ORIGINAL ARTICLE

Effects of intravenous dexmedetomidine on hyperbaric bupivacaine spinal anesthesia: A randomized study

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ABSTRACT

Background and Objectives: The present study was designed to evaluate the effect of intravenous dexmedetomidine on spinal anesthesia with 0.5% of hyperbaric bupivacaine. **Materials and Methods:** One hundred American Society of Anesthesiologists (ASA) physical status I/II patients undergoing elective surgeries under spinal anesthesia were randomized into two groups of 50 each. Immediately after subarachnoid block with 3 ml of 0.5% hyperbaric bupivacaine, patients in group D received a loading dose of 1 μg/kg of dexmedetomidine intravenously by infusion pump over 10 min followed by a maintenance dose of 0.5 μg/kg/h till the end of surgery, whereas patients in group C received an equivalent quantity of normal saline. **Results:** The time taken for regression of motor blockade to modified Bromage scale 0 was significantly prolonged in group D (220.7 ± 16.5 min) compared to group C (131 ± 10.5 min) ($P < 0.001$). The level of sensory block was higher in group D (T 6.88 ± 1.1) than group C (T 7.66 ± 0.8) ($P < 0.001$). The duration for two-dermatomal regression of sensory blockade (137.4 ± 10.9 min vs. 102.8 ± 14.8 min) and the duration of sensory block (269.8 ± 20.7 min vs. 169.2 ± 12.1 min) were significantly prolonged in group D compared to group C ($P < 0.001$). Intraoperative Ramsay sedation scores were higher in group D (4.4 ± 0.7) compared to group C (2 ± 0.1) ($P < 0.001$). Higher proportion of patients in group D had bradycardia (33% vs. 4%) ($P < 0.001$), as compared to group C. The 24-h mean analgesic requirement was less and the time to first request for postoperative analgesic was prolonged in group D than in group C ($P < 0.001$). **Conclusion:** Intravenous dexmedetomidine significantly prolongs the duration of sensory and motor block of bupivacaine spinal anesthesia. The incidence of bradycardia is significantly higher when intravenous dexmedetomidine is used as an adjuvant to bupivacaine spinal anesthesia. Dexmedetomidine provides excellent intraoperative sedation and postoperative analgesia.

Key words: *Dexmedetomidine, hyperbaric bupivacaine, intrathecal, Ramsay sedation scale, spinal anesthesia*

INTRODUCTION

α2-Agonists like clonidine and dexmedetomidine have been used to prolong spinal anesthesia.[1-6] Apart from sedation and analgesia, they also decrease the sympathetic tone and the stress responses to surgery and anesthesia.

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Dexmedetomidine is a more selective α2-A receptor agonist compared to clonidine, with higher sedative and analgesic effects. Few studies have shown the efficacy of intravenous (IV) dexmedetomidine in prolonging prilocaine/bupivacaine/ropivacaine spinal anesthesia in addition to providing good sedation and postoperative analgesia. The present study was designed to evaluate the effect of IV dexmedetomidine on spinal anesthesia with 0.5% of hyperbaric bupivacaine.

MATERIALS AND METHODS

After obtaining approval from the institutional ethics committee and written informed consent from the patients, 100 patients scheduled for surgeries amenable under spinal

anesthesia in M. S. Ramaiah Medical Teaching Hospital, Bangalore, meeting the following selection criteria were included in the study.

Inclusion criteria

1. American Society of Anesthesiologists (ASA) grade I-II
2. Age <60 years

Exclusion criteria

1. ASA grade III-V
2. Patients receiving Calcium channel blockers/ angiotensin-converting-enzyme (ACE) inhibitors/ clonidine/β-blockers
3. Patients on sedative medications/opioids/ antidepressants in the week prior to surgery
4. Patients undergoing caesarean section

One hundred patients were divided into dexmedetomidine group (group D) and control group (group C) of 50 each using computer-generated random list. All the patients were pre-loaded with 10 ml/kg of lactated Ringer's solution/normal saline. Immediately after subarachnoid block with 3 ml of 0.5% hyperbaric bupivacaine, group D patients received a loading dose of 1 μg/kg of dexmedetomidine IV by infusion pump over 10 min followed by a maintenance dose of 0.5 μg/kg/h till the end of surgery, whereas the other group (group C) received an equivalent quantity of normal saline as loading and maintenance dose IV by infusion pump. Vitals were recorded (heart rate, blood pressure, SpO_2, respiratory rate) immediately after the subarachnoid block and every 5 min till the end of surgery and for 30 min after completion of surgery in post-anesthesia care unit (PACU).

Sensory blockade was checked with an alcohol swab in midaxillary line, and the time taken for the highest level of sensory blockade, two-dermatomal regression from the maximum level, and regression to S1 level was noted. Sensory blockade was assessed every 2 min for the first 10 min and thereafter every 15 min during surgery and postoperatively. All the durations were calculated considering the time of spinal injection as time 0.

Motor blockade was assessed by *modified Bromage scale* (modified Bromage 0, the patient is able to move the hip, knee, and ankle; modified Bromage 1, the patient is unable to move the hip, but is able to move the knee and ankle; modified Bromage 2, the patient is unable to move the hip and knee, but is able to move the ankle; and modified Bromage 3, the patient is unable to move the hip, knee, and ankle). Time taken for motor blockade to reach modified Bromage scale 3 and regression of motor blockade to modified Bromage scale 0 was noted. Motor blockade was assessed every 2 mins before the onset of the surgery and every 15 mins in the PACU.

The level of sedation was evaluated using *Ramsay level of sedation scale* (1, patient anxious, agitated, or restless; 2, patient cooperative, oriented, and tranquil alert; 3, patient responds to commands; 4, asleep, but with brisk response to light glabellar tap or loud auditory stimulus; 5, asleep, sluggish response to light glabellar tap or loud auditory stimulus; and 6, asleep, no response). The level of sedation was evaluated both intraoperatively and postoperatively every 15 mins using Ramsay level of sedation scale till the patient was discharged from the PACU. Excessive sedation was defined as score greater than 4/6.

Hypotension (systolic blood pressure less than 90 mm Hg or more than 20% fall from baseline value) and bradycardia (heart rate <50/min) were treated appropriately. Intraoperative requirement of supplemental analgesia (up to 1 μg/kg body weight of fentanyl) and the time for first request for postoperative analgesic were noted. Also, 20 mg/kg (maximum up to 1.2 g) IV paracetamol was given initially when the patient complained of pain. Diclofenac 75 mg in 100 ml normal saline was given as intravenous infusion if the pain persisted after 30 min of paracetamol infusion. Tramadol 50 mg slow IV was given if the pain persisted after 30 min of diclofenac administration.

Sample size

Sample size of 50 in each group was estimated using nMaster software based on the study by Al-Mustafa *et al.* who concluded that intravenous dexmedetomidine prolongs bupivacaine spinal analgesia, considering the sensory regression time to S1 segment in dexmedetomidine group (261.5 ± 34.8 min) and control group (165.2 ± 31.5 min). The precision considered was α-error as 5%, β-error as 10%, and minimum expected difference (clinically significant difference) as 20 min.

Statistical analysis

The statistical software SPSS 16 (SPSS Version 16, SPSS, Inc., Chicago) was used for the analysis of the data. χ^2 or Fisher's exact test was used to find the significance of study parameters on categorical scale and independent samples *t*-test was used for the parameters on continuous scale. Significance was assessed at 5% level of significance. *P* value <0.05 was considered significant.

RESULTS

The demographic data, ASA grade, type of surgery, and duration of surgery were comparable between the two groups [Table 1]. The total amount of dexmedetomidine

given in group D was 126.5 ± 27.4 μg (bolus 60.66 ± 11.7 μg, maintenance dose 66.3 ± 20.6 μg). The duration of sensory blockade, duration for two-dermatomal regression of sensory blockade, and the duration for motor block regression to modified Bromage scale 0 were significantly prolonged in group D [Table 1]. The level of sensory blockade was significantly higher in group D [Table 1]. No significant difference was noted in the time for attaining highest level of sensory blockade and in the time taken for motor blockade to reach modified Bromage scale 3 between the groups [Table 1].

The hemodynamic data, complications, and intraoperative atropine/mephentermine/IV fluid requirement in both the groups are summarized in Figures 1-4 and Tables 2 and 3. Significantly higher proportion of patients in group D had bradycardia and fall in systolic blood pressure more than 20% of baseline value. Systolic, diastolic, and mean arterial blood pressures were relatively lower in group D compared to group C. Higher proportion of patients in group D (26% vs. 4%; *P* value = 0.004) required atropine for management for bradycardia. Mephentermine required to treat hypotension was comparable in both the groups. None of the patients in group D had postoperative shivering compared to 10% in group C.

Intraoperative Ramsay sedation scores were significantly higher in group D (mean 4.4 ± 0.7, range 3-6) as compared to group C (mean 2 ± 0.1, range 2-3) ($P < 0.001$). Maximum scores in group D ranged from 4 to 6, with a mean of 4.68. In group D, the maximum sedation score of more than 4 was achieved in 46% of patients (23/50). Maximum scores in group C ranged

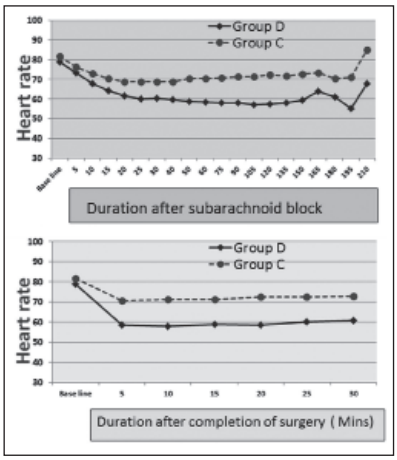

Figure 1: Line diagram comparing the baseline heart rate with intraoperative (a) and postoperative (b) heart rates between the groups

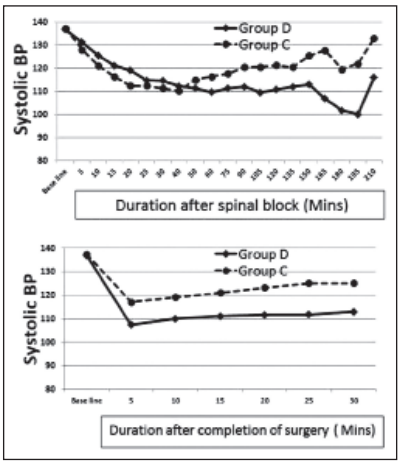

Figure 2: Line diagram comparing the baseline systolic blood pressure with intraoperative (a) and postoperative (b) systolic blood pressures between the groups

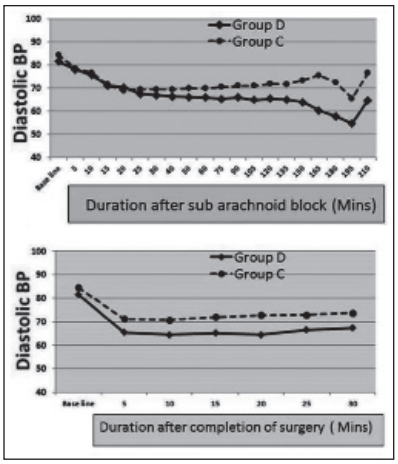

Figure 3: Line diagram comparing the baseline diastolic blood pressure with intraoperative (a) and postoperative (b) diastolic blood pressures between the groups

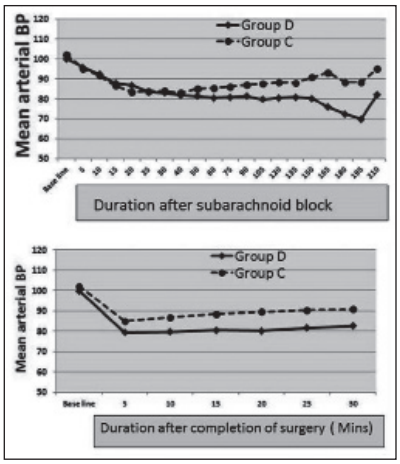

Figure 4: Line diagram comparing the baseline mean arterial blood pressure with intraoperative (a) and postoperative (b) mean arterial blood pressures between the groups

Table 1: Comparison of the demographic data, duration of surgery, motor and sensory blockade between both the groups (values are mean±standard deviations or numbers)

		Group D	Group C	*P* value
Age		40.5±13.2 years	44.9±11.4 years	0.08
Gender (male/female)		32/18	26/24	0.31
Weight		60.8±11.7 kgs	58.4±9.5 kgs	0.26
ASA grade (I/II)		36/14	34/16	0.83
Type of surgery (inguinal hernia repair/vaginal hysterectomy/arthroscopic ACL tear repair)		16/14/20	26/12/12	0.104
Duration of surgery		140.9±33.4 min	137.2±33.1 min	0.57
Sensory block	Highest level (thoracic)	T 6.88±1.1	T 7.66±0.8	<0.001
	Time for attaining highest level	11.6±1.9 mins	11.9±2.1mins	0.41
	Time for two-dermatomal regression	137.4±10.9 mins	102.8±14.8 mins	<0.001
	Duration of sensory blockade	269.8±20.7 mins	169.2±12.1 mins	<0.001
Motor block	Duration to reach modified Bromage scale 3	5.38±1.5 mins	5.04±1.9 mins	0.33
	Duration for regression to modified Bromage scale 0	220.7±16.5 mins	131±10.5 mins	<0.001

ACL: Anterior cruciate ligament tear repair

Table 2: Comparison of intraoperative/postoperative hemodynamic parameters and complications between both the groups [values are mean±standard deviations or numbers (%)]

Hemodynamic parameters/complications		Group D	Group C	*P* value
Intraoperative hemodynamic parameters	Heart rate	60.9±5.8	71.2±4.3	<0.001
	Lowest heart rate	51.7±6.4	64.1±6.2	<0.001
	Bradycardia (<50 beats/min)	15/50 (33%)	2/50 (4%)	<0.001
	Systolic blood pressure (mm of Hg)	113.2±17.4	117.3±8.8	0.14
	Lowest SBP (mm of Hg)	98.6±8.3	105.8±10.9	<0.001
	Number of patients with SBP <20% of baseline	38/50 (76%)	27/50 (54%)	0.03
	Diastolic blood pressure (mm of Hg)	68.3±7.8	71.5±4.6	0.013
	Mean arterial pressure (mm of Hg)	84.1±8.4	86.8±5.7	0.07
Postoperative hemodynamic parameters	Heart rate	59.1±5.8	71.9±4.4	<0.001
	Systolic BP (mm of Hg)	110.9±10.7	122±7.5	<0.001
	Diastolic BP (mm of Hg)	65.7±8.1	72.3±4.1	<0.001
	Mean arterial pressure (mm of Hg)	80.8±8.1	88.5±5	0.005
Complications	Postoperative shivering	0/50 (0%)	5/50 (10%)	0.06
	Postoperative nausea and vomiting	2/50 (4%)	0/50 (0%)	0.49

Table 3: Comparison of intraoperative atropine, mephentermine, and IV fluid requirement in both the groups [values are mean±standard deviations or numbers (%)]

	Group D	Group C	*P* value
Number (%) of patients requiring mephentermine for management of hypotension	7/50 (14%)	4/50 (8%)	0.52
Mephentermine requirement (mg)	1.2 (range 0-12)	0.6 (range 0-12)	0.29
Total IV fluids given during surgery	2822±534 ml	2614±307 ml	0.02
Number (%) of patients requiring atropine due to persistent bradycardia	13/50 (26%)	2/50 (4%)	0.004
Atropine requirement (mg)	0.13 (range 0-0.6)	0.02 (range 0-0.6)	<0.001

from 2 to 3, with a mean of 2.09. There was no significant difference in sedation scores between the groups in the postoperative period. Ramsay sedation scores are summarized in Figure 5.

There was no significant difference in the SpO_2 levels and respiratory rates between both the groups during surgery and in the postoperative period. None of the patients in the dexmedetomidine group required fentanyl during surgery, as compared to 3 (6%) patients in the control group (range 30-70 μg) (*P* value 0.242). Time to first request for rescue analgesic was significantly longer in the dexmedetomidine group (mean 5.27 h) as compared to the control group (mean 3 h) ($P < 0.001$). Average 24-h consumption of analgesics was significantly higher in the control group as compared to the dexmedetomidine group, as summarized in Table 4.

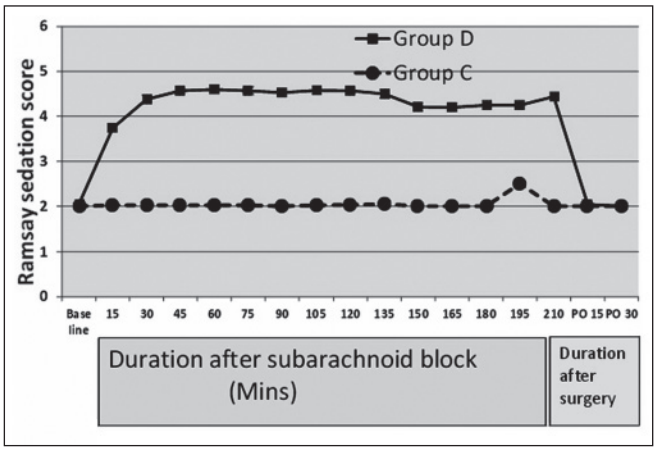

Figure 5: Line diagram comparing the baseline, intraoperative, and postoperative (PO) Ramsay sedation scores between the groups

Table 4: Comparison of 24-h postoperative analgesic requirement in both the groups [values are mean±standard deviations or numbers (%)]

First 24-h requirement	Group D	Group C	*P* value
Paracetamol (gm)	1.87±0.67 g	2.7±0.6 g	<0.001
Diclofenac (mg)	76.5±55.6	117± 40.5	<0.001
Tramadol (mg)	18 (SD 24)	51 ±35.7	<0.001
Number (%) requiring diclofenac in addition to paracetamol	37/50 (74%)	50/50 (100%)	<0.001
Number (%) requiring tramadol in addition to diclofenac and paracetamol	18/50 (36%)	38/50 (76%)	<0.001

DISCUSSION

Recent studies have shown the efficacy of both intrathecal and IV dexmedetomidine in prolonging spinal anesthesia. Prolongation of spinal anesthesia after IV dexmedetomidine is by its supra-spinal action at locus ceruleus and dorsal raphe nucleus. There are three subtypes of α2 receptors: A, B, and C. Dexmedetomidine is a more selective α2-A receptor agonist than clonidine, with more sedative and analgesic effects. Activation of presynaptic α2-A receptors at locus ceruleus decreases norepinephrine release and causes sedative and hypnotic effects, whereas its effect on descending medullo spinal noradrenergic path way results in analgesia by terminating pain signal propagation. At substantia gelatinosa of the spinal cord, it decreases firing in nociceptive neurons and release of substance P, thus producing analgesia. So, dexmedetomidine has a role in modulating pain and inhibiting the transmission and perception of pain. Activation of post-synaptic α2-A receptors in CNS results in hypotension and bradycardia by decreasing the sympathetic activity. Activation of post-synaptic α2-C receptors in CNS results in anxiolysis, whereas activation of post-synaptic α2-B receptors in peripheral vasculature results in transient hypertension.

Dexmedetomidine group had higher level of sensory block compared to the control group in our study, similar to the study results of Kaya *et al.*[7] In our study, the mean time for two-dermatomal regression of sensory blockade was significantly prolonged in the dexmedetomidine group (137.4 ± 10.9 min) compared to the control group (102.8 ± 14.8). Hong *et al.*[8] reported that the mean time to two-segment regression was prolonged in the dexmedetomidine group (78 min vs. 39 min for cold and 61 min vs. 41 min for pinprick for dexmedetomidine group and control group, respectively). Similar observations were noted by others [Kaya *et al.*[7] 145 ± 26 min vs. 97 ± 27 min ($P < 0.001$), Tekin *et al.*[4] 148.3 min vs. 122.8 min ($P < 0.001$) in the dexmedetomidine and control groups, respectively]. The duration of sensory blockade was significantly prolonged in the dexmedetomidine group (269.8 ± 20.7 min) compared to the control group (169.2 ± 12.1) in our study, similar to the results of other studies [Al Mustafa *et al.*[1] 261.5 ± 34.8 min vs. 165.2 ± 31.5 min ($P < 0.05$), Whizar-Lugo *et al.*[5] 208 ± 43.5 min vs. 137 ± 121.9 min ($P = 0.05$) in the dexmedetomidine and control groups, respectively].

In our study, the regression time to reach the modified Bromage scale 0 was significantly prolonged in the dexmedetomidine group (220.7 ± 16.5 min) compared to the control group (131.6 ± 10.5 min). Similar prolongation of motor blockade was reported in previous studies [Al Mustafa *et al.*[1] 199 ± 42.8 min vs. 138.4 ± 31.3 min ($P < 0.05$), Whizar-Lugo *et al.*[5] 191 ± 49.8 min vs. 172 ± 36.4 min (*P* value not significant), Tekin *et al.*[4] 215 min vs. 190.8 min ($P < 0.001$) in dexmedetomidine group and control group, respectively]. Elcicek *et al.*[2] and Hong *et al.*[8] also found that complete resolution of motor blockade was significantly prolonged in the dexmedetomidine group. Contrary to the above studies, Kaya *et al.*[7] reported no significant prolongation in the duration of motor block in the dexmedetomidine group compared to the control group.

Significantly higher proportion of patients in the dexmedetomidine group (33%) had bradycardia compared to the control group (4%), which is similar to the findings of other studies (Al Mustafa *et al.*[1] 16.66% vs. 8.3%, Whizar-Lugo *et al.*[5] 32% vs. 20% in dexmedetomidine group and control group, respectively). Higher proportion of patients in the dexmedetomidine group required atropine (30%) compared to the control group (4%) in our study, as was also reported in other studies (Tekin *et al.*[4] 30% vs. 6.6%, Hong *et al.*[8] 24.0% vs. 3.8% in dexmedetomidine and

control groups, respectively). Contrary to above studies, Al Mustafa *et al.*[1] reported no significant difference in atropine requirement between dexmedetomidine (9%) and control (0%) groups (*P* value 0.65).

Intraoperative and postoperative systolic, diastolic, and mean arterial blood pressures were lower in the dexmedetomidine group as compared to the control group in the present study. Eliceck *et al.*[2] reported significant decrease in mean arterial pressure in the dexmedetomidine group as compared to the control group. Previous studies have shown that the hypotensive effect of dexmedetomidine persists in the intraoperative as well as in the postoperative period.[9,10] Contrary to the above observations, Al Mustafa *et al.*[1] and Tekin *et al.*[4] reported no significant difference in mean arterial pressures in the dexmedetomidine and control groups. In our study, there was no significant difference in the number of patients requiring mephentermine for the management of hypotension in both the groups. Similarly, Tekin *et al.*[4] reported no significant difference between the groups in the number of patients who received ephedrine to treat hypotension. No significant difference in the incidence of hypotension was reported by others [Al Mustafa *et al.*[1] 0% vs. 20% (*P* value 0.15), Whizar-Lugo *et al.*[5] 8% vs. 4% in dexmedetomidine and control groups, respectively]. Total IV fluids administered in the dexmedetomidine group (2822 ± 534.2 ml) was significantly more compared to the control group (2614 ± 307.1 ml). Contrary to our study, total IV infusion was significantly more in the control group (910.8 ± 280.1 ml) compared to the dexmedetomidine group (864.5 ± 172.8 ml) in the study done by Al Mustafa *et al.*[1]

Dexmedetomidine does not cause significant respiratory depression despite providing good sedation resulting in wide safety margins.[11] In the present study, there was no significant difference in the SpO_2 levels between both the groups during surgery and in the postoperative period, similar to the study results of Al Mustafa *et al.*[1] In our study, intraoperative Ramsay sedation scores were significantly higher in the dexmedetomidine group as compared to the control group. Ramsay sedation score during surgery was 2 in all patients in the control group and ranged from 2 to 5 in the dexmedetomidine group in the study done by Al Mustafa *et al.*[1] Hong *et al.*[8] noted that the median sedation scores during surgery were 4 in the dexmedetomidine group and 2 in the control group ($P < 0.001$). Higher average sedation score in the dexmedetomidine group was also reported by others.[2,4,7]

In our study, the time to first request for postoperative analgesic was significantly prolonged and the 24-h mean requirement of analgesics was significantly less in the dexmedetomidine group compared to the control group. Similarly, Hong *et al.*[8] noticed that postoperative pain intensity was lower and the mean time to first request for postoperative analgesia was longer in the dexmedetomidine group compared to the control group (6.6 h vs. 2.1 h). Kaya *et al.*[7] in their study observed that dexmedetomidine increased the time to first request for postoperative analgesia and decreased the analgesic requirements. Whizar-Lugo *et al.*[5] in their study noticed that the time to first request for postoperative analgesic in the dexmedetomidine group was (220 ± 30 min) significantly prolonged as compared to the control group (150 ± 20 min).

Clonidine and dexmedetomidine by inhibition of central thermoregulation and attenuation of hyperadrenergic response to peri-operative stress are known to prevent postoperative shivering.[12] In our study, none of the patients in the dexmedetomidine group had postoperative shivering, as compared to 10% in the control group. Similar results were reported by Tekin *et al.*[4] (0% vs. 30% in dexmedetomidine and control groups, respectively). No significant difference in the incidence of postoperative nausea and vomiting was noted between both the groups in the present study, similar to that reported in previous studies.[1,5]

Loading dose of dexmedetomidine was given prior to surgical incision in our study. The 24-h mean analgesic requirement was less and the time to first request for postoperative analgesic was prolonged in group D than group C. As dexmedetomidine has a role in modulating pain, inhibiting the pain transmission and perception of pain, its role as a pre-emptive analgesic needs to be assessed.

CONCLUSION

IV dexmedetomidine significantly prolongs the duration of sensory and motor block of bupivacaine spinal anesthesia. Dexmedetomidine causes decrease in heart rate and mean arterial/systolic/diastolic blood pressures. The incidence of bradycardia is significantly high when IV dexmedetomidine is used as an adjuvant to bupivacaine spinal anesthesia. Dexmedetomidine-induced bradycardia is transient and responds to atropine. The changes in blood pressure are without significant clinical impact and hypotension can be easily managed with bolus of IV fluids and mephentermine. Dexmedetomidine provides excellent sedation during surgery and sedation scores reach normal within 15 min after stopping the drug. Dexmedetomidine provides significant postoperative analgesia in first 24 h after surgery and prevents postoperative shivering.

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