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Original Research Article

Evaluating the effect of dexmedetomidine premedication on the quality of subarachnoid block, haemodynamics and sedation in patients undergoing lower limb surgeries: a prospective randomized controlled trial

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ABSTRACT

Background: Present study was designed to evaluate the effect of intravenous dexmedetomidine on haemodynamics, sedation and quality of spinal anaesthesia with 0.5% hyperbaric bupivacaine.
Methods: Sixty ASA grade 1 and 2, 18-60 years aged patients scheduled for elective lower limb surgeries were randomly divided into two groups: Group C (Control) and Group D (Study), received intravenous normal saline 10ml and intravenous dexmedetomidine 1µg/kg in dilution of 10ml respectively over 10minutes duration, 10minutes before subarachnoid block with 2.5ml of 0.5% hyperbaric bupivacaine. The heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), quality of sensory and motor block and level of sedation were monitored intraoperatively and postoperatively.
Results: The heart rate was statistically significantly decreased in group D both intraoperatively and postoperatively. Intraoperative and postoperative SBP and DBP were lower in dexmedetomidine group but clinically that was insignificant. Intraoperative Ramsay sedation scores were significantly higher in dexmedetomidine group (3.49±0.240) as compared to control group (2.51±0.249) ($p<0.001$) but the patients were easily arousable. The duration of sensory blockade (208.83±9.53 min vs 162.83±9.62 min), duration for 2 dermatomal regression of sensory blockade (146.5±10.013min vs 98±8.57min) and the duration for motor block regression to Modified Bromage scale 0 (167.33±10.5min vs 137.83±11.94min) were significantly prolonged in dexmedetomidine group as compared to control group. The highest level of sensory blockade was also significantly higher in dexmedetomidine group (T6.90±0.759 vs T7.60±0.621). There was no difference in the time for attaining highest level of sensory blockade, time taken for motor blockade to reach Modified Bromage Scale 3 between both the groups. Average 24hr mean VAS score was significantly lower in dexmedetomidine group (1.37±0.15 vs1.72±0.17, $p<0.001$). Time to first request for rescue analgesic was also significantly longer in dexmedetomidine group (mean 174.33min vs 143.5min). Average 24hour consumption of tramadol analgesic was significantly higher in control group as compared to dexmedetomidine group (391.86±111.62mg vs 279.86±80.55mg, $p<0.001$).
Conclusions: Intravenous dexmedetomidine significantly prolongs the duration of sensory and motor block of bupivacaine spinal anaesthesia. It provides excellent sedation and analgesia. Dexmedetomidine induced decrease in heart rate, systolic/diastolic blood pressure are not clinically significant.

Keywords: Dexmedetomidine, Hyperbaric bupivacaine, Modified bromage scale, Ramsay sedation score, Spinal anaesthesia

INTRODUCTION

Spinal anaesthesia is a commonly used technique in anaesthesia practice for gynaecological, lower abdominal, pelvic and lower limb surgeries. Bupivacaine, a pipecoloxylidide derivative is commonly used drug for subarachnoid block. It is appropriate for procedures lasting for 2-2.5hours.

If the duration of surgery is prolonged, spinal anaesthesia has to be supplemented with an intravenous anaesthetic agent or converted into general anaesthesia. To overcome this, adjuvants like epinephrine, phenylephrine, magnesium sulphate, sodium bicarbonate, neostigmine and alpha 2 agonists like clonidine, dexmedetomidine have been used intrathecally.[1]

Clonidine and dexmedetomidine are also used intravenously to prolong the duration of the spinal anaesthesia.[2-6] Apart from providing sedation and analgesia, they also decrease sympathetic tone and decrease the stress responses to surgery and anaesthesia. Dexmedetomdine is a more suitable adjuvant to spinal anaesthesia compared to clonidine as it has more sedative and analgesic effects due to its more selective alpha 2A receptor agonist activity. Present study was designed to evaluate the effect of intravenous dexmedetomidine on bupivacaine spinal anesthesia.

METHODS

The study was conducted in Department of Anaesthesia, M.B.Government Hospital affiliated to RNT Medical College, Udaipur, Rajasthan. Sixty patients aged between 18-60 years of ASA grade I-II scheduled for elective lower limb surgeries under spinal anaesthesia were included in this study.

Exclusion criteria

Body weight >120Kg, height <140cm, spinal deformity, post spine surgeries, history of allergy to study drugs, pregnancy, cardiovascular disease, renal disease, respiratory disease, coagulopathies, neurological disorders and refusal for spinal anaesthesia.

Sample size

Sample size was estimated based on the study by Al Mustafa et al Considering the power of 90% (β 0.10) and confidence interval of 95% (α 0.05) and minimum expected difference (clinically significant difference) as 30 min, a minimum sample size of 26 patients in each group is required. 30 patients were taken in each group to compensate for dropouts.[2]

Statistical analysis

Results on continuous measurements were presented as Mean ±SD and results on categorical measurements were presented in Number (%). Chi-square test was used to find the significance of study parameters on categorical scale between two or more groups. Paired samples T test was used to find the significance of study parameters on continuous scale within the group (intra group analysis) on metric parameters. Student t-test (two tailed, independent samples), ANOVA was used to find the significance of study parameters on continuous scale between two and more groups (inter group analysis) on metric parameters. Significance was assessed at 5% level of significance. P value <0.05 was considered significant. The data was analysed by Statistical Software Epi Info 6.

Study design

The participants in this prospective, randomized, double blinded clinical comparative study were randomly divided into two groups by a computer-generated randomization table.

Group C (Control): Patients received intravenous normal saline 10ml (as placebo) over 10minutes, 10minutes before subarachnoid block with 0.5% hyperbaric bupivacaine 2.5ml.

Group D (Study): Patients received intravenous dexmedetomidine 1µg/kg in dilution of 10ml over 10minutes, 10minutes before subarachnoid block with 0.5% hyperbaric bupivacaine 2.5ml.

An anaesthesiologist (Person A) prepared the study drugs, another anaesthesiologist (Person B) noted the observations intraoperatively as well as up to 24hours postoperatively. The third anaesthesiologist (Person C) was responsible for study drugs administration (intravenous and intrathecal) to the patients. Person B, C and the patient were kept unaware of the drug injected to enable double-blinding.

Pre-surgical protocol

The day prior to surgery all patients were asked to undergo a detailed pre-anaesthetic evaluation. The patients were advised to fast the night prior to surgery and received tablet alprazolam 0.25mg and tablet ranitidine 150mg orally on the previous night and morning of surgery.

Surgical protocol

On day of surgery, a written informed consent was obtained from each patient. Intravenous access was secured, and infusion of Ringer's lactate solution started. On arrival in OT, routine non-invasive monitoring was applied, and vital signs were monitored. After preloading the patients with Ringer Lactate 15ml/kg, midline lumbar puncture was performed at L3-4 level with quincke type 25gauge spinal needle with patients in the sitting position, under full aseptic precaution. Bupivacaine hyperbaric 2.5ml solution was injected intrathecally over

30seconds after confirming free flow of CSF. Patients were kept in supine position to achieve bilateral block. As per the group allocation, the patients received injection dexmedetomidine 1µg/kg or normal saline in dilution of 10ml intravenously over 10minutes by infusion pump 10minutes before spinal anaesthesia.

Level of sensory loss was assessed by pin-prick test at every minute till it reaches the highest level, upto 15 min and then every 15minutes during surgery and postoperatively.

Grading score for sensory block used was-Grade 0 (Normal sensation), Grade 1 (Blunted sensation) and Grade 2 (No sensation). Grade 2 was taken as onset of sensory block. Time to two dermatomal regression and regression to S1 segment was also noted.

Systolic blood pressure, diastolic blood pressure, heart rate and oxygen saturation (SpO2) were monitored regularly before and after subarachnoid block, in every 5min intraoperatively as well as till 30min postoperatively then at 6hr, 12hr, 18hr and 24hr postoperatively.

Any fall in the heart rate below 60 beats per minute was treated with incremental doses of injection atropine 0.4mg intravenous and systolic blood pressure below 90mmhg or fall greater than 20% of baseline was treated with mephenteramine 6mg intravenously. Postoperative pain was assessed using the visual analogue scale7 (VAS), was assessed every 30min for 6hours, then every 2hours up to 12hours and 6hourly up to 24hours.

Table 1: Visual analogue score.

Visual analogue scale[7]	
pain intensity	Word scale
0	No pain
1-2	Least pain
3-4	Mild pain
5-6	Moderate pain
7-8	Severe pain

When pain score>3, Injection tramadol 2mg/kg intravenously was administered as rescue analgesic. Time for first rescue analgesic was noted and total 24hr analgesic requirement was calculated.

Modified Bromage scale was used to access motor blockade[8]

- Bromage 0- the patient is able to move the hip, knee and ankle;
- Bromage 1- the patient is unable to move the hip, but is able to move the knee and ankle;
- Bromage 2- the patient is unable to move the hip and knee, but is able to move the ankle;
- Bromage 3-The patient is unable to move the hip, knee and ankle.

Motor blockade was assessed every min till it reaches to modified Bromage 3 up to 15min and then postoperatively every 30min till the modified Bromage scale score returns to zero.

Level of sedation was assessed by using Ramsay sedation score at every 15min intraoperatively and every 30min postoperatively till four hours. Excessive sedation was noted as Ramsay score greater than 4/6.[9]

Ramsay Sedation Scale

- Patient anxious, agitated, or restless,
- Patient cooperative, oriented, and tranquil alert,
- Patient responds to commands,
- Asleep, but with brisk response to light glabellar tap or loud auditory stimuli,
- Asleep, sluggish response to light glabellar tap or loud auditory stimulus,
- Asleep, no response.

All durations were calculated using time of spinal injection time as zero. Surgery was initiated when the level of sensory block reached to T10 thoracic dermatome level or above and attainment of complete motor block (Modified Bromage-3). Otherwise it was considered as failed spinal and alternate technique of anaesthesia was chosen and case was excluded from study.

RESULTS

There was no statistically significant difference in demographic distribution (age, gender, weight) among the two groups. ASA grade of patients was also comparable in both the groups with no statistically significant difference.

The site of surgery as well as duration of surgery were comparable among both the groups (87.50±18.27min in group C, 88.33±18.16min in group D, p =0.87) (Table 2).

Table 2: Demographic data.

	Group C	Group D	P value
Age (years)*	37±12.4	31.8 ±11.6	0.104
Gender (m/f)	23/7	24/6	0.754
Weight (kg)*	58.8 ± 10.8	55.5 ± 8.8	0.256
Duration of surgery(min)*	87.50±18.27	88.33±18.16	0.87
Asa grading (I/II)	21/9	20/10	0.828

*Results in mean ±standard deviation

Heart rate

There was significant decrease in the heart rate intraoperatively in both the groups as compared to baseline values (p<0.001) (Table 3). The heart rate was also significantly less in dexmedetomidine group in postoperative period although it was clinically not significant.

Table 3: Comparison of the heart rates within groups.

Group	Heart Rate (HR)		P value
Group C	Base line HR 84.06 ± 2.72	Intraoperative HR 80.63 ± 2.85	<0.001
		Postoperative HR 81.17±4.37	0.003
Group D	Base line HR 83.56±3.00	Intraoperative HR 72.23±2.77	<0.001
		Postoperative HR 73.81±2.48	<0.001

Intraoperative heart rate was significantly lower in dexmedetomidine group as compared to control group from 15-75min. Lowest intraoperative heart rate (mean) was significantly lower in dexmedetomidine group [64.60±5.00] as compared to control group [73.56±5.3] (p value <0.001). Higher number of patients in dexmedetomidine group (4/30) had intraoperative heart rate <60/min as compared to control group (2/30).

However, bradycardia was transient in most of these patients and most of these patients responded well to atropine. The average postoperative heart rate was significantly lower in dexmedetomidine group as compared to control group throughout the postoperative period.

Systolic blood pressure

There was statistically significant decrease in the systolic blood pressure intraoperatively in both the groups as compared to baseline values although that was clinically not important (Table 4).

Intraoperative systolic blood pressure was significantly lower in dexmedetomidine group as compared to control group from 10 min to 85 min.

There was significant difference in lowest intraoperative SBP in dexmedetomidine group 103.88±5.23mmhg as compared to control group 111.56±7.78mmhg (p value <0.047). Higher number of patient in dexmedetomidine group (4/30) developed hypotension compared to control group (2/30).

The average postoperative SBP was lower in dexmedetomidine group as compared to control group but was not statistically significant.

Diastolic blood pressure

There was significant decrease in the diastolic blood pressure intraoperatively in dexmedetomidine group only as compared to baseline value (Table 5).

Table 4: Comparison of systolic blood pressure within groups.

Group	Systolic blood pressure (SBP) mmHg		P value
Group C	Base line SBP 126.47±3.32	Intraoperative SBP 124.04± 2.66	0.003
		Postoperative SBP 129.59±3.91	0.002
Group D	Base line SBP 126.63±4.31	Intraoperative SBP 117.10±2.98	<0.001
		Postoperative SBP 126.15±3.68	0.64

Table 5: Comparison of the diastolic blood pressure within groups.

Group	Diastolic blood pressure (DBP) mmHg		P value
Group C	Base line DBP 81.43 ± 4.11	Intraoperative DBP 79.52± 3.86	0.069
		Postoperative DBP 81.24±1.73	0.82
Group D	Base line DBP 80.83±3.52	Intraoperative DBP 76.73±3.52	<0.001
		Postoperative DBP 79.38±3.33	0.107

Intraoperatively, the diastolic blood pressure (DBP) in dexmedetomidine group was less as compared to control group which attained statistical significance at certain time intervals although clinically it was insignificant. The average postoperative diastolic BP was lower in dexmedetomidine group as compared to control group, but it was clinically insignificant.

Oxygen saturation- SpO2

There was no significant difference in SpO2 levels between both the groups during surgery and in the postoperative period

Ramsay sedation score (RSS)

Intraoperative Ramsay sedation scores were significantly higher in dexmedetomidine group (mean 3.49±0.240) as compared to control group (mean 2.51±0.249) (p<0.001). Maximum scores in dexmedetomidine group was 3.96±0.56 at 75min and then it declined, whereas the maximum score in control group was 2.97±0.76 at 60min

(Table 6). The sedation score remained statistically significant (p<0.001) in dexmedetomidine group till 60min of postoperative period.

Table 6: Ramsay sedation Scores.

		Ramsay sedation score (mean±SD)		P value
		Group C	Group D	
Base line		1.90±.30	1.73±.45	0.098
Duration after subarachnoid block	15	2.03±.18	2.77±.56	<0.001
	30	2.47±.57	3.50±.50	<0.001
	45	2.97±.66	3.73±.52	<0.001
	60	2.97±.76	3.70±.46	<0.001
	75	2.44±.76	3.96±.56	<0.001
	90	2.07±.26	3.56±.72	<0.001
	10	2.00±.00	3.43±.53	<0.001
	120	2.00±.00	3.00±.00	<0.001
Duration after completion of surgery	30	2.00±.00	3.27±.58	<0.001
	60	2.00±.00	2.70±.53	<0.001
	90	2.10±.30	2.23±.43	0.171
	120	2.03±.18	2.03±.32	1.00
	150	1.97±.18	2.00±.26	0.57
	180	2.07±.25	2.00±.45	0.48
	210	1.97±.32	2.00±.00	0.57
	240	1.93±.52	1.80±.40	0.27

Duration of sensory and motor blockade

The duration of sensory blockade, duration for 2 dermatomal regression of sensory blockade and the duration for motor block regression to Modified Bromage scale 0 were significantly prolonged in dexmedetomidine group as compared to control group (p<0.001) (Table 7). The highest level of sensory blockade was significantly higher in dexmedetomidine group (p<0.001). There was no difference in the time for attaining highest level of sensory blockade, time taken for motor blockade to reach Modified Bromage Scale 3 between both the groups.

Postoperative analgesia

Average 24hour mean VAS score was lower in dexmedetomidine group 1.37±0.15 as compared to control group 1.72±0.17 and difference was statistically significant (p<0.001).

Time to first request for rescue analgesic

Time to first request for rescue analgesic was significantly longer in dexmedetomidine group (mean 174.33min) as compared to control group (mean 143.5min, p <0.001).

Total analgesic requirement within 24hour after surgery

Average 24hour consumption of tramadol analgesic was significantly higher in control group (391.86±111.62mg) as compared to dexmedetomidine group (279.86±80.55mg) and was statistically significant (p<0.001).

Postoperative nausea and vomiting

Postoperative vomiting was noted in 2 of 30 patients in each group.

Table 7: Comparison of sensory and motor blockade in both groups.

	Group C (min)	Group D (min)	P value
Highest level of sensory block (t-thoracic)	T 7.6±0.621	T 6.90±0.759	<0.001
Time for attaining highest level of sensory block	9.60±0.675	9.23±0.898	0.079
Duration for 2 dermatomal regression of sensory blockade	98±8.57	146.5±10.013	<0.001
Duration of sensory blockade	162.83±9.621	208.83±9.53	<0.001
Duration for motor blockade to reach modified bromage scale 3	5.37±0.890	5.07±0.740	0.161
Duration for motor block regression to modified bromage scale 0	137.83±11.94	167.33±10.5	<0.001

DISCUSSION

Spinal anaesthesia remains one of the basic techniques in modern anaesthesia since its introduction into clinical practices.

Different drugs like epinephrine, phenylephrine, adenosine, magnesium sulphate, sodium bicarbonate, neostigmine and alpha2 agonists like clonidine, dexmedetomidine have been used as adjuvants to local anaesthetics to prolong the duration of spinal anaesthesia. Among the alpha2 agonists, Dexmedetomidine is a more suitable adjuvant to spinal anaesthesia compared to clonidine as it has more sedative and analgesic effects due to its more selective alpha 2A receptor agonist activity.Recent studies have shown the efficacy of both intrathecal and intravenous dexmedetomidine in prolonging spinal anaesthesia and analgesia. Systemic and intrathecal injection of dexmedetomidine produces analgesia by acting at laminae VII and VIII of ventral horns in spinal cord. The drug also acts at locus ceruleus

and dorsal raphe nucleus to produce sedation and analgesia. This supra spinal action explains the prolongation of spinal anaesthesia after intravenous dexmedetomidine.

In the present study, the time for attaining highest level of sensory block was decreased in dexmedetomidine group but was statistically insignificant (9.23±0.898minutes vs 9.60±0.675minutes). In contrast, Dinesh et al used dexmedetomidine 1µg/kg as loading dose and 0.5µg/kg/hr. as infusion intraoperatively till completion of surgery and found decreased time for attaining peak sensory level (11.6±1.9minutes) in dexmedetomidine group as compared to control group (11.9±2.1minutes) and that was statistically significant.[10,11]

In our study, the highest level of sensory block was significantly higher in demedetomidine group compared to control group (T6.90±0.759 vs T 7.6±0.621, p<0.001) similar to study by Annamalai et al (T4.5±0.5 vs T6.3±0.8) and Hamed et al group (mean T6 vs mean T8).[11,12]

In our study, the mean time for two dermatomal regression of sensory blockade was significantly prolonged in dexmedetomidine group compared to control group (146.5±10.013minutes vs 98±8.57minutes, p <0.001) similar to study by Hamed et al (105 ±39minutes vs 70 ± 22minutes, p < 0.001) and Lee et al (C-57.6±23.2 vs 86.5±24.3 @ 0.5µg/kg intravenous dexmedetomidine v/s 92.5±30.7minutes@1.0µg/kg intravenous dexmedetomidine, p= 0.0002).[13]

In present study the duration of sensory blockade i.e. time for regression to S1 dermatome was significantly prolonged in dexmedetomidine group compared to control group (p < 0.001). In study by Hamed et al they also found prolonged duration of sensory block in dexmedetomidine group (263±75min) compared to control group (181±45minutes) (p value <0.001).[12]

These results in present study may be due to fact that dexmedetomidine has an inhibitory effect on the locus ceruleus (A6 group) located at the brain stem. This supraspinal action explains the prolongation of spinal anesthesia after intravenous administration of dexmedetomidine. The noradrenergic innervation of the spinal cord arises from the noradrenergic nuclei in the brain stem including the locus ceruleus, the A5 and the A7 noradrenergic nuclei. Neurons in the locus ceruleus are connected to the noradrenergic nuclei in the brain stem. Axon terminals of the noradrenergic nuclei reach lamina VII and VIII of the ventral horns of the spinal cord. The activity of the noradrenergic neurons is decreased by agonists acting at α2-adrenergic receptors on the locus ceruleus cell bodies.

Therefore, inhibition of the locus ceruleus results in disinhibition of the noradrenergic nuclei and exerted descending inhibitory effect on nociception in the spinal cord. In addition to dexmedetomidine's action in the locus ceruleus of the brain stem, it has been shown to stimulate α2 receptors directly in the spinal cord, thus inhibiting the firing of nociceptive neurons. Even peripheral α2 adrenoceptors may mediate anti nociception.

In the present study there was lower but no significant difference in time taken for motor blockade to reach modified Bromage Scale 3 in both the groups (5.07±0.740 minutes vs 5.37±0.890 minutes, p=0.161).

Time to reach modified Bromage 3 was lower but not significant in dexmedetomidine group (5.38±1.5min) as compared to control group (5.04±1.9min) (p value 0.327); in study done by Dinesh et al.[10]

However, in present study, the regression time to reach the modified Bromage Scale 0 scale was significantly prolonged in dexmedetomidine group (167.3±11.65minutes) compared to control group (137.8±11.9minutes, p < 0.001); similar to study by Hamed et al (210±32minutes vs 152±41 minutes, p< 0.001), But contrary to these studies, Kaya et al reported no significant prolongation in the duration of motor block in dexmedetomidine group compared to control group.[12,14] It may be due to lower dose of dexmedetomidine (0.5 mcg/kg) used in that study.[10]

There is some evidence that clonidine results in direct inhibition of impulse conduction in the large, myelinated Aα fibers and the 50% effective concentration (EC 50%) measured approximately 4 folds of that in small, unmyelinated c fibers. This could explain the less prolonged motor block compared with sensory block, as conduction of motor nerve fibers was less inhibited than sensory nerve fibre at the same concentration of clonidine. The same process might be applied to dexmedetomidine, and would explain the more sensory than motor block prolongation in present study.

In present study the mean intraoperative heart rate from 15 to 75min was significantly lower in dexmedetomidine group (72.23±2.77) as compared to control group (80.63±2.85) (p< 0.001). Hamed et al also found mean heart rate was significantly lower in dexmedetomidine group from 20 min to 60min intraoperatively. Lower duration in their study may be due to lower dose of intravenous dexmedetomidine (0.5µg/kg) compared to our study (1.0µg/kg).[12]

In our study higher proportion of patients in dexmedetomidine group (4/30- 13.3%) had bradycardia (HR <60/min) as compared to control group [2/30-6.1%]; but this was statistically not significant. Moreover, bradycardia was transient in nature and responded well to atropine. This may be because we have given dexmedetomidine in infusion over 10minutes, so it may have decreased the incidence of bradycardia compared to bolus dexmedetomidine. Decrease in heart rate in present

study may be related to decrease in plasma catecholamine and the sympathetic outflow caused by α2-adrenergic receptor activation by dexmedetomidine.

In our study, the average intra operative systolic blood pressure (SBP) after spinal block was significantly lower in dexmedetomidine group (117.10±2.98mmhg) as compared to control group (124.04±2.66 mmHg) from 10-85minutes intraoperatively. The average postoperative SBP was also lower in dexmedetomidine group (127.72±3.71mmHg) as compared to control group (129.59±3.91mm Hg) but was statistically not significant. There was significant difference in intraoperative DBP in dexmedetomidine group (76.73±3.52mmHg) compared to control group (79.52±3.86 mmHg).

In a study by Annamalai et al, they found significant decrease in mean arterial pressure from 5-120minutes intra operatively in both groups.[11] Eliceck et al reported significant decrease in mean arterial pressure after 30min of dexmedetomidine infusion as compared to control group. Contrary to it, Hamed et al observed that there was decrease in systolic and diastolic blood pressure intraoperatively but was statistically not significant. It may be because they had used lower doses of dexmedetomidine 0.5µg /kg in study group.[4,12]

In present study despite providing good sedation, dexmedetomidine does not cause significant respiratory depression, providing wide safety margins. There was no significant difference in SpO2 levels between both the groups during surgery and in the postoperative period similar to the study of Annamalai et al, Mustafa et al.[2,11]

In our study intraoperative Ramsay sedation scores were significantly higher in dexmedetomidine group (mean 3.49±0.24) as compared to control group (mean 2.51±0.24). Maximum scores in dexmedetomidine group ranged from 3-5 with a mean of 4.1 but the patient was easily arousable and SpO2 never declined. Maximum scores in control group ranged from 2-4 with a mean of 3.4. There was significant difference in sedation scores between the groups in the postoperative period in present study (2.25±0.19) compared to control group (2.00±0.11).

In present study patients were easily arousable after giving intravenous dexmedetomidine. This may be due to fact that dexmedetomidine affects the locus ceruleus area of brain, which induces sedation resembling natural sleep by means of sleep modulation and respiration control. It is related with cooperative sedation, which is different from the clouding of consciousness that occurs with drugs that act on GABA receptors, such as Propofol and midazolam.

Dexmedetomidine inhibits the release of substance P from the dorsal horn of the spinal cord, leading to primary analgesic effects. Dexmedetomidine was found to be effective in providing postoperative analgesia in the present study. The time to first request for postoperative analgesic was significantly prolonged in dexmedetomidine group (174.33±17.0minutes) as compared to control group (143.5±15.65minutes, p<0.001). Total 24hr tramadol requirement was significantly lower in study group (279.86±80.55mg) as compared to control group (391.86±111.62mg). The number of rescue analgesic doses in 24hr was also significantly lower in study group (2.5±0.57) compared to control group (3.3±0.71). Mean 24hr VAS score was lower in dexmedetomidine group (1.37±0.15) compared to control group (1.72± 0.17).

In study by Hamed et al the time to first request for postoperative analgesic was significantly prolonged in dexmedetomidine group (3.29±0.85hr) as compared to control group (0.232±0.13hr) (p value <0.05) as well as the rescue analgesic requirement in 24hr was significantly decreased in study group compared to control group (142.84±13.06mg vs 309.98±12.06mg, p<0.05).[12]

Hong et al noticed that post-operative pain intensity was lower and the mean time to first request for post-operative analgesia was longer in the dexmedetomidine group compared to the control group (6.6hour v/s. 2.1hour).[15] Since, dexmedetomidne has a role in pain modulation, inhibition of pain transmission as well as pain perception, its use as routine pre emptive analgesic needs to be considered. Patients with advanced age, cardiovascular diseases and other comorbidities were excluded from the study hence our findings cannot be extrapolated in these patients. Further studies are needed to investigate the efficacy of dexmedetomidine in geriatric patients or medically compromised patient populations. In this study, we investigated the effects of dexmedetomidine on spinal anaesthesia at a single dose of 1.0ug/kg. Hence, it is difficult to interpret the dose-response relationship of the dexmedetomidine dose and the duration of spinal anaesthesia. Further studies are needed to determine the dose-response relationship

CONCLUSION

Intravenous dexmedetomidine significantly prolongs the duration of sensory and motor block of bupivacaine spinal anaesthesia. It provides excellent sedation during surgery as well as postoperatively.

Dexmedetomidine is effective in providing significant postoperative analgesia in first 24 hours. Dexmedetomidine causes significant decrease in heart rate, mean arterial/systolic/diastolic blood pressures. Dexmedetomidine induced bradycardia is transient and responds well to atropine. The changes in blood pressure are without significant clinical impact.

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