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Original Research Article

To Study the Effects of Intravenous Dexmedetomidine on Characteristics of Subarachnoid Block Using Hyperbaric Bupivacaine in Lower Limb Orthopaedic Surgery

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ABSTRACT

Background: We studied the effects of intravenous dexmedetomidine on characteristics of subarachnoid block with respect to sensory block, motor block, duration of postoperative analgesia and complications encountered.

Materials and methods: Hundred patients of American Society of Anesthesiologists (ASA) grade I or II (20 – 50 years) presenting for lower limb orthopaedic surgery were included in the study. All patients received 2.5 ml of 0.5% hyperbaric bupivacaine intrathecally followed by: Group D (n=50) - Loading dose of 1 µg kg^{-1} dexmedetomidine over 10 minutes started 20 minutes after spinal block + maintenance dose of 0.4 µg kg^{-1} hr^{-1} dexmedetomidine till the end of surgery; Group P (n=50) - same calculated volume of normal saline as loading dose over 10 minutes + maintenance till end of surgery. Data regarding the onset and regression of sensory and motor block, VAS score, duration of analgesia, sedation score, haemodynamic parameters and complications were recorded.

Results: The time of two segment regression, regression to S_2 dermatome and time of VAS ≥ 4 was more in group D than in group P ($p< 0.001$). Patients in group D had a significantly higher sedation score than group P ($p< 0.001$). Dexmedetomidine significantly reduced the post-operative requirement of diclofenac injection ($p < 0.001$). No other complications were observed in the two groups.

Conclusion: Intravenous dexmedetomidine after spinal block resulted in significant prolongation of time to two segment regression of sensory block, motor block and time to VAS ≥ 4; reduced postoperative analgesic requirement and good sedation levels with maintenance of haemodynamic parameters.

Key words: Adjuvant, Dexmedetomidine, Subarachnoid block.

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INTRODUCTION

Spinal anaesthesia is a commonly used technique for lower limb orthopaedic surgeries. However, postoperative pain control is a major problem because spinal anaesthesia using only local anaesthetics is associated with relatively short duration of postoperative analgesia and early intervention is required for it in the postoperative period. Alpha$_2$-agonists have been used as adjuvants by intrathecal, epidural, caudal, intravenous routes and for peripheral nerve blocks. They potentiate the effect of local anaesthetics and prolong the duration of both motor, sensory spinal blockade and postoperative analgesia.[1]

Dexmedetomidine belongs to the imidazole subclass of α_2-receptor agonists similar to clonidine. It shows a high ratio of specificity for the α_2-receptor (α_2/α_1 1600:1) compared with clonidine (α_2/α_1 200:1), making it a complete α_2-agonist. The α_2-agonists produce their sedative-hypnotic effects by an action on α_2-receptors in the locus ceruleus.[2] Dexmedetomidine has been found to exert its analgesic actions both at the spinal and supraspinal levels.[3] Analgesic and sedative properties have been found when intrathecal, epidural or intravenous dexmedetomidine is used as an adjuvant.[1]

We aimed to study the characteristics of motor and sensory block and the complications following the administration of intravenous dexmedetomidine as an adjuvant to intrathecal hyperbaric bupivacaine.

MATERIAL AND METHODS

The present study was conducted in a prospective double-blind randomised manner. Hundred patients of ASA grade I or II (20 – 50 years) presenting for lower limb orthopaedic surgery were included in the study. Patients having any contraindications to

spinal anaesthesia, known allergy to study drug, heart block / dysrhythmia and patients on treatment with α-adrenergic antagonists were not included in the study.

After obtaining informed consent and ethical clearance, patients were preloaded with lactated Ringer's solution at 15 ml kg^{-1} and were monitored for non-invasive blood pressure (NIBP), pulse oximetry (SpO_2) and electrocardiogram (ECG).

All patients received 2.5 ml of 0.5% hyperbaric bupivacaine intrathecally. Patients were randomly allocated on the basis of a sealed envelope technique to receive one of the following after subarachnoid block:

Group D (n=50) - Loading dose of 1 µg kg^{-1} dexmedetomidine over 10 minutes started 20 minutes after spinal block + maintenance dose of 0.4 µg kg^{-1} hr^{-1} dexmedetomidine till the end of surgery. Group P (n=50) - same calculated volume of normal saline as loading dose over 10 minutes + maintenance till end of surgery.

Oxygen was administered via a facemask. Hypotension defined as decrease in systolic blood pressure by more than 20% from baseline or less than 90 mm Hg was treated with incremental intravenous (IV) doses of ephedrine 3 mg along with of IV fluids as required. Bradycardia defined as heart rate (HR) less than 50 bpm was treated with IV atropine 0.6 mg.

Primary outcome variables like the highest level of sensory block and time to reach this level, time of two segment regression, time of regression to S_2 dermatome, highest Bromage score, duration of motor block and sedation score were recorded. Secondary outcome variables like time to VAS ≥ 4, intraoperative requirement of ephedrine and atropine, time to void and postoperative analgesic requirement were also noted. Assessment of onset and regression of motor block was done according to Bromage scale.[4]

Grade	Criteria	Degree of block
0	Free movement of legs and feet	Nil (0%)
1	Just able to flex knees with free movement of feet	Partial (33%)
2	Unable to flex knees, but with free movement of feet	Almost complete (66%)
3	Unable to move legs or feet	Complete (100%)

Sedation was assessed according to the Modified Wilson Sedation Scale.[5]

Score	Description
1	Oriented; eyes may be closed but can respond to "Can you tell me your name?" "Can you tell me where you are right now?"
2	Drowsy; eyes may be closed, arousable only to command: "(name), please open your eyes."
3	Arousable to mild physical stimulation (earlobe tug)
4	Unarousable to mild physical stimulation

Incidence of side effects like nausea, vomiting, shivering, pruritus, respiratory depression, bradycardia and hypotension was also recorded. Postoperatively pain scores were recorded using Visual Analog Scale (VAS) between 0 and 10 (0= no pain, 10= most severe pain) at every 30 minutes for 3 hours. Injection diclofenac 75 mg intramuscular was given as rescue analgesia when VAS ≥ 4. The patient was observed for 24 hours postoperatively for the need of analgesic requirement. At the end of the study, the data thus obtained was compiled and analysed statistically using:

- Unpaired t-test for quantitative data.
- Chi-square test / Fisher's exact test for qualitative data.

The value of $p< 0.05$ was considered as statistically significant, $p< 0.01$ as highly significant and $p< 0.001$ as very highly significant for statistical analysis.

Table 1: Comparison of Demographic Parameters (Mean ± S.D.)

	GROUP D	GROUP P	p-value
Age (yrs)	34.32 ± 9.95	33.28 ± 9.89	0.301
Weight (kg)	66.00 ± 9.77	68.80 ± 9.18	0.072
Height (mt)	1.69 ± 0.06	1.71 ± 0.07	0.187
Duration of surgery (min)	93.74 ± 34.71	87.26 ± 27.12	0.150

Table 2: Distribution of Highest Level Of Sensory Block

Group	Highest sensory level							Mean ± S.D.
	T_4	T_5	T_6	T_7	T_8	T_{10}	T_{12}	
D	6	3	27	0	11	3	0	**6.38 ± 1.51**
P	4	8	25	1	9	2	1	**6.34 ± 1.59**
p-value								**0.449**

OBSERVATIONS AND RESULTS

All the patients in both the study groups belonged to ASA status I. There were 43 male and 7 female patients in group D; 45 male and 5 female patients in group P ($p= 0.269$). The age, weight, height, duration of surgery and mean maximum sensory block achieved in both the groups was also comparable. (Table 1,2)

The time of two segment regression, regression to S_2 dermatome and time of VAS ≥ 4 was more in group D than in group P ($p< 0.001$). Patients in group D had a significantly higher sedation score than group P ($p< 0.001$). Dexmedetomidine significantly reduced the post-op requirement of diclofenac injection ($p< 0.001$). (Table-3)

The mean basal values of haemodynamic data for both the groups were statistically comparable. The infusions were continued during episodes of hypotension and/or bradycardia and the severity of these effects did not warrant stoppage of infusions at any point of time. No other complications like dizziness, fatigue, pruritus, tremors, headache etc. were observed in the two groups.

Table 3: Comparison of Different Observations Between The Two Groups

Parameter		Group D	Group P	p-value
Time to highest level of sensory block (min)		8.56 ± 2.03	8.76 ± 2.02	0.311
Time to two segment regression (min)		100.50 ± 26.73	83.40 ± 20.51	< 0.001
Time to S_2 regression (min)		321.90 ± 47.55	236.90 ± 30.17	< 0.001
Time of VAS ≥ 4 (min)		268.10 ± 57.36	194.00 ± 26.80	< 0.001
Highest Bromage scale		2.78 ± 0.42	2.82 ± 0.39	0.311
Duration of motor block (min)		196.00 ± 51.70	147.30 ± 25.34	< 0.001
No. of patients having	**Hypotension**	10	6	0.138
	Bradycardia	9	5	0.125
	Shivering	2	4	0.200
Dose of ephedrine (mg)	**None**	40	44	0.138
	3	6	5	0.375
	6	4	1	0.084
Dose of atropine (mg)	**None**	41	45	0.125
	0.6	9	5	0.125
Sedation score	**1**	0	50	
	2	13	0	
	3	36	0	
	4	1	0	
	Mean ± S.D.	2.76 ± 0.48	1.00 ± 0.00	< 0.001
Time to void (hr)		6.65 ± 0.89	5.03 ± 0.67	< 0.001
No. of diclofenac injections	**1**	23	4	
	2	24	38	
	3	3	8	
	Mean ± S.D.	**1.60 ± 0.61**	**2.08 ± 0.49**	< 0.001

DISCUSSION

Our observations are consistent with Harsoor et al who noted that the median level of cephalad spread of sensory blockade in group D was T_{10} (T_8 – T_{12}) compared with T_8 (T_6 – T_{10}) in group C which was not found to be significant (p= 0.362). They observed that the time required for two segment regression was significantly prolonged in group D (111.52 ± 30.9 minutes) compared with group C (53.6 ± 18.22 minutes), (p< 0.001). The duration of analgesia (time to VAS ≥ 3) was significantly prolonged in group D as compared to group C (222.8 ± 123.4 minutes vs 138.36 ± 21.62 minutes, p< 0.001) despite using a lower initial loading dose of 0.5 µg kg^{-1}. They explained this analgesic effect primarily due to inhibition of locus ceruleus at the brain stem and increased activation of α_2-receptors at the spinal cord resulting in inhibition of nociceptive impulse transmission. This effect seems to be mediated through both pre-synaptic and the post synaptic α_2-receptors. They also found that the complete regression of motor blockade took longer time in group D (256.44 ± 53.10 minutes) compared with group C (231.16 ± 32.2 minutes), (p< 0.001). They argued that the effect of clonidine on motor blockade was concentration dependant and the same theory might explain this phenomenon with dexmedetomidine as well. The prolongation of motor block in spite the use of 0.5 µg kg^{-1} initial loading dose, observed by them may be attributed to continuous infusion following loading dose. The incidence of shivering was comparable between their two study groups (1 patient in group D vs 5 patients in group C, p= 0.095). The mean intra-operative RSS in Group D was 2.34 ± 1.1 where as in Group C, it was 2.0 ± 0.0 (p= 0.034). However, RSS was comparable in both groups in the postoperative period. Dexmedetomidine produces sedation by its central effect and this seems to be dose dependant. Most of the patients receiving dexmedetomidine were sedated, but easily arousable. None of the patients had RSS greater than 3 at any point of observation highlighting the advantage of lower dose.[6]

Whizar-Lugo et al also noted that the mean time to reach the highest cephalad dermatome level was 15 minutes in all groups. sensory block duration was longer in both dexmedetomidine and clonidine groups, 208 ± 43.5 minutes and 225 ± 58.8 minutes respectively v/s placebo group 137 ± 121.9 minutes (p= 0.05) noted that the postoperative need for analgesics provided at VAS 4/10, was first given in the placebo group at 150 minutes, dexmedetomidine patients received their first analgesic dose at 220 minutes, and clonidine patients at 240 minutes after the end of surgery (dexmedetomidine vs placebo 220 ± 30 minutes vs 150 ± 20 minutes, p< 0.05 and clonidine vs placebo 240 ± 20 minutes vs 150 ± 20 minutes, p< 0.05). No statistical differences were found between dexmedetomidine vs clonidine (p> 0.05). They explained that systemic or neuraxial injection of α_2-adrenergic agonists produces analgesia by acting at the spinal level, laminae VII and VIII of the ventral horns. The most accepted mechanism is the release of acetylcholine and nitric oxide. The locus ceruleus and the dorsal raphe nucleus are also important central neural structures where these drugs act producing sedation-analgesia. They noted that the motor block duration was longer in dexmedetomidine and clonidine groups (191 ± 49.8 minutes and 192 ± 63.4 minutes) v/s placebo group (172 ± 36.4 minutes) without significant statistical difference. Our observations are consistent with their results.[7]

Al-Oweidi et al. also observed that the time to regression to S_1 dermatome was significantly prolonged in group D in comparison to group P and C. The regression time to S_1 was 149.4 ± 14.6 minutes in group C, 152.8 ± 16.6 minutes in group P and 209.6 ± 25.9 minutes in group D, (p< 0.0001). They explained that dexmedetomidine produces analgesia by binding to

adrenoceptors in the spinal cord and the prolongation of spinal analgesia after intravenous dexmedetomidine could be due to its inhibitory effect on the locus ceruleus (A6 group) which is located at the brain stem. They noted that regression time to Bromage 0 scale was 184.6 ± 22.8 minutes in group C, 190.0 ± 21.0 minutes in group P and 255.8 ± 36.7 minutes in group D (p< 0.0001). Also the need to give ephedrine and atropine were comparable in their three study groups.[8]

Al-Mustafa et al observed significant prolongation of time to S_1 regression in group D as compared to group C (261.5 ± 34.8 minutes vs 165.2 ± 31.5 minutes, p< 0.0001). They also observed significant prolongation of regression time to Bromage scale 0 in group D as compared to group C. The regression time to reach the Bromage scale 0 was 138.4 ± 31.3 minutes in group C and 199.9 ± 42.8 minutes in group D (p< 0.0001). The need to give ephedrine and atropine were comparable in the two groups (p= 0.60, p= 0.65 respectively). Ramsay sedation score (RSS) was 2 in all patients in group C, and ranged from 2 – 5 in group D, the maximum score was 5 in three patients, 4 in nineteen patients and 3 in one patient, and the maximum mean score of sedation (3.96 ± 0.55) was achieved 30 minutes after starting dexmedetomidine infusion. They explained that dexmedetomidine produces sedation and anxiolysis by binding to α_2-receptors in the locus ceruleus, which diminishes the release of norepinephrine and inhibits sympathetic activity, thus decreasing heart rate and blood pressure.[9]

In our study the mean basal values of haemodynamic data for both the groups were comparable. There was a significant fall in SBP, DBP and PR in group D as compared to group P at different time intervals (p< 0.05). The SpO_2 and RR in the two groups were found to be comparable at different time intervals (p> 0.05). The infusions were continued during episodes of hypotension and/or bradycardia and the severity of these effects did not warrant stoppage of infusions at any point of time. In our study hypotension was noted in 10 patients in group D and 6 patients in group P, which was statistically insignificant (p= 0.138). Nine patients in group D and 5 patients in group P had bradycardia, which was also statistically insignificant (p= 0.125). Al-Mustafa et al also observed that the incidence of hypotension and bradycardia in the intraoperative and PACU time were comparable in both groups (p= 0.15, p= 0.46 respectively). They explained that dexmedetomidine has an onset of action of 30 minutes when the maintenance dose is used intravenously. Standard loading dose of (1 μg kg^{-1} hr^{-1} infused over 10 minutes) decreases the onset of action of dexmedetomidine. The side effects of dexmedetomidine such as hypotension and bradycardia are dose dependent. Infusion of loading dose over 10 minutes and then infusing the maintenance dose decreases the incidence of these side effects.[9]

The mean time to void was 6.65 ± 0.89 hours in group D and 5.03 ± 0.67 hours in group P which was found to be very highly significant (p< 0.001). There is no available study yet in which the effect of intravenous dexmedetomidine has been studied on the time to void. This prolongation in group D can be explained due to prolongation of sensory block. The number of injection diclofenac required for pain relief in 24 hours postoperative period was significantly less in group D as compared to group P (1.60 ± 0.61 vs 2.08 ± 0.49) (p< 0.001).Complications like dizziness, fatigue, pruritus, tremors, headache etc. were not observed in the two groups.

CONCLUSION

After studying various factors we conclude that loading dose of 1 μg kg^{-1} dexmedetomidine over 10 minutes started 20 minutes after spinal block followed by maintenance dose of 0.4 μg kg^{-1} hr^{-1} till the end of surgery resulted in significant prolongation of time to two segment regression, sensory block and motor block with maintenance of haemodynamic parameters and a reduced postoperative analgesic requirement. Dexmedetomidine resulted in good sedation levels in all the patients without significant respiratory depression and complications. So we conclude that intravenous dexmedetomidine can be used as an adjuvant to SAB when prolongation of spinal anaesthesia is desired.

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