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ARTIGO ORIGINAL / ORIGINAL ARTICLE

Effects of Intravenous Dexmedetomidine on Sensory and Motor Block Characteristics in Adults Undergoing Infraumbilical Surgery Under Subarachnoid Block

Efeitos da Dexmedetomidina Intravenosa nas Características do Bloqueio Sensorial e Motor em Adultos Submetidos à Cirurgia Infraumbilical sob Bloqueio Subaracnóideo

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Keywords
Anesthesia, Spinal; Dexmedetomidine/therapeutic use; Nerve Block.

Palavras-chave
Bloqueio Nervoso; Dexmedetomidina/uso terapêutico; Raquianestesia.

ABSTRACT

Introduction: Subarachnoid block is enhanced by intrathecal administration of adjuvants (opioids, α2-agonists, etc.) with bupivacaine. A potent α2-agonist, dexmedetomidine, causes sedation and analgesia when administered intravenously. Its intrathecal use is not approved. This study was designed to evaluate the effects of intravenous dexmedetomidine on subarachnoid block (SAB).

Methods: Sixty patients of the American Society of Anesthesiologists grade 1-2, of either sex, aged 18–60 years, undergoing infraumbilical surgery under SAB were randomized into two groups, group-C receiving 50 mL normal saline and group-D receiving dexmedetomidine infusion (0.5 mcg/kg in 50 mL normal saline) over 15 minutes within two minutes of SAB. The onset and duration of sensory and motor blocks were measured. Heart rate (HR), systolic (SBP), diastolic (DBP), and mean arterial pressure (MAP) were taken preoperatively, at SAB, 2, 4, 6, 8, 10, 15, 20, 25, 30, 45, 60, 90, and 120 minutes after SAB. Intraoperative sedation was assessed pre-drug infusion (post-SAB) and 30 and 60 minutes after SAB. Postoperative pain and sedation were assessed at 1, 2, 4, 8, 12 and 24 hours.

Results: Group-D showed a faster onset of sensory block and longer sensory and motor block than group-C (p<0.05). The onset of motor block was comparable between the groups. Group-C showed higher HR, SBP, DBP, and MAP than group-D (p<0.05). Group-D had higher sedation scores and less postoperative pain than group-C (p<0.05).

Conclusion: Dexmedetomidine causes a faster onset of sensory block, prolonged sensory and motor blocks with stable hemodynamics, better sedation, and postoperative analgesia.

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RESUMO

Introdução: O bloqueio subaracnóide é potenciado pela administração intratecal de adjuvantes (opióides, α2-agonistas, etc.). Um potente α2-agonista, a dexmedetomidina, causa sedação e analgesia quando administrada por via intravenosa. O seu uso intratecal não está aprovado. Este estudo foi desenhado para avaliar os efeitos da dexmedetomidina intravenosa no bloqueio subaracnóide (BSA).

Métodos: Sessenta doentes de grau 1-2 da Sociedade Americana de Anestesiologistas, de ambos os sexos, com idades compreendidas entre os 18-60 anos, submetidos a cirurgia infraumbilical sob BSA, foram randomizados em dois grupos: o grupo-C, que recebeu 50 mL de soro fisiológico, e o grupo-D, que recebeu uma infusão de dexmedetomidina (0,5 mcg/kg em 50 mL de soro fisiológico) durante 15 minutos após o BAS. O início e a duração dos bloqueios sensitivos e motores foram medidos. A frequência cardíaca (FC), a pressão sistólica (PAS), a pressão diastólica (PAD) e a pressão arterial média (PAM) foram registadas no pré-operatório, no momento do BSA e 2, 4, 6, 8, 10, 15, 20, 25, 30, 45, 60, 90 e 120 minutos após o BSA. A sedação intraoperatória foi avaliada antes da infusão do fármaco (pós-BSA) e 30 e 60 minutos após o BSA. A dor e a sedação pós-operatórias foram avaliadas às 1, 2, 4, 8, 12 e 24 horas.

Resultados: O Grupo-D apresentou início de bloqueio sensitivo mais rápido e maior duração do bloqueio sensitivo e motor que o Grupo-C (p<0,05). O Grupo-C apresentou FC, PAS, PAD e PAM mais elevados que o Grupo-D. A sedação foi maior e a dor menor no Grupo-D (p<0,05).

Conclusão: A dexmedetomidina causa início de bloqueios sensitivos mais rápidos, bloqueios sensitivos e motores mais prolongados com estabilidade hemodinâmica, melhor sedação e analgesia pós-operatória.

INTRODUCTION

Subarachnoid block (SAB) is the preferred mode of anesthesia for infraumbilical surgery. Hyperbaric bupivacaine is a commonly administered local anesthetic drug in SAB. Drugs like opioids (fentanyl), alpha-2 (α_2) agonists (clonidine), etc., have usually been

added with the local anesthetic agent as adjuvants to prolong the duration and quality of sensory and motor blocks.[1]

Dexmedetomidine is an α_2-adrenergic agonist with more selective action on α_2-receptors than clonidine.[2] It has sedative, analgesic, sympatholytic and opioid-sparing effects on the human body.[3] It directly stimulates α_2-receptors located in the substantia gelatinosa of the dorsal horn in the spinal cord,[2] and inhibits the release of substance-P.[4] The Food and Drug Administration (FDA) has not approved intrathecal use of dexmedetomidine. The existing literature showed the analgesic effects of intravenous infusion of dexmedetomidine in patients receiving general anaesthesia.[5] However, there is a paucity of literature on the effects of intravenous dexmedetomidine on block characteristics in patients undergoing infra-umbilical surgery under spinal anesthesia.

This study was planned to assess the effects of intravenous dexmedetomidine on sensory and motor block characteristics in patients receiving SAB.

MATERIAL AND METHODS

This prospective, randomized, and comparative study was performed with prior approval from the institutional ethics committee (IEC/VMMC/SJH/Thesis/2019-10/180 dated 30-10-2019) following principles outlined in the 'Helsinki Declaration, 2013'. Sixty patients of either sex, aged between 18 and 60 years, and American Society of Anesthesiologists (ASA) physical status 1-2, undergoing infraumbilical surgery under SAB were enrolled after obtaining written informed consent. Patients with a history of hypersensitivity to dexmedetomidine, a baseline heart rate ≤ 60 beats per minute or receiving beta blocker therapy, on chronic hypnotic and antihypertensive medications, or with a history of hypertension, diabetes mellitus, autonomic dysfunction, obstructive sleep apnea, pregnancy or lactation were excluded from the study.

The primary objective of the study was to assess the effects of intravenous dexmedetomidine on the onset and duration of sensory and motor blocks in patients receiving spinal anesthesia, and the secondary objectives were to observe the effects of intravenous dexmedetomidine on hemodynamic parameters (heart rate, systolic, diastolic, and mean blood pressure), sedation scores, and postoperative pain.

All participants received tab alprazolam (0.25 mg) the night before and tab ranitidine (150 mg) in the morning of surgery on an empty stomach. After a thorough pre-anesthetic check-up and ensuring adequate preoperative fasting as per ASA guidelines, all participants were randomized into two groups, group-C (participants receiving normal saline) and group-D (participants receiving intravenous dexmedetomidine), using a computer-generated randomization technique. Intravenous access was secured with an 18 G cannula, and Ringer's Lactate infusion was started at 8 mL/kg/h. All patients were taken to the operating theatre and ASA standard monitors were attached. Baseline parameters like heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded. SAB was given 2.8 mL hyperbaric bupivacaine in a sitting position at the level of L3-L4 interspace in a midline approach using 25G spinal needles after confirmation of free flow of clear CSF using aseptic techniques. Thereafter, the patients were made supine and group-D patients received an injection of dexmedetomidine at 0.5 mcg/kg body weight diluted in 50 mL of normal saline over 15 minutes and group-C patients received 50 mL of normal saline over 15 minutes, within two minutes of administration of the spinal drug. All patients received supplemental oxygen at 6 L per minute via a facemask.

The following parameters were noted:

i) The onset of sensory block: Sensory block was assessed with the help of a spirit-soaked swab, and the onset of sensory block was determined as the time required for the sensory block to reach the T10 level from the spinal anesthesia time.

ii) The onset of motor block: The motor block was assessed by the Modified Bromage scale. The scale ranges from '0' to '3' (0 - no motor weakness, 1- inability to raise a straight leg, but knee and foot can be moved, 2 - only the foot can be moved, 3 - total paralysis). The onset of a motor block was defined as the time taken for the motor block to attain the Modified Bromage score of '3' from the spinal drug injection time.

iii) The duration of sensory block: It was estimated by the time taken for the two-segment dermatomal regression from the T10 dermatomal level.

iv) The duration of motor block: It was defined as the time taken for regression of the Modified Bromage score from 3 to 2.

v) HR, SBP, DBP, and MAP at preoperative period (baseline/ T0), at the time of SAB (T1), and after 2 (T2), 4 (T4), 6 (T6), 8 (T8), 10 (T10), 15 (T15), 20 (T20), 25 (T25), 30 (T30), 45 (T45), 60 (T60), 90 (T90), and 120 (T120) minutes of SAB.
Intravenous mephentermine was given in increments of 3 mg if the MAP falls below 20% of the baseline value or MAP <65 mmHg. Intravenous atropine (0.6 mg) was given if the HR was ≤50 beats per minute.

vi) Level of sedation: It was assessed using the Ramsey Sedation Scale (RSS) just after spinal anesthesia and before the study-drug infusion (baseline), after 30 and 60 minutes of SAB, and postoperatively at 1, 2, 4, 8, 12 and 24 hours. RSS ranges from 1-6, where 1 represents the most awake and alert state and 6 indicates the deepest level of sedation or 'no response'.[6]

vii) Postoperative pain: It was assessed using the Visual Analog Scale (VAS) at 1, 2, 4, 8, 12 and 24 hours, ranging from 0 "no pain" to 10 "maximum pain".[7] Intravenous infusion of paracetamol (1g) was given six hourly postoperatively for 24 hours to all patients. Slow infusion of tramadol (50 mg) was given as rescue analgesia if the VAS score was more than 4.

In cases of prolonged surgery (more than 120 minutes), or if surgery required conversion to general anesthesia, or in any intraoperative adverse events like bleeding, etc., the case was excluded from the study.

The sample size was calculated as per the study of Reddy VS *et al*.[8] They observed that times of onset of sensory and motor blocks were 4.26 ±1.37 minutes and 4.57±0.83 minutes, respectively, in the control group, and 2.91±1.16 minutes and 3.64±0.75 minutes, respectively, in the dexmedetomidine group. Taking these values as reference, the minimum required sample size with 95% power of the study and 5% level of significance was 23 patients in each study group. To reduce the margin of error, the total sample size taken was 60 (30 patients per group).

The data was entered into a computer-based spreadsheet and analyzed using SPSS 30.0. The statistical analysis consisted of calculating means and proportions. The Chi-square was used to test the statistical association of the usage of dexmedetomidine or normal saline with gender and ASA grade. The Mann-Whitney U test was performed to compare age, BMI, hemodynamic parameters (HR, SBP, DBP & MAP), sedation assessment using the RSS and pain assessment using the VAS in both groups. The level of significance was taken as $p<0.05$. Graphs were made using Microsoft Excel 2016.

RESULTS

Sixty patients fulfilled the inclusion criteria. No dropouts occurred from any group. The patient characteristics (age, gender, and ASA physical status) and the duration of surgery were comparable in both groups. Group-D had a faster mean onset time of sensory block than group-C ($p<0.05$). The mean onset time of motor block was comparable between the groups. The duration of both motor and sensory block was statistically longer in group-D than group-C ($p<0.05$) [**Table 1**].

Post-spinal and before the study drug infusion, the baseline sedation scores were comparable in both groups. However, group-D showed statistically higher sedation scores than group-C intraoperatively (30 and 60 minutes after SAB), and postoperatively at 1, 2, 4, 8, and 12 hours ($p<0.05$). The 24-hour postoperative sedation scores were comparable between the groups. None of the patients in either group had pain in the first hour in the postoperative period. Group-C had statistically higher pain scores than group-D in 2, 4, 8, and 12 hours postoperatively ($p<0.05$). The pain scores were comparable between the groups 24 hours postoperatively [**Table 1**].

The baseline heart rates were comparable between the groups at T0. Group-C showed statistically higher heart rates than group-D at T8, T15, T20, and T25 ($p<0.05$). There was no statistically significant difference found in heart rates at other time-points, and none of the patients in both the

Table 1. Comparison of patients' characteristics of motor and sensory block, intra and postoperative sedation scores and postoperative pain scores.

Parameters		Group D (n= 30)	Group C (n= 30)	*P*-value
Age (years)		34.13 ± 10.34	35.07 ± 11.93	0.58
Male, n (%) Female, n (%)		18 (60%) 12 (40%)	21 (70%) 09 (30%)	0.42
BMI (kg/m^2)		22.10 ± 2.83	23.16 ± 1.98	0.25
ASA I, n (%) ASA II, n (%)		24 (54.5%) 06 (37.5%)	20 (45.5%) 10 (62.5%)	0.24
Onset of sensory block (min)		4.07 ± 0.36	5.13 ± 1.00	**< 0.01**
Onset of motor block (min)		4.33 ± 1.29	4.26 ± 0.69	0.87
Duration of sensory block (min)		170.00 ± 20.74	124.86 ± 20.82	**< 0.01**
Duration of sensory block (min)		144.00 ± 48.82	122.00 ± 53.39	**0.03**
Duration of surgery (min)		83.86 ± 11.28	86.33 ± 12.71	0.42
Intra-operative Ramsay Sedation Scale Score	Post SAB, before drug	2.00 ± 0.37	2.00 ± 0.00	1.00
	30 min after SAB	3.53 ± 0.57	2.17 ± 0.38	**< 0.01**
	60 min after SAB	3.40 ± 0.56	2.77 ± 0.43	**< 0.01**
Postoperative Ramsay Sedation Scale Score	1 h	3.03 ± 0.18	1.77 ± 0.43	**< 0.01**
	2 h	2.77 ± 0.62	1.60 ± 0.50	**< 0.01**
	4 h	2.93 ±0.52	1.33 ± 0.48	**< 0.01**
	8 h	2.67 ± 0.48	1.50 ± 0.51	**< 0.01**
	12 h	2.50 ± 0.51	1.47 ± 0.50	**< 0.01**
	24 h	1.87 ± 0.43	1.8 ± 0.41	0.54
Postoperative Visual Analog Scale (VAS)	1 h	0.00 ± 0.00	0.00 ± 0.00	1.00
	2 h	0.00 ± 0.00	2.00 ± 1.57	**<0.01**
	4 h	0.63 ±0.76	4.60 ± 1.67	**<0.01**
	8 h	0.77 ± 1.00	4.30 ± 1.18	**<0.01**
	12 h	2.17 ± 0.87	4.57 ± 1.10	**<0.01**
	24 h	3.83 ± 0.91	3.97 ± 0.71	0.71

Data is expressed as mean ± standard deviation (SD) or n, percentage. BMI: body mass index; Kg: kilogram; m^2: meter square; SAB: Subarachnoid block.

groups showed bradycardia (HR< 60 bpm) or tachycardia (HR >100 bpm) [**Table 2**]. The baseline MAP values were comparable between the groups at T1. Group-C showed statistically higher MAP than group-D at T2, T6, T8, T10, T15, T20, T25, and T30 (p<0.05). There was no statistically significant difference found in MAP at other time-points between the groups [**Table 3**].

Group-C showed statistically higher SBP than group-D at T4, T6, T8, T10, T15, T20, T25, T30, T60, T90 and T120 (p<0.05) [**Fig. 1**]. The differences in DBP were statistically significant between group-C and D at T2, T6, T8, T10, T45, and T120 [**Fig. 2**].

DISCUSSION

Adjuvants can improve the duration and quality of spinal anesthesia. Clonidine, an α_2-agonist, is used intrathecally as an adjuvant in spinal anesthesia. However, dexmedetomidine is a very potent α_2-agonist with sedative and analgesic properties,[3] but its intrathecal administration is not widely approved. The common route of administration for dexmedetomidine is intravenous. The effects of intravenous dexmedetomidine on SAB characteristics have not been extensively studied.

In the present study, the mean onset time of sensory block was significantly faster in group-D than in group-C (p<0.05), while the mean onset time of motor block was comparable

Table 2. Comparison of heart rates (beats per minute) at different time intervals.

Time-points	Group D (n= 30)	Group C (n= 30)	*P*-value
Baseline	82.20 ± 8.65	78.50 ± 6.94	0.07
At SAB	82.20 ± 8.65	83.33 ± 8.61	0.71
2 min	82.73 ± 6.92	87.03 ± 12.31	0.24
4 min	76.80 ± 10.37	82.70 ± 8.70	0.06
6 min	76.87 ± 10.00	78.83 ± 7.77	0.34
8 min	78.80 ± 6.67	82.70 ± 6.67	**0.04**
10 min	77.17 ± 6.67	77.47 ± 8.86	0.52
15 min	70.50 ± 5.88	82.07 ± 6.26	**<0.01**
20 min	71.47 ± 9.03	81.13 ± 5.91	**<0.01**
25 min	72.90 ± 6.96	79.63 ± 7.98	**< 0.01**
30 min	76.86 ± 2.94	77.87 ± 8.55	0.54
45 min	76.10 ± 5.46	79.20 ± 7.12	0.06
60 min	74.66 ± 4.17	76.57 ± 6.51	0.18
90 min	75.70 ± 4.62	76.57 ± 6.51	0.55
120 min	79.17 ± 7.46	80.40 ± 6.78	0.52

Data is expressed as mean ± standard deviation (SD). SAB: Subarachnoid block..

Table 3. Comparison of mean arterial pressure (mmHg) at different time intervals.

Time-points	Group D (n= 30)	Group C (n= 30)	*P*-value
Baseline	89.20 ± 7.90	87.37 ± 7.70	0.367
At SAB	86.4 ± 7.39	91.13 ± 8.73	**0.027**
2 min	79.33 ± 6	84.6 ± 5.86	**0.001**
4 min	76.47 ± 5.38	77.47 ± 5.26	0.470
6 min	70.3 ± 7.52	78.4 ± 7.29	**<0.001**
8 min	70.07 ± 5.26	80.87 ± 6.07	**<0.001**
10 min	73.67±5.3	81.7 ± 4.01	**<0.001**
15 min	80.27 ± 4.69	85.3 ± 4.28	**<0.001**
20 min	80.53 ± 5.15	85.3 ±4.28	**<0.001**
25 min	80.27 ± 4.69	85.3 ± 4.28	**<0.001**
30 min	82.33 ± 10.15	89.03 ±6.42	**0.003**
45 min	85.03 ±7.92	88.93 ± 5.04	**0.027**
60 min	79.6 ± 5.08	84.47 ±7.11	**0.003**
90 min	79.9 ± 5.78	85 ± 7.47	**0.004**
120 min	80.27 ± 5.6	81.77 ± 4.25	0.247

Data is expressed as mean ± standard deviation (SD). SAB: Subarachnoid block.

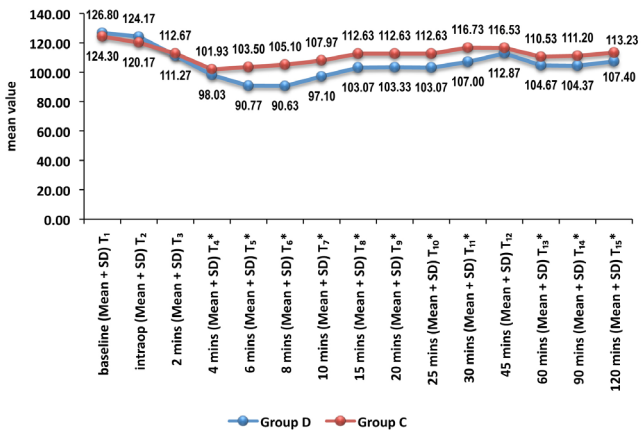

Figure 1. Comparison of trend of systolic blood pressure (mmHg) at different time intervals between group-D and group-C.

(* indicates *p*-value <0.05)

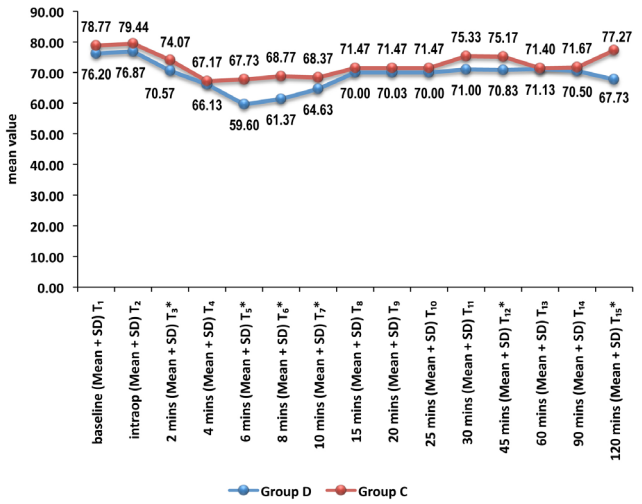

Figure 2. Comparison of trend of diastolic blood pressure at different time intervals between group-D and group-C.

(* indicates *p*-value <0.05)

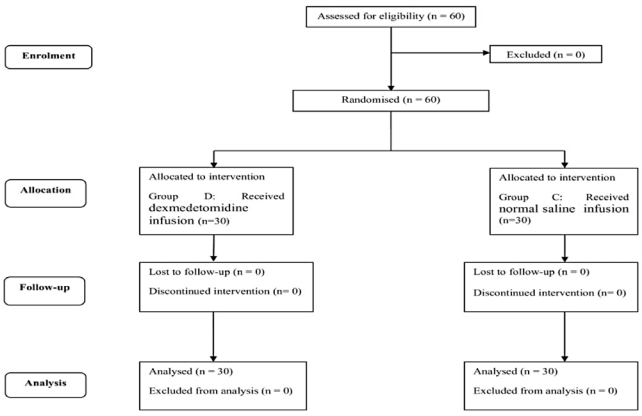

Figure 3. CONSORT flow diagram.

between the groups. Group-D showed a longer duration of motor and sensory blocks than group-C (p<0.05). Group-D had a more stable hemodynamic profile than group-C. Group-D had statistically lower postoperative pain scores than group-C at 1, 2, 4, 8, and 12 hours postoperatively (p<0.05). The sedation scores were higher in group-D than in group-C intraoperatively (30 and 60 minutes after SAB), and again at 1, 2, 4, 8, and 12 hours postoperatively (p<0.05). Kaya FN *et al* conducted a study on three groups receiving dexmedetomidine (0.5 µg/kg) or midazolam, or saline infusion before spinal anesthesia. The dexmedetomidine group showed a longer sensory block than the saline group, though the duration of motor block was comparable between the groups. Postoperative pain was less in the dexmedetomidine group compared with the other groups. The sedation score was higher in the dexmedetomidine group than in the saline group.[9] Reddy VS *et al* also compared three groups, dexmedetomidine (0.5 µg/kg), clonidine (1.0 µg/kg) and placebo before subarachnoid block. They found a higher sensory block level, better analgesia, and a higher sedation score with dexmedetomidine than in the other groups.[8] The findings of these studies supported the findings of the present study, except that the duration of motor block was significantly higher in the dexmedetomidine group, unlike the findings of the study by Kaya FN *et al.*

Kumari R *et al* studied intravenous infusion of dexmedetomidine (1 µg/kg over 10 min, followed by a maintenance dose of 0.6 µg/kg) on spinal anesthesia and found it to be effective in prolonging both sensory and motor block. The dexmedetomidine group had a higher sedation score than the control (p<0.05), which supported the present study findings. Kumari R *et al* found bradycardia in 26.7% of cases in the dexmedetomidine group, requiring atropine therapy, unlike the present study.[10] In the present study, the dexmedetomidine group had a more stable heart rate profile, and none of the patients had bradycardia requiring intravenous atropine. Bhirud PH *et al* compared the effects of two maintenance infusion doses of dexmedetomidine, 0.25 mcg/kg/h and 0.5 mcg/kg/h, following a loading dose of 0.5 µg/kg dexmedetomidine over 10 min on subarachnoid block. They found that the maintenance infusion dose of 0.5 µg/kg/h significantly prolonged the duration of motor block and the time for two-segment regression, along with stable hemodynamics and adequate sedation.[11] Sekar EB *et al* studied the effect of dexmedetomidine (a loading dose of 1 µg/kg followed by an infusion of 0.5 µg/kg) given 15 minutes after spinal anesthesia. They did not find any change in the onset time of sensory and motor blocks. They concluded that intravenous dexmedetomidine prolongs the duration of sensory and motor blocks, provides sedation with easy arousability and enhances analgesia postoperatively while maintaining hemodynamic stability with no significant side effects.[12] Kavya UR *et al* compared three groups, one group received saline infusion over 10 minutes before spinal anesthesia followed by saline infusion over 60 minutes, the second group received intravenous dexmedetomidine (1 µg/kg) over 10 minutes before spinal anesthesia followed by saline infusion over 60 minutes, and the third group received intravenous dexmedetomidine (0.5 µg/kg) over 10 minutes before spinal anesthesia followed by dexmedetomidine (0.5 µg/kg) infusion over 60 minutes. They concluded that dexmedetomidine given as a bolus or bolus plus infusion prolongs both sensory and motor

blockade of spinal anaesthesia.[13] The above study findings are consistent with the present study, except that the mean onset time of sensory block was faster in the dexmedetomidine group than in the control group in the present study, unlike the study by Sekar EB *et al*.

Moreover, in the present study, dexmedetomidine was administered at a bolus dose of 0.5 µg/kg over 15 minutes, two minutes after subarachnoid block. Verghese T *et al* studied the effects of intravenous dexmedetomidine bolus (0.5 µg/kg) on subarachnoid block, and they found it to be effective in prolonging the duration of sensory and motor blocks with arousable sedation, without any respiratory depression.[14] Sahoo A *et al* compared two loading doses of dexmedetomidine: 0.5 and 1.0 µg/kg administered intravenously over 10 minutes after subarachnoid block. They concluded that both loading doses of dexmedetomidine were clinically efficacious in prolonging the duration of analgesia of spinal anaesthesia.[15] Thakkar PD *et al* studied dexmedetomidine (0.5 µg/kg) over 10 minutes on SAB characteristics. They found that dexmedetomidine hastens the onset of sensory and motor blocks and prolongs the duration of sensory and motor blocks with better postoperative analgesia. It also decreases the heart rate and blood pressure while maintaining arousable sedation without respiratory depression.[16]

The present study followed a uniform methodology in all the groups to avoid bias from various confounding factors as much as possible, although this study is not free from limitations. A fixed-dose spinal drug was administered to all participants irrespective of their height, weight and gender differences. These factors may affect the block level.[17] Moreover, the maximum block level was not taken into consideration. The block level could determine the duration of the block, and a more cephalad spread could result in more hemodynamic instability.[17] Future research may explore further information regarding the effects of intravenous dexmedetomidine on SAB for critical analysis, comparison, and correlation with the present study findings.

CONCLUSION

Based on the findings of the present study, it can be concluded that administration of intravenous dexmedetomidine at a dose of 0.5 µg/kg over 15 minutes after subarachnoid block results in a faster onset of sensory block, a prolonged sensory and motor blocks, and more stable hemodynamics. It also provides better sedation and superior postoperative analgesia.

DECLARAÇÃO DE CONTRIBUIÇÃO / *CONTRIBUTORSHIP STATEMENT*

Todos os autores são responsáveis por contribuições substanciais para a concepção ou o desenho do trabalho. Todos os autores aprovaram a versão final a ser publicada.

All authors are responsible for substantial contributions to the conception or design of the work. All authors approved the final version to be published.

Responsabilidades Éticas

Conflitos de Interesse: Os autores declaram a inexistência de conflitos de interesse na realização do presente trabalho.

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Confidencialidade dos Dados: Os autores declaram ter seguido os protocolos da sua instituição acerca da publicação dos dados de doentes.

Proteção de Pessoas e Animais: Os autores declaram que os procedimentos seguidos estavam de acordo com os regulamentos estabelecidos pela Comissão de Ética responsável e de acordo com a Declaração de Helsínquia revista em 2024 e da Associação Médica Mundial.

Proveniência e Revisão por Pares: Não comissionado; revisão externa por pares.

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