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Original Article

Effects of intravenous dexmedetomidine in patients receiving 0.5% hyperbaric bupivacaine spinal anaesthesia

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Abstract

Background & Methods: The aim of the study is to study the effects of intravenous dexmedetomidine in patients receiving 0.5% hyperbaric bupivacaine spinal anaesthesia. Procedure of spinal anaesthesia and the use of VAS scale were explained and informed written consent was obtained. Patient baseline parameters were recorded and preloading was done with Inj.

Results: The onset of motor block was 2.27±2.69 and 2.17±1.81 minutes in Group I and Group II respectively. Duration of motor blockade (181.48±1.34 mins vs. 168.18±3.27 mins) was more in the dexmedetomidine group than the normal saline group. The mean time for the duration of analgesia in group I was 201.72 minutes and in group II 159.20 minutes.

Conclusion: Dexmedetomidine intravenously or intrathecally showed to be a safe supplement to bupivacaine spinal anesthesia. When comparing intravenous dexmedetomidine to spinal bupivacaine, intrathecal dexmedetomidine was a better adjuvant. It offered less overall side effects, improved perioperative analgesia, increased augmentation to sensory and motor block, and more stable hemodynamics.

Keywords: intravenous, dexmedetomidine, hyperbaric, bupivacaine, spinal & anaesthesia.

Study Design: Observational Study.

Introduction

Spinal anesthesia is a commonly used technique in anesthetic practice for gynecological, lower abdominal, pelvic, and lower limb surgeries. Different adjuvants have been used to prolong spinal anesthesia, with the probable benefits of late commencement of postoperative pain and reduced analgesic requirements[1]. Adjuvants like alpha-2 adrenoceptor agonists like dexmedetomidine and clonidine have been recently used for their sedative, analgesic, and perioperative sympatholytic and cardiovascular stabilizing effects with reduced anesthetic requirements[2].

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Anesthesia can be administered locally, regionally (spinal or epidural), or generally for lower limb and abdominal procedures; nonetheless, neuraxial blocking is the recommended kind of anesthesia. Spinal block is still the favored technique due to its early onset, excellent blockage, low failure rates, reduced risk of infection from catheter in situ, and cost-effectiveness. Nevertheless, it has certain drawbacks, including no postoperative analgesia and a shorter block length. Spinal anesthetic also has the benefit of preserving spontaneous breathing and relaxing the muscles required for operation. Additionally, it has the benefit of not carrying the hazards of pulmonary aspiration or intubation[3].

Dexmedetomidine is a highly selective α2-agonist. α2-receptors are found in many sites throughout the body includin central nervous system, spinal cord and peripheral tissues[4].There is a hypothesis that by its actions on the substantia gelatinosa in the spinal cord and locus coeruleus in the brain, Dexmedetomidine can prolong spinal anaesthesia when given IV or intrathecally. Dexmedetomidine leads to sedation without respiratory depression2 and causes decrease in stress response by lowering secretion of catecholamines. This can be of great value in perioperative period during which haemodynamic variations occur due to stress.3with the above background, the present study was aimed to evaluate the effects of IV dexmedetomidine on spinal anaesthesia with 0.5% of hyperbaric bupivacaine[5-6].

Material and Methods

A prospective study of patients undergoing elective spinal surgeries was taken after institutional approval of sample size 50 for 06 months. We included patients of 18-65years of age, both sexes, ASA-I/II for the study and randomly divided into two groups of 25 each group. Pregnant women, patients having deformity of spine or local infection, poorly controlled hypertension, hypovolemic shock, coagulation abnormality, severe liver and renal disease, pre-existing severe bradycardia, congestive heart failure were excluded. After pre anaesthestic checkup, patients were kept fasting from previous night.

Group I: 25 patients received hyperbaric bupivacaine intrathecally with 5 µg in 0.5 ml dexmedetomidine, followed by infusion of intravenous isotonic saline (10 ml) over 10 minutes and maintenance volume equivalent to the other patients groups throughout the period of surgery.

Group II: 25 patients received mg in 3 ml hyperbaric bupivacaine intrathecally with normal saline 0.5 ml, followed by intravenous bolus dose of dexmedetomidine 0.5 µg/kg diluted in 10 ml normal saline over 5 minutes, followed by intravenous infusion of maintenance volume of normal saline equivalent to other groups for the whole period of surgery.

Result

Table No. 1: Demographic Profile

	Group I	Group II
Age (Yrs)	49.71±14.196	44.19±12.17
Weight (kg)	62.42±7.336	64.12±11.45
Height (cm)	161±8.11	166.38±8.73
Male/Female	22/3	21/4

The age, weight, height, and gender of patients were comparable in the groups.

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Table No. 2: Comparison of duration (mins)

S. No.	Parameter	Group I	Group II
1	Onset of analgesia	2.27 ± 2.69	2.17 ± 1.81
2	Motor blockade	181.48 ± 1.34	168.18 ± 3.27
3	Duration of analgesia	201.72 ± 29.93	159.20 ± 3.46

The onset of motor block was 2.27±2.69 and 2.17±1.81 minutes in Group I and Group II respectively. Duration of motor blockade (181.48±1.34 mins vs. 168.18±3.27 mins) was more in the dexmedetomidine group than the normal saline group. The mean time for the duration of analgesia in group I was 201.72 minutes and in group II 159.20 minutes.

Table No. 3: Baseline hemodynamic parameters

S. No.	Parameter	Group I	Group II	P Value
1	HR(bpm)	81.6±4.13	79.34±5.49	0.294
2	SBP(mmHg)	123.60±6.15	121.47±6.40	
3	DBP(mmHg)	77.78±8.31	75.93±3.66	
4	MAP(mmHg)	93.64±4.96	91.98±2.07	

The patients in both the groups were comparable in terms of baseline heart rate, systolic and diastolic blood pressure and mean arterial pressure (P> 0.05) not significant.

Table No. 4: ASA score

S. No.	ASA score	Group I	Group II
1	ASA I	(09) 35%	(08) 32%
2	ASA II	(16) 65%	(17) 68%

The ASA I 35% and 32% whereas ASA II 65% & 68% in Group I and Group II respectively.

Discussion

Spinal anaesthesia is a widely used regional anaesthetic technique for lower abdominal surgeries. Spinal anaesthesia produces intense sensory, motor and sympathetic blockade with significantly lesser dosage of local anaesthetics[7]. Adjuvants like buprenorphine, tramadol, fentanyl, $\alpha 2$ agonists can be used to overcome limited duration of spinal anaesthesia. $\alpha 2$ agonists produces sedation, analgesia, anxiolysis, perioperative sympatholysis, cardiovascular stabilizing effects, reduced anaesthetic requirements and preservation of respiratory function. These $\alpha 2$ receptors are found in many sites in CNS, spinal cord and peripheral tissues. $\alpha 2$ receptors are present in highest densities in the locus coeruleus in the brain. Dexmedetomidine

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is a highly selective $\alpha 2$ agonist.1, 2, 3 the hypnotic, sedative effects and anti-nociceptive action of dexmedetomidine is due to the action on the locus coeruleus, it prolongs the duration of spinal anaesthesia due to the action on substantia gelatinosa[8]. Dexmedetomidine causes a decrease in HR and BP by lowering secretion of catecholamines which is beneficial in the perioperative period during which haemodynamic variationoccurs due to stress.

Recent studies have shown the efficacy of both intrathecal and IV dexmedetomidine in prolonging spinal anesthesia[9]. Prolongation of spinal anesthesia after IV dexmedetomidine is by its supra-spinal action at locus ceruleus and dorsal raphe nucleus. There are three subtypes of α2 receptors: A, B, and C. Dexmedetomidine is a more selective α2-A receptor agonist than clonidine, with more sedative and analgesic effects[10]. Activation of presynaptic α2-A receptors at locus ceruleus decreases norepinephrine release and causes sedative and hypnotic effects, whereas its effect on descending medullo spinal noradrenergic path way results in analgesia by terminating pain signal propagation. At substantia gelatinosa of the spinal cord, it decreases fi ring in nociceptive neurons and release of substance P, thus producing analgesia. So, dexmedetomidine has a role in modulating pain and inhibiting the transmission and perception of pain. Activation of post-synaptic α2-A receptors in CNS results in hypotension and bradycardia by decreasing the sympathetic activity[11-14]. Activation of post-synaptic α2-C receptors in CNS results in anxiolysis, whereas activation of post-synaptic α2-B receptors in peripheral vasculature results in transient hypertension.

Conclusion

Dexmedetomidine intravenously or intrathecally showed to be a safe supplement to bupivacaine spinal anesthesia. When comparing intravenous dexmedetomidine to spinal bupivacaine, intrathecal dexmedetomidine was a better adjuvant. It offered less overall side effects, improved perioperative analgesia, increased augmentation to sensory and motor block, and more stable hemodynamics.

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