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COMPARISON OF INTRATHECAL DEXMEDETOMIDINE AND INTRATHECAL BUPRENORPHINE AS AN ADJUVANT TO BUPIVACAINE FOR SPINAL ANAESTHESIA IN LOWER ABDOMINAL SURGERIES

Anaesthesiology

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ABSTRACT

BACKGROUND: Various adjuvants have been used with local anesthetics in spinal anesthesia to avoid intraoperative visceral and somatic pain and to provide prolonged postoperative analgesia. Dexmedetomidine, the new highly selective α2-agonist drug, is now being used as a neuraxial adjuvant. Buprenorphine is a centrally acting lipid soluble analogue of alkaloid thebaine. It exhibits analgesic property both at spinal and supraspinal levels.6The aim of the study was to compare onset of sensory and motor block, duration of complete analgesia, time for two segmental regression of sensory block, time for complete recovery of motor block, hemodynamic effects and side effects due to intrathecal buprenorphine, dexmedetomidine with hyperbaric 0.5% bupivacaine.

MATERIALS & METHOD: 90 ASA I and II patients of either sex, scheduled for elective lower abdominal surgeries under spinal anesthesia were randomly allocated to receive either 3ml of 0.5% hyperbaric bupivacaine with 0.5ml Normal saline (group A, n=30) or 3ml of 0.5% hyperbaric bupivacaine with 60µg Buprenorphine (group B, n=30) or 3ml of 0.5% hyperbaric bupivacaine with 5µg of Dexmedetomidine (group D, n=30) intrathecal.

RESULTS: Patients in dexmedetomidine group (D) had a significantly longer duration of complete analgesia, motor block and time for two segment regression. The onset of motor blockade is faster with patients in Buprenorphine group (B). Nausea and vomiting (20%) was observed significantly more in group B (p>0.05).

CONCLUSION: Use of Intrathecal Dexmedetomidine is an excellent additive to Bupivacaine for quality of anesthesia and prolonged duration of analgesia without any deleterious effects

KEYWORDS

Dexmedetomidine, Bupivacaine, Spinal Anesthesia

INTRODUCTION:

Spinal anaesthesia is one of the most suitable modalities of anaesthesia for lower abdominal surgeries. Supplementation of local anaesthetics with adjuvants is to reduce the dose of local anaesthetic, minimize side effects and prolong the duration of anaesthesia.[1,2] Dexmedetomidine is a specific α-2 adrenergic agonist.[3] It has been extensively used as premedicant, for sedation in the Intensive Care Unit (ICU) and for awake fibreoptic intubation.[3,4]It was first used intrathecally in humans for transurethral resection of prostate.[5] It prolongs both sensory and motor block and has nociceptive action for both visceral and somatic pain..

Buprenorphine is a centrally acting lipid soluble analogue of alkaloid thebaine. It exhibits analgesic property both at spinal and supraspinal levels.[6]It has consistently proven to prolong the duration of anaesthesia.[1,2,7] At higher doses, it causes pruritus, drowsiness, nausea and vomiting.[8]

MATERIALS & METHOD:

The study was conducted after approval of ethical committee of the institution. Written informed consent was obtained from all patients. Inclusion criteria were American Society of Anesthesiologists (ASA) physical status I or II, either sex, age 18–60 years, presenting for lower abdominal surgeries. Exclusion criteria were patient allergic to drug, heart block/dysrhythmia, or on therapy with adrenergic receptor antagonist, calcium channel blocker, and/or ACE inhibitor.

All patients were preloaded with Lactated Ringer's solution 10 mL/kg and monitored with noninvasive blood pressure, pulse oximetry, and electrocardiogram. 25G Quincke type spinal needles were introduced through L3–L4 interspaces in left lateral position using aseptic precautions. Patients were randomly divided into the following groups: Group D—to receive 3mL volume of 0.5% hyperbaric bupivacaine and 5 μg dexmedetomidine intrathecal, Group B—to receive 3mL volume of 0.5% hyperbaric bupivacaine with 60µg Buprenorphine and Group A- to receive 3mL volume of 0.5% hyperbaric bupivacaine with 0.5% Normal saline intrathecal. Intrathecal injection was given at a rate of 1 ml for 3-4 sec. Immediately after completion of the injection patients were made to lie supine.

Hypotension, defined as a decrease of systolic blood pressure by more than 30% from baseline or a fall below 90 mmHg, was treated with IV doses of Mephentermine 3-6mg and IV fluid as required. Bradycardia, defined as heart rate < 50 bpm, was treated with IV atropine 0.3–0.6 mg. The incidence of adverse effects, such as nausea, vomiting, shivering, pruritus, respiratory depression, sedation, and hypotension were recorded. Sensory testing was assessed by loss of pinprick sensation to 23G hypodermic needle. The time of onset was taken from the time of injection of drug into subarachnoid space to loss of pin prick sensation at T8. The highest level of sensory block and time was noted. The time for 2 dermatomal segment regression of sensory level was noted. Intensity of motor blockade was assessed by modified bromage scale. Observations were made at 5,10,20,30,45,60,90 and 120 min after injection of the drug.

STATISTICAL ANALYSIS:

Descriptive statistics of sensory block, motor block, time to rescue analgesia would be analyzed and expressed in terms of mean and standard deviation, number, and frequencies. Parametric data are analyzed using an analysis of variance test among groups followed by post-hoc test if needed. Complications were expressed in frequencies and analyzed through chi-square test. P-value less than 0.05 is considered significant.

RESULTS:

The groups were comparable with respect to age, gender, height, and weight (Table 1).

Table 1: Demography

	Group D	Group B	Group A	P value
Age in yrs (Mean ± SD)	36.26±11.09	39.73±13.32	34.73±13.09	0.291 (NS)
Height in Cms (Mean ± SD)	155.90±2.95	156.03±2.68	157.00±3.34	0.307(NS)
Weight (Mean ± SD)	57.30±3.27	58.30±4.08	59.20±4.24	0.173 (NS)
Male	22 (73.33%)	25 (83.33%)	27 (90%)	0.236(Not Significant)
Female	8 (26.67%)	5 (16.67%)	3 (10%)	

The characteristics of sensory block are summarized in Table 2

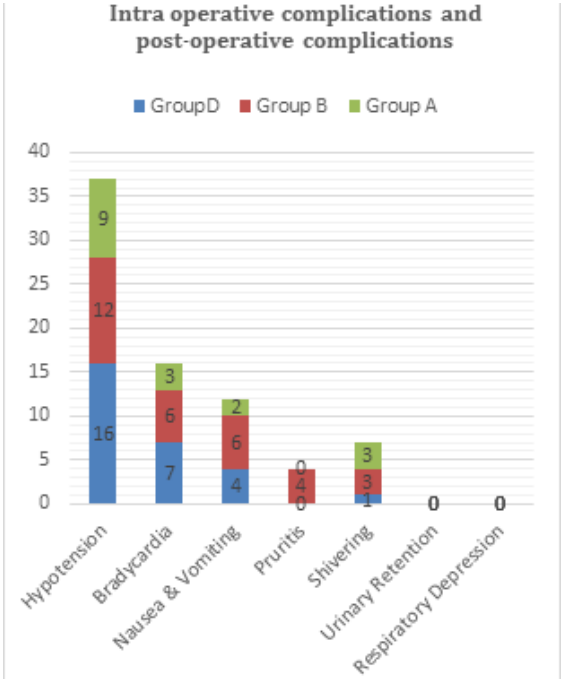
	Group D	Group B	Group A	P value
Time of onset of sensory block in Min	3.10 ±0.54	3.06 ± 0.73	3.20 ±0.61	0.424(NS)
Time for Peak sensory block in Min	9.40 ± 1.22	9.66 ± 1.60	10.00 ±1.36	0.379(NS)
Time for 2 segment regression in Min	150.40± 11.38	120.70 ± 9.78	90.46 ± 6.92	0.001(Significant)
Duration of complete analgesia in Min	300.53± 11.06	220.43 ± 13.71	180.56 ± 11.30	0.001(Significant)

The characteristics of motor block are summarized in Table 3

	Group D	Group B	Group A	P value
Time of onset of motor block	4.10 ±0.75	4.03 ± 0.85	4.46 ± 0.62	0.029 (Significant)
Duration of motor block	270.20± 24.37	190.13 ± 15.80	150.80 ± 6.06	0.001 (Significant)

There was no significant difference between three groups with respect to hemodynamic parameters (Heart rate, Systolic blood pressure, Diastolic blood pressure, Mean Arterial Pressure, SpO2) at any point of time. (p>0.05).

Among Intra operative complications and post-operative complications**,** Nausea and vomiting (20%) was observed significantly more in group B patients. (p>0.05). The intraoperative and postoperative complications were summarized in graph 1.



DISCUSSION:

Alleviation of pain has been a challenging task to all pain therapists and anaesthesiologists.[9]The intrathecal route has advantages of greater technical ease and a single injection producing pain relief of sufficient duration is always beneficial.[7]Various adjuvants have been added to local anaesthetics to enhance sensory and motor block along with postoperative analgesia.[10]The mechanism by which in trathecal α2 adrenoreceptor agonists prolong the motor and sensory block of local anaesthetics is not well understood. The prolongation of effects might result from synergism between the local anaesthetic and α2 adrenoreceptor agonists.[11] Dexmedetomidine, an imidazole compound, is the pharmacologically active dextroisomer of medetomidine that displays specific and selective. Intrathecal dexmedetomidine combined with spinal bupivacaine prolongs the sensory block through suppression of C-fibre transmitter release and hyperpolarization of post synaptic dorsal horn neurons while prolongation of motor block of spinal anaesthetics might result from the binding of α2 adrenoreceptoragonists to motor neurons.[12]

Opioids administered in subarachnoid space appear to act principally on μ receptor in substantia gelatinosa of dorsal horn of spinal cord by suppressing excitatory neuropeptide release from C- fibres.[13] The combination of local anaesthetics and opioids, allow reduction in both doses of drugs, thus lessening the side effects attributable to each. Buprenorphine is a mixed agonist – antagonist type of opioid with a long duration of action. The high lipid solubility; high affinity for opioid receptors and prolonged duration of action makes buprenorphine a suitable choice for intrathecal site administration.[14,15]

In the present study, duration of complete analgesia, time for two segmental regression of sensory block, time for complete recovery of motor blockade were significantly prolonged in group D when compared with other groups B and A.

On intergroup comparison between A and D groups, dexmedetomidine group has prolonged duration of analgesia compared to control group and it is statistically significant. Intrathecal α2 adrenoreceptor agonists have antinociceptive actions for both somatic and visceral pain. Intrathecal dexmedetomidine when combined with spinal bupivacaine prolongs the sensory block by depressing the release of C-fibre transmitter and by hyperpolarisation of post synaptic dorsal horn neurons.[16]

In the present study, mean duration of motor block in group D was significantly prolonged when compared with groups B and A. Prolongation of motor block by dexmedetomidine might be caused by direct impairment of excitatory amino acid release from spinal interneurons.[17]The higher motor blockade in dexmedetomidine, may be because of the tendency of α2 adrenoreceptor agonists to bind with motor neurons in the dorsal horn of the spinal cord.[16]

Incidence of hypotension and bradycardia was higher, and incidence of shivering lower in dexmedetomidine group. Puritus was more in buprenorphine group compared with other groups yet the difference between three groups was not significant statistically. Incidence of Nausea and vomiting was significantly more in buprenorphine group.

CONCLUSION:

Use of Intrathecal Dexmedetomidine is an excellent additive to Bupivacaine for quality of anesthesia and prolonged duration of analgesia without any deleterious effects.

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