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A COMPARATIVE STUDY OF DIFFERENT DOSES OF DEXMEDETOMIDINE IN SPINAL ANAESTHESIA IN LOWER LIMB ORTHOPEDIC PROCEDURES

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Abstract:

Introduction: Unless contraindicated, spinal anaesthesia is the preferred mode of anaesthesia in patients undergoing surgeries of lower limbs. Used alone in spinal anaesthesia, hyperbaric bupivacaine 0.5% is associated with relatively short duration of action leading to the need to rescue with general anaesthesia if the surgical procedure exceeds beyond the drug's duration of action. Over the years many drugs have been used as an additive to spinal anaesthesia in order to hasten its onset of action, decrease the time to surgical incision, prolong the duration of action and to provide adequate postoperative analgesia.

Materials and Methods: This prospective randomized double blind study was conducted with 180 consenting patients of ASA grade I and II, scheduled for lower limb orthopaedic surgeries. Using the sealed envelope method, the patients were randomly allotted into 3 groups, 60 patients in each group. Group BS, Group BD1, Group BD2. The surgeon, patient and the observing anaesthesiologist were blinded to the patient group. All patients received drug volume of 3 ml containing 2.5 ml (12.5 mg) hyperbaric bupivacaine hydrochloride. The study groups received dexmedetomidine 5 μg (group BD1) or 10 μg (BD2) diluted to 0.5ml with 0.9% saline, added to bupivacaine in the same syringe. The control group BS received an identical volume of 0.9% saline added to bupivacaine. Standard monitoring with non-invasive BP, HR and ECG were started.

Results: 180 patients were enrolled into the study. The groups were comparable with respect to age, weight, height, effect distribution and operative time. Sedation score and hemodynamic data did not differ significantly among the groups. The duration of sensory block was significantly prolonged in the group receiving intrathecal dexmedetomidine as adjuvant as compare to the group receiving local anaesthetic alone. The mean sensory block duration in BD1 group and BD2 was significantly prolonged.

Conclusion: Supplementation of spinal bupivacaine with dexmedetomidine significantly prolonged both sensory and motor block compared with intrathecal bupivacaine alone. Patients in the groups that received dexmedetomidine had reduced post-operative pain scores and a longer analgesic duration than those who received spinal bupivacaine alone.

Key Words: spinal anaesthesia, hyperbaric bupivacaine, postoperative analgesia.

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INTRODUCTION

Unless otherwise required, spinal anaesthesia is the preferred form of anaesthetic in patients undergoing lower limb surgery. When used alone in spinal anaesthesia, hyperbaric bupivacaine 0.5% has a relatively short duration of effect, necessitating rescue with general anaesthesia if the surgical procedure lasts longer than the drug's duration of action.[1] Many medicines have been utilised as additives to spinal anaesthesia over the years in order to speed up the onset of effect, reduce the time to surgical incision, extend the duration of action, and provide acceptable postoperative analgesia.[2] These medicines include midazolam, ketamine, fentanyl, clonidine, as well as a variety of opioids and nonopioids. Use of opioids is associated with its side effects like pruritis, nausea, vomiting, constipation, and respiratory depression which can be distressing for the patient.[3]

Dexmedetomidine, a highly selective α2 agonist, is quickly becoming the preferred additive to spinal anaesthesia due to its ability to give analgesia and awake sedation without respiratory depression while maintaining stable haemodynamics.[4] Various studies conducted by different authors used dexmedetomidine at dosages of 3 μg, 5 μg, 10 μg, and 15 μg, and there may be a dose-related lengthening of motor blockade as well as an increase in the occurrence of dexmedetomidine side effects, particularly hypotension and bradycardia.[5] As a result, there appears to be no clear consensus on the dose of dexmedetomidine to be used as an addition to hyperbaric bupivacaine in daily practise spinal anaesthesia. Avoidance of side effects of dexmedetomidine while ensuring a pain free peri operative period is vital for successful outcome of any surgical procedure.[6]

The study aims to determine the effect of adding dexmedetomidine to hyperbaric bupivacaine and comparing with bupivacaine alone for neuraxial anaesthesia and post-operative analgesia.

MATERIALS AND METHODS

Study design: A prospective randomized double blind study

Study location: Department of Anaesthesia, Govt General and Chest Hospital, Erragadda, Hyderabad.
Study Duration: January 2021 to December 2022 (two year).

Sample Size: 180 patients

This prospective randomized double blind study was conducted with 180 consenting patients of ASA grade I and II, scheduled for lower limb orthopaedic surgeries. Using the sealed envelope method, the patients were randomly allotted into 3 groups, 60 patients in each group. Group BS, Group BD1, Group BD2. The surgeon, patient and the observing anaesthesiologist were blinded to the patient group.

Exclusion criteria:

- Any contra-indication to spinal anaesthesia like hypotension, coagulation defects, spine abnormalities etc.
- Body weight ≥120 kg and height ≤150 cm.
- Patients with labile hypertension, heart block, arrhythmias.
- Patients on calcium channel blockers, adrenergic receptor blockers, ACE inhibitors.
- Allergic to the drug.

All patients received drug volume of 3 ml containing 2.5 ml (12.5 mg) hyperbaric bupivacaine hydrochloride. The study groups received dexmedetomidine 5 μg (group BD1) or 10 μg (BD2) diluted to 0.5ml with 0.9% saline, added

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to bupivacaine in the same syringe. The control group BS received an identical volume of 0.9% saline added to bupivacaine. Standard monitoring with non-invasive BP, HR and ECG were started. The patients were preloaded with RL solution 15ml/kg. Patients were placed in sitting position and lumbar puncture was performed at L3-4 interspace through a midline approach using a 25 gauge quincke needle under strict asepsis. Heart rate, MABP and oxygen saturation were monitored and recorded after the block every 5 minutes for half an hour then every 15 minutes until the end of surgery. The level of sensory block was assessed (By a blinded anaesthetist not involved in this study) by pin prick sensation using a blunt 25 gauge needle along the mid clavicular line bilaterally. The times from intrathecal injection to two dermatome sensory regression, sensory regression to S1 dermatome, and motor block regression to Bromage 1 were recorded. The motor level will be assessed according to modified bromage scale: Bromage;

0 – able to move hip, knee and ankle.

1 – unable to move the hip

2 – unable to move the hip and knee

3 – unable to move the hip, knee and ankle.

The hemodynamic variables were recorded before spinal anaesthesia and there after every 5 minutes for half an hour, then every 15 minutes until the end of procedure and PACU. A decrease of ≥ 20% from base line or less than 90 mmHg in systolic blood pressure, was defined as hypotension was treated with 5 mg intravenous ephedrine and a bolus administration of 500 ml of RL solution over 20 minutes. Bradycardia was defined as HR ≤ 50 beats/ minute and was treated with 0.6mg atropine.

Statistical Analysis: Student t test and ANOVA test for parametric data. Chi square test for non-parametric data.

RESULTS

180 patients were enrolled into the study. The groups were comparable with respect to age, weight, height, effect distribution and operative time. Sedation score and hemodynamic data did not differ significantly among the groups.

The duration of sensory block was significantly prolonged in the group receiving intrathecal dexmedetomidine as adjuvant as compare to the group receiving local anaesthetic alone. The mean sensory block duration in BD1 group and BD2 was significantly prolonged.

Respiratory distress and pruritus was not reported in any of the patient among all the groups, had peripheral oxygen saturation ≥95% and did not required oxygen administration in PACU.

Demographic data	BS	BD1	BD2
Age	38.7 ± 12.90	37.2 ± 10.4	38.2±11.5
Male	32	30	32
Female	28	30	28
ASA I	50	48	48
ASA II	10	12	12
Height	156±6	160±5	158±6
Weight	65±8	65±6	64±7

Table 1: Demographic characters

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Variable (min)	BS	BD1	BD2	P-Value
Time of onset of sensory block	7.6±1.7	8.2±2.6	8.3±2.3	0.124
Time of onset of motor block	9.3±2.8	9.8±3.7	9.6±3.1	0.086
Time to reach maximum sensory level	10.2±3.6	9.6±3.0	10.3±3.3	3.30
Duration of sensory block	102.3±15.3	116.2±20.3	146.6±20.5	0.0001
Duration of motor block	160.4±18.3	196.4±25.4	270.4±24.3	0.0001
Duration of spinal anaesthesia	183.0±32.0	242.4±54.7	294.5±44.3	0.001

Table 2: Time of onset of sensory block, motor block, Duration of spinal anaesthesia

VAS < 3 was observed in all the groups intra operatively and there was no need for additional analgesic throughout the surgery. Post-operative VAS and total analgesic requirement in 24 hours were minimal in group BD2 as compare to BD1 group.

All the patients achieved modified Bromage scale 3 motor block and there was dose dependent prolongation of motor block in BD1 and BD2 groups. Similarly regression of motor block to modified Bromage 0 was significantly prolonged in group BD2 than BD1 and BS group. Complete recovery of sensory and motor functions was observed in all the patients.

At first post-operative visit, 15 days after surgery the patients were evaluated and none of them had neurological deficit.

DISCUSSION

Dexmedetomidine is α2 adrenoreceptor agonist. It acts by depressing the release of C-fibres transmitter and by hyperpolarization of post synaptic dorsal horn neuron, which produces analgesia.[7]

(Correa-Saler C, Rabin BC, Maze M) DXM and clonidine both is [alpha] 2-adrenoreceptor agonist agents initially prescribed for hypertension and intravenous sedation. Gradually the role of these two agents extended beyond wards to operation theatre for the provision of intraoperative and postoperative analgesia and sedation.[8]

Van Tuijl I, added various doses of clonidine (0, 15 or 30 μg) to 5 mg hyperbaric bupivacaine and evaluated their effect on the duration of the motor block, analgesic quality and ability to void. They opined that addition of 15 and 30 μg of clonidine increased the motor block duration by 25 and 34 min, respectively and also resulted in better analgesic quality.

Hutschala D, Mascher H added clonidine to bupivacaine and found that it enhances and prolongs analgesia after brachial plexus block via a local mechanism in healthy volunteers.

Niemi L studied effects of intrathecal clonidine on duration of bupivacaine spinal anesthesia, hemodynamics, and postoperative analgesia in patients undergoing knee arthroscopy and found that intrathecal clonidine significantly prolongs the anesthetic and analgesic effects of bupivacaine.[9]

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Studies conducted by Kaabachi et al. Elia et al, Saxena et al, B. S. Sethi el al, Cao JP similarly showed that addition of various doses of clonidine to bupivacaine intrathecally significantly prolongs duration of analgesia of bupivacaine.[10]

When we compared the dexmedetomidine in different doses, we found that onset of motor block and sensory block was delayed as compared to control group. The difference was statistically insignificant. Higher dose of Dexmedetomidine produced significantly longer duration of sensory and motor block as compared to low dose.

CONCLUSION

Supplementation of spinal bupivacaine with dexmedetomidine significantly prolonged both sensory and motor block compared with intrathecal bupivacaine alone. Patients in the groups that received dexmedetomidine had reduced post-operative pain scores and a longer analgesic duration than those who received spinal bupivacaine alone.

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