinal block and need for general anaesthesia
- Patients with severe systemic disease, metabolic, cardiac, congenital or neurologic disorders.

Statistical Methods

Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented as mean SD (min-max) and results on categorical measurements are presented as number (%). Significance is assessed at a 5% level of significance. Student t-test (two-tailed, independent) has been used to find the significance of study parameters on a continuous scale between two groups, intergroup analysis) on metric parameters. Levene's test for homogeneity of variance has been performed to assess the homogeneity of variance. Chi-square or Fisher Exact test has been used to find the significance of study parameters on a categorical scale between two or more groups. SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1, Systat 12.0 and R environment version 2.11.1 were used for the analysis of the data and Microsoft Word and Excel were used to generate graphs, tables etc**.**

RESULTS

Variables	**Group BC**	**Group BD**	**P-Value**
Onset of sensory block (secs)	136.67±49.99	107.00±26.8	0.006**
Duration of sensory block (mins)	293.00±25.35	404.57±26.16	<0.001**
Duration of effective analgesia (mins)	375.00±24.46	466.67±27.96	<0.001**
Peak sensory level in mins	27.80±5.44	19.83±4.64	<0.001**
Comparison of Intra and Post-Operative Sensory Block Assessment in Two Groups of Patients Studied			
Variables	**Group BC**	**Group BD**	**P-Value**
Onset of motor block (secs)	150±57.72	123.00±38.34	0.037
Peak motor block (mins)	6.7±1.44	5.63±1.71	0.011*
Duration of motor block (mins)	242±33.88	344.67±29.33	<0.001**
Comparison of Intra and Post-Operative Motor Block Assessment in Two Groups of Patients Studied			
VAS Score	**Group BC**	**Group BD**	**P-Value**
2 hours	0.00±0.00	0.00±0.00	-
3 hours	2.10±0.31	1.33±0.96	<0.001**
4 hours	2.63±0.81	2.80±0.41	0.317
5 hours	3.83±0.75	3.43±0.50	0.018*
6 hours	4.79±0.41	4.43±0.50	0.007**
Table:1 Comparison of VAS Score in Two Groups of Patients Studied			

Early onset of sensory block was noted in group BD (107.00±26.8 secs) when compared to group BC which was 136.67±49.99 secs with a significant p-value of < 0.006.

The duration of sensory block, i.e., time to S1 regression was significantly prolonged in group BD (404.57±26.16 min) when compared to group BC (293.00±25.35 min) with a significant p-value of <0.001.

The duration of effective analgesia was significantly longer in group BD (466.67±27.96 min) when compared with group BC (375.00±24.46 min) with a significant p-value of <0.001. Peak sensory level was achieved early in group BD (19.83±4.64 mins) while in group BC it was 27.80±5.44 mins with a significant statistical p-value difference of <0.001.

It is evident that onset of motor block, i.e., time of injection to Bromage 0 (BD-123.00±38.34 secs, BC-150±57.72 secs) and peak motor block, i.e., time to achieve Bromage-

3 (BD 5.63±1.71 min, BC-6.7±1.44 min) were early in group BD-as compared to group BC with a statistically significant p-value of 0.037 and 0.011 respectively.

The duration of motor block was significantly prolonged with the addition of dexmedetomidine (344.67±29.33 min) than with clonidine (242±33.88 min) with a statistically significant p-value of 0.001.

VAS score was lower in the dexmedetomidine group at 3rd, 5th and 6th hours compared to clonidine which was statistically significant with p-values of <0.001, 0.018 and 0.007 respectively.

Sedation Score	**Group BC**		**Group BD**	
	No	**%**	**No**	**%**
Grade 0	24	80.0	0	0.0
Grade 1	5	16.7	26	86.7
Grade 2	1	3.3	4	13.3
Grade 3	-	-	-	-
Grade 4	-	-	-	-
Grade 5	0	0.0	0	0.0
Total	30	100.0	30	100.0

Table 2: Comparison of Sedation Score in Two Groups of Patients Studied

80% of the patients in group BC were in grade 0, whereas none of the patients in group BD had grade 0 sedation. 86.7% of the patients in group BD had a sedation score of grade 1, while it was only 16.7% in group BC.

The rest of the patients (13.3% in group BD and 3.3% in group BC) had a sedation score of grade 2. A higher sedation score was significantly more associated with group BD with p<0.001 patients.

Heart Rate/Min	**Group BC**	**Group BD**	**P-Value**
0 minute	81.73±4.75	80.8±2.81	0.358
5 minutes	76.7±5.06	78.23±3.44	0.175
10 minutes	72.52±5.37	74.3±3.34	0.130
15 minutes	68.53±5.76	70.53±3.01	0.098+

30 minutes	65.97±6.45	69.3±2.26	0.010*
60 minutes	67.97±7.92	70.13±2.15	0.154
120 minutes	72.1±2.84	71.77±2.21	0.614
180 minutes	73.63±3.64	74.37±2.58	0.372
Comparison of Heart Rate/Min in Two Groups of Patients Studied			
SBP mm hg	**Group BC**	**Group BD**	**P-Value**
0 minute	119.80±5.67	121.13±5.37	0.354
5 minutes	113.8±5.67	116±5.38	0.129
10 minutes	109.43±5.65	111.83±6.08	0.119
15 minutes	103.87±5.68	106.07±5.16	0.122
30 minutes	99.3±8.81	102.43±5.82	0.110
60 minutes	102.2±7.76	105.67±4.84	0.042*
120 minutes	107.13±4.78	108.77±3.66	0.143
180 minutes	109.97±3.37	110.5±3.51	0.551
Table 3: Comparison of SBP mm hg in Two Groups of Patients Studied			

The systolic blood pressure at 60 minutes is significantly lower in the clonidine group than the dexmedetomidine group (p-value of 0.042). There was no change in systolic blood pressure between the two groups during the rest of the period.

DBP mm hg	**Group BC**	**Group BD**	**P-Value**
0 minute	79.33±5.31	80.27±5.75	0.516
5 minutes	73.87±5.73	76.67±5.66	0.062+
10 minutes	69.43±4.55	71.8±5.67	0.080+
15 minutes	65.23±5.13	67.03±5.73	0.205
30 minutes	60.97±4.51	63.97±4.89	0.016*
60 minutes	60.4±3.16	66.2±4.71	<0.001**
120 minutes	68.27±3.81	68.47±4.49	0.853
180 minutes	70.1±4.14	69.7±3.83	0.699
Comparison of DBP mm hg in Two Groups of Patients Studied			

MBP mm hg	**Group BC**	**Group BD**	**P-Value**
0 minute	92.50±5.37	93.53±5.62	0.470
5 minutes	86.3±5.48	88.53±5.12	0.108
10 minutes	83.37±5.96	84.27±6.01	0.562
15 minutes	77.07±4.98	79.23±6.24	0.142
30 minutes	74.53±5.54	77.27±5.31	0.056+
60 minutes	75.33±4.63	80±4.91	<0.001**
120 minutes	79.63±5.86	81.53±4.08	0.151
180 minutes	83.53±3.94	83.5±3.62	0.973
Comparison of MBP mm hg in Two Groups of Patients Studied			
Respiratory Rate	**Group C**	**Group M**	**P-Value**
Pre op	17.60±3.07	17.90±3.13	0.709
5 minute	17.53±3.14	18.30±3.58	0.382
10 minutes	16.83±2.17	17.20±2.54	0.550
15 minutes	17.03±2.77	17.47±2.89	0.555
30 minutes	18.10±3.06	17.57±2.90	0.491
60 minutes	17.70±3.16	19.70±19.03	0.572
90 minutes	17.63±2.98	16.23±3.14	0.081+
120 minutes	15.97±3.62	18.23±2.53	0.007**
180 minutes	16.03±3.1017.83±2.69	17.83±2.69	0.020*
Table 4: Comparison of Respiratory Rate in Two Groups of Patients Studied			

The diastolic blood pressures at different intervals of time are statistically similar (p>0.05) in both groups except at 5, 10, 30 and 60 minutes, where the clonidine group has a lower systolic blood pressure than the dexmedetomidine group, which was statistically significant (p<0.05).

The mean blood pressure at 30 and 60 minutes are significantly lower in the clonidine group than the dexmedetomidine group (p-value of <0.05). We infer from the above result that no significant hemodynamic changes in terms of heart rate, systolic BP, diastolic BP, or mean arterial pressure were noted during most of the period between the two groups under study.

Respiratory rates at different time intervals, in both groups are comparable to each other most of the time.

Intra-Op Side Effects	Group BC (N=30)		Group BD (N=30)		P-value
	No.	%	No.	%	
Hypotension	3	10.0	0	0.0	0.003**
Bradycardia	3	10.0	0	0.0	0.237
Respiratory Depression	0	0.0	0	0.0	-

Table 5: Comparison of Intra Op Side Effects in Two Groups of Patients Studied

The incidence of side effects (hypotension and bradycardia) was significantly higher in patients who received intrathecal clonidine; hypotension occurring in 3 patients compared to none in the dexmedetomidine group ($p<0.001$) and bradycardia occurring in 3 patients compared to none in the dexmedetomidine group ($p<0.001$).

Hypotension in the BC group was corrected with IV fluids (Ringer Lactate 500 ml) alone. None of them required ephedrine for hypotension correction.

None of the patients in either group required atropine for bradycardia even though bradycardia was noted in 3 patients in the BC group, bradycardia noted was of little clinical significance.

None of the patients in either group had respiratory depression.

DISCUSSION

Subarachnoid block with bupivacaine has been most extensively used for lower abdominal and lower limb surgeries because of its simplicity, speed, reliability and minimal exposure to depressant drugs. However, a single intrathecal injection of bupivacaine provides analgesia for only 2-2.5 hours. Most patients require further analgesia during the post-operativeperiod.

Various adjuvants to intrathecal local anaesthetics such as opioids, ketamine, and neostigmine are often added to enhance the duration and quality of spinal anaesthesia. Their use is limited because of significant adverse effects such as pruritus, urinary retention, respiratory depression, hemodynamic instability, nystagmus, nausea and vomiting.

Clonidine is a mixed α_1 and α_2 adrenoceptor agonist with a predominant α_2 action. Traditionally, it has been used as an antihypertensive agent since the late 1960s. Its primary effect is sympatholysis and it reduces peripheral norepinephrine release by stimulating the prejunctional inhibitory α_2-adrenoceptors. Further uses based on its sedative, anxiolytic and analgesic properties are being developed.[12]

In view of the unwanted side effect profile associated with a larger dose of clonidine, the dose of 30 μg for neuraxial block was chosen in our study, as this was proven to be the optimum dose in earlier studies by Alex Tiong-Heng Sia et al. in 2000 and H. Saxena et al. in 2010.[13]

Most of the clinical experience gained in the use of intrathecal a2-adrenoceptor agonists has been described with clonidine. The use of intrathecal clonidine has a well-established synergistic effect with local anaesthetics.[14,15] Studies using a combination of intrathecal dexmedetomidine and local anaesthetics are lacking. In our study, the intrathecal dose of dexmedetomidine selected was based on previous animal studies.[16,17,18] Kalso et al.[16] and Post et al.[18] showed that a 1:10 dose ratio between intrathecal dexmedetomidine and clonidine produced a similar effect in animal models. Asano et al.[17] showed that the potency of epidurally administered a2-adrenoceptor agonists was well correlated with their binding affinity to spinal a2-adrenoreceptors. The intrathecal use of dexmedetomidine is off-label. Thehighest dose of intrathecal dexmedetomidine used in animal studies was 100 μg.[19] Thus, we hypothesized that 5 μg of intrathecal dexmedetomidine might be equipotent to 50 μg of intrathecal clonidine. Strebel et al. concluded in their study, that small doses of intrathecal clonidine (≤150 μg) significantly prolonged the anaesthetic and analgesic effects of bupivacaine in a dose-dependent manner.

The patients studied across the group did not vary much with respect to age, height, and weight. All the patients selected were male and the types of surgeries performed were almost similar in both groups. These parameters were kept identical in both groups to avoid variations in the intra-operative and post-operative outcomes of the patients.

In the present study, the mean onset of sensory block in the clonidine group was 136.67±49.99 secs and in the dexmedetomidine group it was 107.00±26.8 secs. There was a significant difference between the two groups with respect to the onset of block, as the significance value obtained from the independent sample's t test was 0.006. Similar results were obtained with regard to the onset of sensory block with the use of low dose clonidine in the studies conducted by B. S. Sethi et al. in 2007 and Alex Tiong-Heng Sia et al. in 2000.

The mean duration of sensory block in our study in group BC was 293.00±25.35 min and in group BD it was 404.57±26.16 min. There was a significant difference ($p=0.001$) in the mean duration of sensory block between the two groups. Santiveri X et al. in 2002 found that the duration of sensory block was longer in the clonidine group (165.5 ± 30.6 mins) than the control group (139.7 ± 40.4 mins); $p<0.05$. A study by Rajini Gupta et al. in 2009 with the use of D5 (dexmedetomidine 5 μg) found that the mean time of sensory regression to S1 was

476±23 min in the dexmedetomidine group. Mahmoude M. Al-Mustafa in 2009 found that regression time to reach S1 dermatome was 338.9+/-44.8 minutes in group D10, and 277.1+/-23.2 minutes in group D5.

The mean duration of effective analgesia in group BC was 375.00±24.46 min and in group BD it was 466.67±27.96 min. A significant difference (P=0.001) in the mean duration of effective analgesia between the two groups was noted. B.S. Sethi et al. (2007) with the use of low dose intrathecal clonidine as adjuvant to bupivacaine, found a significant difference with respect to the mean duration of effective analgesia between the two groups, the recordings being 614 min (480–1140 min) in the clonidine group as against 223 min (150-300 min) in the control group. In our study, the mean onset of motor blockade in group-BC was 150±57.72 secs compared to 123.00±38.34 secs in group-BD which shows that the onset of motor block in the dexmedetomidine (BD) group is earlier than the clonidine (BC) group. The duration of motor block in the BC group was 242±33.88 min compared to 344.67±29.33 in the BD group which was statistically significant with $p<0.001$ inferring that the duration of motor block is prolonged in dexmedetomidine group than the clonidine group. B.S. Sethi, Deepak Sreevastava in 2007 found that the duration of motor blockade was longer in the clonidine group than in control group ($p<0.05$). Olfa Kaabachi et al. in 2007 showed that the time to full recovery of motor block were 251±79 minutes in the adolescents with clonidine versus 181±59 in the controls indicating that the duration of motor block is prolonged by addition of clonidine. Mahmoude M. Al-Mustafa et al. in 2009 showed that the regression to Bromage 0 was 246.4 +/-25.7 minutes with dexmedetomidine 5 mcg (D5). A study done in 2009 by Rajini Gupta found that the regression time of motor block to reach modified Bromage 0 was 421±21 min with dexmedetomidine 5 mcg(D5).

Early onset of sensory and motor block was noted in the dexmedetomidine group compared to the clonidine group and the duration of motor and sensory block was significantly longer in dexmedetomidine group in comparison to the clonidine group. Kanazi et al. found in their study, that the supplementation of bupivacaine (12 mg) spinal block with a low dose of dexmedetomidine (3 μg) produces a significantly shorter onset of motor block, and a significantly longer sensory and motor block than bupivacaine alone. Our results, with the usage of a higher dose (5 μg) of dexmedetomidine, support the conclusion made by Mahmoude M. Al-Mustafa et al. and Hala E. A. Eid et al. that dexmedetomidine has a dose-dependent effect on the onset and regression of sensory and motor block when used as an adjuvant to bupivacaine in spinal anaesthesia. In addition, we found that the onset of sensory block was shorter with the usage of dexmedetomidine. A possible explanation for why we have a

significant onset of sensory block in our results is that we used a higher dose of dexmedetomidine than Kanazi et al. and a larger volume injected into the subarachnoid space. There was no statistically significant difference in the mean heart rates at different time intervals in the BC and BD groups in the intra operative period except at 15 and 30 minutes.

At 15 and 30 minutes the clonidine (BC) group had a lower mean heart rate than the dexmedetomidine group which was statistically significant. Thereafter the heart rates in both groups in our study did not vary significantly. This shows that our study results corroborate with the studies done by B.S. Sethi et al. in 2007 who also found that the decrease in the mean heart rate from 45 minutes until the end of 6 hours was greater in the clonidine group than the control group ($p<0.05$). Hala E. A. Eid, Mohamed A. Shafie in 2011 also noticed no hemodynamic instability with 10 mcg and 15 mcg dexmedetomidine.

The mean systolic blood pressures in the BC group were lower than those in the BD group at 60 minutes, the difference being statistically significant ($p<0.05$). The mean diastolic blood pressure was statistically significantly lower in the BC group only at 30 and 60 minutes ($p<0.05$) This indicates that the fall in systolic blood pressure and diastolic blood pressure following administration of clonidine intrathecal is greater than that of dexmedetomidine. Though a fall in blood pressure was noted in the clonidine group; it was of only a mild degree which was addressed with 500 ml of IV Ringers lactate fluid, none required ephedrine for correction. Stephan Strebel et al. (2004), observed a small but statistically significant decrease ($p<0.05$) in systolic and diastolic blood pressures in group 3 (bupivacaine + 75 mcg clonidine) and group 4 (bupivacaine + 150 mcg clonidine) at 1, 3 and 4 hours. B.S. Sethi et al. with the use of low-dose clonidine also found a statistically significant decrease in blood pressure in the clonidine group compared to the control group, but none of the patients required any therapeutic intervention for either. Mahmoude M. Al-Mustafa et al. noted stable blood pressure throughoutthe intraoperative period with the use of dexmedetomidine 5 and 10 mcg (D5 and D10).

There was no statistically significant difference in the mean respiratory rates at different time intervals in the BC and BD groups in the intra operative period up to 90 minutes. B.S. Sethi et al. in 2007 showed that there was no significant change in the respiratory rate or $Sp0_2$ from the baseline in the clonidine group, ($p>0.05$) and supplemental oxygen or any other form of airway management was not needed. Kanzi et al. found no respiratory depression with the use of dexmedetomidine 3 mcg (D3).

In our study, the sedation scores (assessed by the score given by Chernic et al.) were statistically significantly higher in the BD group than the BC group ($p<0.05$) which shows that dexmedetomidine produces better sedation when administered intrathecally than clonidine.

This is in accordance with the study done by Strebel and colleagues, who found lack of sedative effect with doses of clonidine as high as 150 μg. Mahmoude M. Al-Mustafa et al. found a Ramsay sedation score of 2 without premeditating their patients with 5 and 10 mcg of dexmedetomidine.

The VAS score at the time of the request of first pain medication in the BC group was 6.03±0.69 hours compared to 7.87±0.90 hours in the BD group which was statistically significant. Brian D. Sites et al. in 2003 concluded that co-administration of intrathecal clonidine decreases the 24 hour IV morphine consumption and 24 hour VAS scores when compared to intrathecal morphine alone. Thus, we can infer that both intrathecal dexmedetomidine and clonidine prolong the duration of post-operative analgesia thereby reducing the visual analogue scores in the post-operative period but dexmedetomidine provides better post-operative analgesia than clonidine when used along with bupivacaine.

None of the patients in either group had any post op complications like nausea, vomiting, hypotension or bradycardia. This result was comparable to that of Kanzi et al.

The intrathecal use of dexmedetomidine is off-label. The highest dose of intrathecal dexmedetomidine used in animal studies was 100 μg. Konakci and colleagues reported white matter injury in rats when high dose epidural dexmedetomidine (6 μg/kg) was used alone; however, subsequently Brummett and coworkers demonstrated no injury and a protective effect when doses of 26-40 μg/kg were used perineurally.

In humans the largest epidural dose used was 2μg/kg and the largest intrathecal dose used was 100 μg. Although no major neurological complications have been reported so far, larger studies are required to rule out any short term or long term adverse effects. Based on the above studies in our patients, we used dexmedetomidine in doses of 5 μg and the 2-week follow up showed no neurological deficit or any complaint from the patients regarding the spinal anaesthesia.

CONCLUSION

In conclusion, 5 μg dexmedetomidine seems to be an attractive alternative to 30 μg clonidine asan adjuvant to spinal bupivacaine in surgical procedures. It provides good quality of intraoperative analgesia, hemodynamically stable conditions, adequate sedation, minimal side effects, and excellent quality of postoperative analgesia.

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