

A COMPARATIVE STUDY OF THE EFFECT OF ADDITION OF MAGNESIUM SULPHATE TO HYPERBARIC BUPIVACAINE IN PATIENTS UNDERGOING LOWER ABDOMINAL SURGERIES

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ABSTRACT

BACKGROUND

Magnesium is a noncompetitive blocker of N-Methyl-D-Aspartate (NMDA) receptor with anti-nociceptive effects. Recently it has gained popularity as an adjuvant in spinal anaesthesia. The addition of intrathecal magnesium sulphate to hyperbaric bupivacaine prolongs the duration of analgesia. In this prospective, randomized, double-blind study, we investigated the effect of adding intrathecal magnesium sulphate to bupivacaine in spinal anaesthesia for lower abdominal surgeries.

MATERIALS AND METHODS

Sixty ASA I or II adult patients of either sex undergoing lower abdominal surgeries were recruited. They were randomly allocated into two groups- Group M received intrathecal 15 mg bupivacaine (0.5% hyperbaric solution) combined with 0.5 mL of 10% MgSO₄; Group B received 15 mg bupivacaine (0.5% hyperbaric solution) combined with 0.5 mL of normal saline. We evaluated the onset of sensory and motor block, the duration of sensory and motor blockade and duration of spinal anaesthesia.

RESULTS

Onset of sensory and motor block was significantly shorter in Group B (2.06±0.79, 3.03±0.65 min) than Group M (4.84±2.24, 7.38±1.14 min). The duration of sensory block was significantly higher in Group M (220.78±9.4 min) than in Group B (165.78±17.6). The mean duration for complete motor recovery was maximum in Group M (271.44±34.72 min) than in Group B (180±18.41 min). Duration of analgesia was also longer in Group M (340.62±15.4 min) than with Group B (234.6±27.14 min) which was statistically significant (P<0.05).

CONCLUSION

In patients undergoing lower abdominal surgery, the addition of intrathecal magnesium sulphate to spinal anaesthesia induced by bupivacaine delayed the onset of both sensory and motor blockade, but improved the quality and the duration of postoperative analgesia.

KEYWORDS

Adjuvant, Magnesium Sulphate, Bupivacaine, Intrathecal, NMDA Receptor.

HOW TO CITE THIS ARTICLE: Anshu, Angra R, Gupta H, et al. A comparative study of the effect of addition of magnesium sulphate to hyperbaric bupivacaine in patients undergoing lower abdominal surgeries. J. Evid. Based Med. Healthc. 2018; 5(41), 2918-2921. DOI: 10.18410/jebmh/2018/596

BACKGROUND

Neuraxial anaesthesia has been widely used to provide anaesthesia, especially in the lower abdominal surgeries. Local anaesthetics are the commonest agents used for spinal anaesthesia, but their relatively short duration of action may lead to early analgesic intervention in the postoperative period.^{1,2} A number of adjuvants to local anaesthetics have been used intrathecally to prolong the intraoperative as well

as postoperative analgesia.³ Opioids are commonly used as intrathecal adjuvants to improve the quality of intraoperative analgesia and prolong it in the postoperative period. However, side effects such as pruritus, nausea, vomiting, urinary retention, and delayed respiratory depression have prompted further research toward non-opioid analgesics with less serious side effects.

Magnesium blocks NMDA channels in a voltage-dependent way, and the addition of magnesium produces a reduction of NMDA-induced currents. Magnesium sulphate has been used systemically, and has shown antinociceptive effects.^{4,5} Intrathecal magnesium has been found to prolong the duration of analgesia in various surgical procedures like lower limb surgeries^{6,7} and as adjuncts to epidural and general anaesthesia.⁸⁻¹³

Therefore, the present study was designed to evaluate whether addition of intrathecal magnesium sulphate would

Financial or Other, Competing Interest: None.
Submission 17-09-2018, Peer Review 18-09-2018,
Acceptance 06-10-2018, Published 08-10-2018.

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DOI: 10.18410/jebmh/2018/596



enhance the analgesic efficacy of intrathecal bupivacaine in patients undergoing lower abdominal surgeries.

MATERIALS AND METHODS

Sixty ASA class I-II patients of either sex in the age group of 18 to 55 years scheduled for lower limb surgeries under spinal anaesthesia, were included in this prospective, randomized, double-blinded clinical study. Exclusion criteria included patients with uncontrolled diabetes mellitus and hypertension, history of long-term steroid therapy, chronic pain patients, neurologic or psychological disorders, morbid obesity, pregnancy and lactating mothers, patients with liver and kidney diseases.

After the ethics committee approval, written informed consent was obtained from all patients preoperatively. Patients were randomly allocated into two groups. Group M received intrathecal 15 mg bupivacaine (0.5% hyperbaric solution) combined with 0.5 mL of 10% MgSO4. Group B received 15 mg bupivacaine (0.5% hyperbaric solution) combined with 0.5 mL of normal saline. An insulin syringe was used to measure volumes <1 mL.

Thorough pre-anaesthetic check-up of all patients and all routine investigations were done. Tablet ranitidine 150 mg and tab alprazolam 0.25 mg were given as pre-medication on the night before planned surgery. All patients were kept nil orally for at least 8 h before surgery.

In the operating room, baseline values of heart rate, systolic blood pressure, diastolic blood pressure and oxygen saturation were recorded. After securing intravenous (IV) access with 18-G intravenous cannula, all patients received an IV preload of 10 ml/kg/h infusion of 0.9% sodium chloride (NaCl) solution before the subarachnoid block. After antiseptic skin preparation and sterile draping, lumbar puncture was performed at the level of L3–L4 vertebra with a 26-G Quincke spinal needle in lateral position and 3.5 ml of study drug was injected by anaesthesiologist blinded to the group assigned. After placing the patient in supine position, we assessed the onset and duration of sensory block, the duration of sensory and motor block and the total duration of spinal anaesthesia.

The sensory level was assessed by means of pinprick sensation using a blunt 25-G needle along the midclavicular line. The onset of sensory block was defined as the time to reach the sensory level up to T10 dermatome. The time for regression to S1 segment was taken to assess the duration of sensory block.

Motor blockade was assessed by using Modified Bromage Scale. The onset of motor block was defined as the time taken to achieve Bromage grade 3 motor block. The time taken for complete motor recovery to Bromage grade 0 was taken to assess the duration of motor block.

The duration of spinal anaesthesia was defined as the period from spinal injection of the study drug to the time when the patient received first rescue analgesic.

The hemodynamic parameters were monitored continuously during the perioperative period. Any hypotension (mean arterial pressure <70 mmHg) episode was treated with ephedrine intravenous 6 mg bolus, and

episodes of bradycardia (heart rate <60 beats/min) were treated with 0.02 mg/kg of atropine. Postoperatively, Severity of pain was measured using a 10-point VAS score with 10 being severe pain and 0 being no pain. VAS score of more than 4, patients received rescue analgesia with inj. Tramadol 1 mg/kg iv. Any side effects during intraoperative and postoperative period were noted.

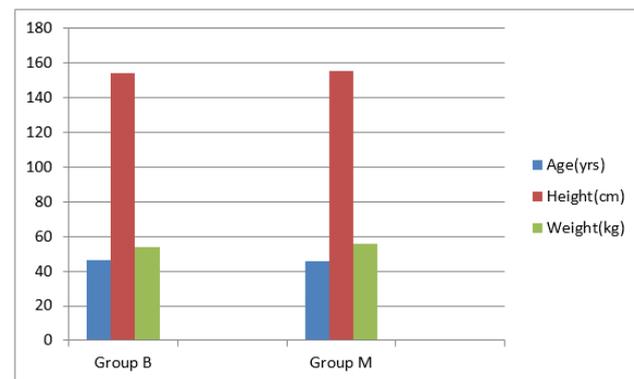
RESULTS

Statistical analysis was performed using the Chi-square or Fisher’s tests and Independent Student’s t-test where appropriate. A value of P < 0.05 was considered statistically significant. The results were expressed as mean±standard deviation.

There were no statistical differences in demographic data like age, height, and body weight between the two groups.

	Group B		Group M		t	P value
	Mean	SD	Mean	SD		
Age (yrs.)	46.4	9.8	45.8	7.5	0.25	0.80
Height (cm)	154.4	5.25	155.33	5.01	-0.70	0.48
Weight (kg)	54.20	8.49	55.60	8.16	-0.65	0.52

Table 1. Demographic Profile



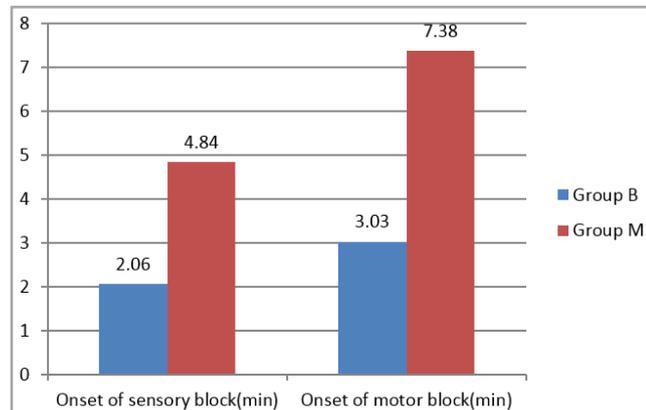
Graph 1. Demographic Profile

Onset of sensory and motor block was significantly shorter in Group B (2.06±0.79, 3.03±0.65 min) than Group M (4.84±2.24, 7.38±1.14 min). The duration of sensory block was significantly higher in Group M (220.78±9.4 min) than in Group B (165.78±17.6). The mean duration for complete motor recovery was maximum in Group M (271.44±34.72 min) than in Group B (180±18.41 min). Duration of analgesia was also longer in Group M (340.62±15.4 min) than with Group B (234.6±27.14 min) which was statistically significant (P<0.05).

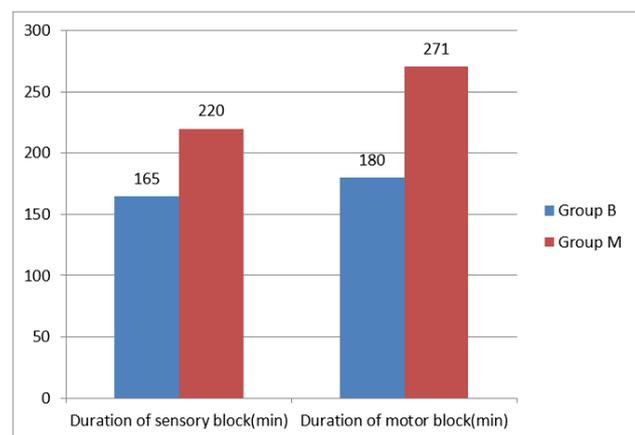
	Group B (Mean±SD) Min.	Group M (Mean±SD) Min.	
Onset of sensory block	2.06±0.79	4.84±2.24	P<0.05
Onset of motor block	3.03±0.65	7.38±1.14	P<0.05

Duration of sensory block	165.78±17.6	220.78±9.4	P<0.05
Duration of motor block	180±18.41	271.44±34.72	P<0.05
Duration of analgesia	234.6±27.14	340.62±15.4	P<0.05

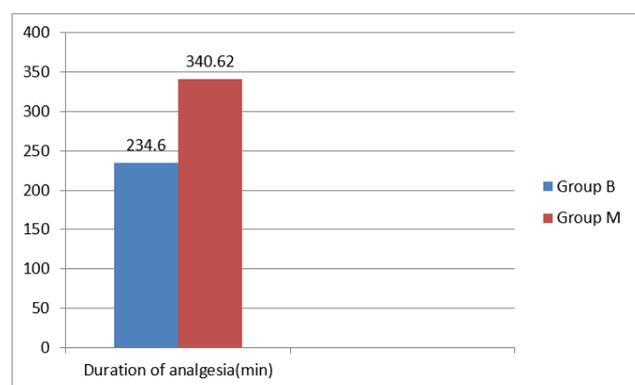
Table 2. Characteristics of Spinal Anaesthesia



Graph 2. Onset of Sensory and Motor Block



Graph 3. Duration of Sensory and Motor Block



Graph 4. Duration of Analgesia

DISCUSSION

Magnesium is a noncompetitive blocker of N-methyl-D-aspartate (NMDA) receptor with antinociceptive effects.^{4,5} Magnesium sulphate has been reported to be effective in perioperative pain treatment and in blunting somatic,

autonomic and endocrine reflexes provoked by noxious stimuli.^{9,10}

Recent studies suggest the role of magnesium sulphate as an adjuvant to local anaesthetics in spinal anaesthesia in different doses.¹⁴ Buvanendran et al.¹⁵ evaluated whether intrathecal magnesium could prolong spinal opioid analgesia in fifty-two patients who received either intrathecal fentanyl 25 µg plus saline or fentanyl 25 µg plus magnesium sulphate 50 mg as part of a combined spinal-epidural technique. Significant prolongation in the median duration of analgesia (75 min.) in the magnesium plus fentanyl group was observed compared with the fentanyl alone group (60 min.) without increased adverse effects.

The present study was conducted on sixty ASA class I-II patients of either sex in the age group of 18 to 55 years scheduled for lower abdominal surgeries under spinal anaesthesia. Patients were randomly allocated into two groups. Group M received intrathecal 15 mg bupivacaine (0.5% hyperbaric solution) combined with 0.5 mL of 10% MgSO₄. Group B received 15 mg bupivacaine (0.5% hyperbaric solution) combined with 0.5 mL of normal saline. In the present study, it was observed that the onset of sensory and motor block was delayed in group M. These results were also reported Ozalevli et al¹⁶ who observed that the onset of sensory and motor block was directly related to the dose of magnesium sulfate used, and, with an increase in dose, the onset was delayed. Ozalevli explained this delay is due to difference in pH and baricity of the solution containing magnesium.

In addition, we noticed that the duration of both sensory and motor block was prolonged in group M. Our finding is consistent with the findings of study by Manjula et al¹⁷ who demonstrated that the addition of intrathecal magnesium to intrathecal lipophilic opioid fentanyl along with local anaesthetic leads to delay in the onset of both sensory and motor blockade but significantly prolongs the duration of sensory and motor block following spinal anaesthesia. The total duration of analgesia was also found to be increased with magnesium as adjuvant. This was consistent with the results of other studies.¹⁸⁻²¹ Similar finding was observed by Ali E. Rashad and Emad El-Hefnawy²² who concluded that single dose of magnesium sulphate prolonged postoperative analgesia in patients receiving spinal anaesthesia and reduced the total dose of postoperative opioids with minimal side effects. This prolongation of anaesthesia is consistent with the experimental synergistic action between spinal local anaesthetic and NMDA antagonists like magnesium sulphate, which use anti-nociceptive effects via different mechanisms.²³⁻²⁵

CONCLUSION

From the present study, it can be concluded that the addition of magnesium sulphate 100 mg to bupivacaine for spinal anaesthesia in patients undergoing lower abdominal surgeries delays the onset of sensory and motor blockade but improved the quality and the duration of postoperative analgesia.

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