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Original Research Article

EFFECT OF INTRAVENOUS DEXMEDETOMIDINE ON PROLONGING SPINAL ANAESTHESIA - A PROSPECTIVE RANDOMIZED CONTROLLED STUDY

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ABSTRACT

Background: Spinal anesthesia is commonly used in gynecological and lower abdominal surgeries. However, it can have a limited duration, and adjuvants are sometimes needed to extend analgesia. The most popular adjuvant is dexmedetomidine, a selective alpha-2 adrenergic receptor agonist. It also has minor antidepressant, sedative, analgesic, and sympatholytic effects. This study was designed to evaluate the efficacy of dexmedetomidine as an adjuvant at the dose of (0.5 µg/kg) for spinal anesthesia.

Materials and Methods: This prospective randomized controlled study included 60 patients scheduled for abdominal or vaginal hysterectomy under spinal anesthesia. There was random allocation of cases to group A (n=30) and group B (n=30). Group A received intravenous dexmedetomidine (0.5 ug/kg over 10 minutes) group B received normal saline, and hemodynamic parameters, sensory and motor block characteristics, sedation scores, and adverse effects were assessed and compared between groups A and B.

Results: The baseline demographic profile distributions were similar across both groups. Dexmedetomidine significantly reduced heart rate and mean arterial pressure at various intervals ($p<0.001$) because of its sympatholytic effects. Sensory and motor block durations were significantly longer in the dexmedetomidine group A as compared to group B (255.2 ± 8.6 min vs. 210.8 ± 33.1 min and 243.6 ± 17.0 min vs. 211.2 ± 16.7 min, respectively; $p<0.001$). Two-dermatome regression time was prolonged in group A. At 160 minutes, 86.7% of the dexmedetomidine group maintained Bromage Grade 3, with 100% retaining a sensory level at or above T8. Sedation scores were significantly higher without respiratory depression.

Conclusion: Intravenous dexmedetomidine at 0.5 µg/kg significantly prolongs spinal anesthesia, enhances sedation, and maintains hemodynamic stability with minimal adverse effects. It is a valuable adjuvant for improving intraoperative and postoperative outcomes in spinal anesthesia for gynecological surgeries.

Keywords: Spinal Anesthesia, Adjuvant, Abdominal Surgeries, Dexmedetomidine.

INTRODUCTION

Spinal anesthesia is a well-known technique of regional anesthesia and is always considered a safe option for general anesthesia when the surgical site is located in the lower extremities, perineum, or lower abdomen.[1] Spinal anesthesia produces intense sensory, motor, and sympathetic blockade with significantly lower concentrations of local anesthetics than other modes of regional anesthesia. Although the operating site is anesthetized and the patient cannot appreciate pain, they remain awake during the whole procedure, which contributes to mental stress ranging from mild to severe, depending on the patient's mentality. Spinal anesthesia has many advantages, such as low cost, reduced risk of aspiration even in patients who are considered to have a full stomach, and reduced blood loss. The main limitation of spinal anesthesia is its short duration. Usually, spinal anesthesia with hyperbaric bupivacaine lasts for 2 to 2.5 hours.[2] To extend the

duration of spinal anesthesia, adjuvants such as opioids, epinephrine, and neostigmine are added to local anesthetics and instilled into the subarachnoid space. These added substances have their advantages and disadvantages. Sedation at an adequate dose during neuraxial block alleviates patient anxiety.[3] When the patient is relaxed, the surgeon finds it easy to operate.[4] Intravenous propofol at a dose of 0.2–0.3 mg/kg was used for sedation. This results in a rapid decline in the level of consciousness. With a continuous infusion of propofol, both cardiovascular and respiratory functions are depressed to a considerable extent. The newer water-soluble benzodiazepine, midazolam, administered at a dose of 0.03 mg/kg, has a rapid onset of action. But recovery is slow. In day-to-day practice, although we use midazolam and propofol to sedate patients, they are vulnerable to causing significant reductions in blood pressure and respiratory function. This effect can be deleterious to patients. Hence, there is a search for an ideal sedative that can be used to relieve anxiety. Dexmedetomidine is a newer drug that is a more specific alpha-2 adrenoreceptor agonist. It causes analgesia, sedation, and sympatholysis. The Food and Drug Administration (FDA) approved the use of dexmedetomidine in 1999 for short-term sedation and analgesia (<24 h) in the intensive care unit.[5] It is becoming very popular because it maintains hemodynamic stability and does not cause significant respiratory depression.

Alpha-2 adrenergic receptor (α-2 AR) agonists have been utilized in various clinical settings due to their actions, which include sedation, analgesia, anxiolysis, perioperative sympatholysis, cardiovascular stabilization, reduced anesthetic requirements, and preservation of respiratory function. Many studies have proven the efficacy of clonidine, a first-generation alpha2 agonist, in prolonging spinal anesthesia when administered intravenously or intrathecally. [2,6] Clonidine is also known to decrease the anesthetic requirements during general anesthesia.[7] Dexmedetomidine being a second-generation alpha2 agonist is more specific for alpha2 receptors. Dexmedetomidine has all the properties of an ideal sedative. It is hypothesized that dexmedetomidine can prolong spinal anesthesia through its actions in the substantia gelatinosa of the spinal cord (spinal action) and locus ceruleus of the brain (supraspinal action). This is the basis of its antinociceptive action.[8] Studies have shown that dexmedetomidine, when administered intravenously or intrathecally, prolongs the duration of spinal anesthesia. [9-11] In this study, we investigated the effect of a single intravenous dose of dexmedetomidine on hyperbaric bupivacaine spinal anesthesia.

MATERIALS AND METHODS

This prospective study was conducted in the Department of Anesthesiology, Kakatiya Medical College, and MGM Hospital, Warangal. Institutional Ethical approval was obtained for the study. Recruitment and data collection were done from August 2022 to January 2024. Written consent was obtained from all the participants of the study after explaining the nature of the study in vernacular language.

Inclusion Criteria

1. ASA grade I-II
2. Age < 60 years
3. Patients who were posted for Total abdominal Hysterectomy and vaginal
4. Hysterectomy under spinal anesthesia.

Exclusion Criteria

1. Patients on sedatives/opioids/antidepressants in the week before surgery.
2. Patients with morbid obesity.
3. Patients with diabetes and renal disease.
4. Pre-operative baseline heart rate equal to or less than 60/min
5. Pre-operative baseline systolic blood pressure equal to or less than 90mmHg.

All patients were examined on the day prior to surgery and pre anesthetic evaluation chart was checked. Special consideration was given to elicit hypertension, breathlessness, pain, cough, wheezing, previous anesthesia, and drug sensitivity. The patient's weight, and height were measured. The nutritional status, airway assessment, and spine examination were also done on the previous day. A detailed examination of all systems was done. Pre-operative routine investigations such as hematocrit, renal function tests, complete blood count, blood grouping, platelet count, chest radiography, and electrocardiography were checked properly.

All patients were informed about the procedure and written consent was taken. All patients were kept nil per oral for 10 hours and were given premedication with tablet alprazolam 0.5 milligram, tablet. Ranitidine 150 milligrams and tablet metoclopramide 10 milligrams on the night before surgery. After putting the patient on an operating table electrocardiography, peripheral saturation of oxygen (SpO2), and non-invasive blood pressure monitor and all the basal parameters were recorded. An IV access with an 18-gauge cannula and all patients were preloaded with Ringer lactate solution 10 ml/kg body weight. Patients were randomly allocated to one of the two groups by slips-in-box technique.

The patient was put in a lateral decubitus position. Lumbar puncture was performed at L3-L4 level with Quincke type 25-gauge spinal needle and injection hyperbaric bupivacaine 17.5 mg was given intrathecally over 30 seconds. If there was technical difficulty at the L3- L4 level, one more try was given at the L2-L3 level with Quincke's needle 25 gauge. If found unsuccessful, those patients were excluded from the study. In group A, patients received hyperbaric intrathecal bupivacaine anesthesia 3.5ml 0.5% (17.5 mg) and intravenous Dexmedetomidine 0.5 µg/Kg in 10 ml normal saline over 10 minutes. In group B, patients received Hyperbaric bupivacaine

anesthesia 3.5 ml 0.5% (17.5mg) and intravenous normal saline 10 ml over 10 minutes. Vitals were recorded [Heart rate, Non-invasive blood pressure monitoring, pulse oximetry, Respiratory rate] every 5 minutes till the end of surgery and then every 5 minutes in the post-anesthesia care unit.

Assessment of Sensory Blockade: Sensory blockade was checked with an alcohol swab in mid axillary line. Sensory blockade was assessed after 5 minutes and there after maximum level of blockade was noted. After this point surgery was started. Vitals were monitored throughout the procedure. At the end of the surgery, sensory level was noted. Two dermatome regression times from the maximal level and regression to level S1 were noted every 20 min postoperatively. The time of spinal injection was taken as 0.

Assessment of Motor Level: Motor level was assessed using the Modified Bromage scale,[12] at the 5th minute and every 20 min after the end of surgery.

Assessment of Sedation: Sedation was assessed by the Ramsay sedation scale,[13] at the 5th minute. Again, sedation was assessed at the end of the surgery. The level of sedation was evaluated every 20 minutes postoperatively for 4 hours.

Statistical analysis: All the available data was uploaded to an MS Excel spreadsheet and analyzed by SPSS version 25 in Windows format. All the continuous variables were represented as frequency, mean, and standard deviations. The categorical variables were calculated by the Students T-test for comparison of the means of two groups and the chi-square test was applied to calculate the differences between the two groups. The values of p (<0.05) were considered significant.

RESULTS

The baseline characteristics of the two groups of cases in the study are depicted in [Table 1]. A critical analysis of the table shows that both groups were well-matched for age, weight, height, and BMI. The mean age was 48.67 ± 4.29 years in Group A and 48.00 ± 4.41 years in Group B and the p values were (p = 0.555). Weight and height also showed no statistically significant differences, with p-values of (0.244) and (0.337), respectively. The BMI was slightly lower in the Dexmedetomidine group (23.10 ± 0.98) compared to the control group (23.66 ± 1.52) although was not statistically significant (p = 0.091). This indicates that the groups were comparable at baseline reducing confounding in outcome analysis.

Table 1: Baseline Characteristics

Characteristic	Dexmedetomidine Group A (n=30)	Normal Saline Group B (n=30)	p-value
Age (years)	48.67 ± 4.29	48.00 ± 4.41	0.555
Weight (kg)	53.27 ± 2.12	54.00 ± 2.68	0.244
Height (cm)	151.87 ± 2.43	151.13 ± 3.36	0.337
BMI (kg/m2.)	23.10 ± 0.98	23.66 ± 1.52	0.091

The hemodynamic characteristics of two groups of cases recorded at various intervals during the surgery were compared and given in [Table 2]. The analysis of the table shows that preoperatively, both groups had comparable heart rates. From the interval of 20 minutes onward, the Dexmedetomidine group showed significantly lower heart rates at 20, 60, 120, and 240 minutes (p < 0.005) because of the sympatholytic effect. The mean arterial pressure values were found to be significantly lower in group A at 60 and 80 minutes (p = 0.007 and <0.001, respectively) which shows the hemodynamic stability of the dexmedetomidine group. However, MAP differences at 180 minutes were not statistically significant (p = 0.39), the consistent trend supports the cardiovascular-modulating effects of dexmedetomidine.

Table 2: Hemodynamic Parameters Over Time

Parameter	Time Point	Dexmedetomidine Group A	Normal Saline Group B	p-value
Heart Rate (bpm)	Pre-op	79.86 ± 11.19	80.3.3 ± 1.666	0.94
	20 min	62.93 ± 10.53	73.53 ± 11.60	<0.001*
	60 min	59.86 ± 6.53	71.53 ± 6.21	<0.001*
	120 min	59.66 ± 8.24	70.40 ± 9.86	<0.001*
	240 min	76.66 ± 10.28	84.00 ± 8.61	0.004*
MAP (mmHg)	Pre-op	97.33 ± 6.99	100.51 ± 6.89	0.82
	60 min	75.00 ± 10.46	85.86 ± 4.68	0.007*
	80 min	78.91 ± 7.13	87.44 ± 8.74	<0.001*
	180 min	76.26 ± 7.10	89.71 ± 5.83	0.39

*Significant

[Table 3] shows the block characteristics and duration recorded in the cases at various intervals. Analysis of the table shows that the onset of both motor and sensory blocks (Bromage 3 and T4) was achieved by 100% of patients in both groups at 5 minutes. However, the duration of both motor and sensory blocks was significantly longer in the Dexmedetomidine group (243.60 ± 17.0 min and 255.20 ± 8.6 min and p <0.001) than in the control group (211.20 ± 16.7 min and 210.80 ± 33.1 min and p <0.001). Regression of block by two dermatomes was prolonged in the Dexmedetomidine group

(125.20 ± 17.5 min vs. 94.60 ± 18.9 min, and p < 0.001). It was found that at the interval of 160 minutes, 86.7% of Group A patients still exhibited Bromage 3 motor block, while only 6.7% did in Group B.

Table 3: Block Characteristics and Duration (Time in minutes; Mean ± SD)

Parameter	Dexmedetomidine Group A	Normal Saline Group B	p-value
Motor Block Onset (Bromage 3 at 5 min)	100%	100%	1.000
Sensory Block Onset (T4 at 5 min)	100%	100%	1.000
Motor Block Duration	243.60 ± 17.0	211.20 ± 16.7	<0.001*
Sensory Block Duration	255.20 ± 8.6	210.80 ± 33.1	<0.001*
Two-Dermatome Regression	125.20 ± 17.5	94.60 ± 18.9	<0.001*
Motor Block at 160 min (Bromage 3)	86.70%	6.70%	<0.001*

*Significant

[Table 4] shows the comparison of adverse events recorded in the cases and interventions done for the treatment. The analysis of the table shows that the incidence of bradycardia was more in the dexmedetomidine group consequently atropine was needed more frequently in the dexmedetomidine group, especially at 20 and 60 minutes (p = 0.04). This shows that expected bradycardia is due to α2 agonism. Sedation was significantly deeper in the dexmedetomidine group at 140 minutes (p < 0.001), with 73.3% of patients being in Ramsay Grade R3 (responsive to commands) and 26.7% in R2 (cooperative). However, 26.7% of the control group exhibited any sedation. These results confirm dexmedetomidine's sedative effect, which is advantageous intraoperatively without respiratory depression.

Table 4: Adverse Events and Interventions

Parameter	Dexmedetomidine Group A	Normal Saline Group B	p-value
Atropine Requirements			
20 min	6 (20.0%)	2 (6.7%)	0.04*
40 min	4 (13.3%)	2 (6.7%)	0.18
60 min	6 (20.0%)	2 (6.7%)	0.04*
Total Bradycardia Events Sedation at 140 min (Ramsay Scale)	20 (66.7%)	8 (26.7%)	<0.001*
R2 (Cooperative)	26.70%	6.70%	<0.001*
R3 (Responsive to commands)	73.30%	0.0%	<0.001*

*Significant

The level of sensory block at the interval of 160 minutes is given in [Table 5]. The analysis of the table shows that at 160-minute intervals, sensory block level was notably higher in the dexmedetomidine group. About 46.7% maintained sensory blockade at T6 and 53.3% at T8, while none regressed to T10 or lower. Conversely, in the control group, 33.3% had regressed to T10 and 60% to T12, with only 6.7% maintaining at T8. This shows a clear and statistically significant difference in sensory block regression, with 100% of the dexmedetomidine group maintaining a block level at or above T8, compared to 93.3% of the control group showing regression to T10 or below.

Table 5: Sensory Block Level at 160 Minutes

Sensory Level	Dexmedetomidine Group A	Normal Saline Group B
T6	46.70%	0%
T8	53.30%	6.70%
TI0	0.0%	33.30%
T12	0.0%	60%
Findings	100% maintained ≥ T8 level	93.3% regressed ≤ TI0 level

DISCUSSION

This prospective randomized controlled study was designed to evaluate the efficacy of intravenous dexmedetomidine in prolonging the effects of spinal anesthesia. The use of adjuvants is common for the enhancement of the duration of spinal anesthesia through intrathecal routes. One of the commonly used adjuvants is dexmedetomidine, it is an alpha-2 adrenergic agonist and has proven benefits such as analgesia, sedation, and hemodynamic stability with minimal respiratory depression.[14] The initial evaluation of the distribution of two groups of cases based on age, weight, height, and BMI, with no statistically significant differences [Table 1]. This shows that any observed differences in the outcomes were not due to demographic confounding factors and strengthened the analysis of the study. The results of the study showed that there were significant hemodynamic changes observed in the dexmedetomidine group. This is evident with reduced heart rate and mean arterial pressure at

multiple intervals as depicted in [Table 2]. This is because dexmedetomidine has significant sympatholytic action. [15,16] However, bradycardia is a common occurrence in such cases because of α2 agonism requiring atropine. This was shown in this study as more frequent use of atropine was there in Group A, it responded promptly to intervention and was not associated with adverse outcomes [Table 4]. The duration of sensory and motor block was significantly increased with dexmedetomidine administration (255.2 8.6 min vs. 210.8 33.1 min and 243.6 17.0 min vs. 211.2 16.7 min, respectively; [Table 3]. These results are consistent with those of Al-Mustafa et al. and Dinesh et al., who showed similar block extension using IV dexmedetomidine.[18,19] Moreover, two-dermatomal regression time was also prolonged in the study group, adding more weight to the role of dexmedetomidine in long-lasting spinal anesthesia. After 160 min, 86.7% of the patients in Group A still had Bromage Grade 3 motor block, but only 6.7% of the patients in Group B had it; 83.3% of the patients in Group B were sensory-regressed to T10 or below, whereas only 1.7% of the patients in Group A were sensory-regressed to T10 or below [Table 5]. These findings substantiate the role of the drug in extending clinically relevant analgesia.

As far as sedation was concerned the results of this study showed that 73.3% of patients in Group A achieved a Ramsay sedation score of R3, indicating calm yet arousable sedation, significantly higher than in the control group [Table 4]. This sedation profile was beneficial intraoperatively. These results are in concordance with previous studies that endorse dexmedetomidine over midazolam due to fewer paradoxical reactions and better sedation quality.[20,21] Our study did not find cases of transient hypertension. It could be because of a moderate IV dose (0.5 µg/kg) administered slowly over 10 minutes. Studies support this as a safe and effective dose range for minor to moderate procedures. [22,23] The drug's short half-life (2–3 hours) compared to clonidine (6–10 hours) provides better control of sedation and side effects (10). Finally, this study reinforces that intravenous dexmedetomidine at 0.5 µg/kg enhances spinal anesthesia by significantly prolonging sensory and motor block, providing sedation without respiratory depression, and maintaining hemodynamic stability. These attributes support its utility as a valuable adjuvant in regional anesthesia.

CONCLUSION

In conclusion, this study found that intravenous dexmedetomidine at a dose of 0.5 µg/kg significantly enhanced the duration of spinal anesthesia by prolonging sensory and motor blocks. It also improves intraoperative sedation and maintains hemodynamic stability with minimal adverse effects. This drug demonstrated significant clinical analgesic and sedative duration extension, without respiratory depression. Therefore, it is a valuable adjuvant for regional anesthesia with a good safety profile. Overall, this study supports the role of dexmedetomidine as an adjuvant for abdominal surgeries, offering improved patient comfort and extended postoperative analgesia.

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