

ORIGINAL RESEARCH

Role of Intravenous Magnesium Sulphate in Spine Surgery for Hypotensive Anesthesia; A Randomized Control Trial

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ABSTRACT

Introduction: This randomized, double-blind placebo controlled study was undertaken to evaluate effect of intravenous magnesium sulphate for hypotensive anesthesia in patient for spine surgery.

Material and method: 60 patient undergoing spine surgery were included in two parallel groups. The magnesium group (M) received magnesium sulphate 40mg/kg as a bolus before induction of anesthesia and 10mg/kg/hr by continuous infusion during operation throughout surgery. The control group (C) as above received isotonic saline solution both as bolus and infusion at the same rate.

Result: The surgical time was reduced in magnesium group [170 (40) min] versus [180 (45) min] in control group. The anesthetic requirements (fentanyl, vecuronium, isoflurane), mean arterial pressure ($p < 0.005$) and heart rate ($p < 0.005$) were also significantly reduced. However the anesthetic time was longer as observed thus a prolongation in emergence from anesthesia.

Conclusion: Intravenous magnesium sulphate led to a significant decrease in mean arterial pressure, heart rate, and also reduced duration of surgery. It also alters anesthetic dose requirements.

Keywords: Anesthetic type, hypotensive; pharmacology, magnesium sulphate; surgery, spine surgery

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INTRODUCTION

Spine surgery, may be elective or emergency in patients presenting with problems like- trauma, infection, malignancy, congenital and idiopathic. Spine surgeries are associated with significant blood loss from decorticated bone and disruption of rich vascular network which increases time of surgery as well as time of anesthesia. Intraoperative bleeding reduces visibility in operative field. Hypotensive anesthesia is used to decrease blood loss and to shorten surgical time.

Anesthetic management depends on operative site, spine pathology, surgical approach and anesthetic intervention¹ Anesthetic consideration is to provide optimal surgical condition with ensuring adequate oxygenation to brain and spine cord.

Magnesium sulphate is a noncompetitive N-methyl-D-aspartate receptor antagonist. Magnesium inhibits calcium activation dependent on sarcoplasmic channel and limits outflow of calcium from sarcoplasmic reticulum. It also exerts its effect on L-type calcium channel in membrane to decrease blood pressure. It also acts as a vasodilator by increasing the synthesis of prostacyclin, as well as inhibiting angiotensin converting enzyme activity.³

It increases duration of effect of neuromuscular blocking agent and adds to antinociceptive effect.

Therefore we hypothesized that Magnesium sulphate may help in producing hypotensive anesthesia during spine surgery as well as decreases in anesthetic requirement.

MATERIALS AND METHODS

After obtaining institutional ethical committee approval, study was carried out on 60 patients, presented for elective spine surgery in Department of Anesthesia, Mahatma Gandhi Medical College, Sitapura, Jaipur, who were willing to give consent for study in a period of 2 years from 2012. They were assigned randomly by using computerized selection in one of two groups, 30 patients each.

Group M :- received 40 mg/kg Magnesium sulphate as bolus in 15 min with infusion later on till end of surgery at the rate of 10 mg/kg/hr

Group C:- received isotonic normal saline with same rate and volume

Patients were excluded on basis of following criteria:-

1. Refusal for consent
2. Associated with Hypertension, Morbid Obesity, Recent Myocardial Infarction
3. History Of Drug abuse,Addiction
4. Severe systemic dysfunction

Before induction, routine monitoring(electrocardiography, heart rate, blood pressure) was started, intravenous line was secured.

Heart rate, Mean arterial pressure were recorded before induction of anesthesia.

After preoxygenation for 5 min, anesthesia was induced with intravenous fentanyl 1.5ug/kg and intravenous-propofol 30 mg /5 second. When stable neuromuscular response achieved, intravenous vecuronium 100mcg/kg was given after which orotracheal intubation was performed. Hemodynamic data were collected 1 min before and 1 min after induction of anesthesia as well as 1min after intubation.

Anesthesia was maintained with 40% oxygen with Nitrous oxide, Isoflurane and intermittent dose of intravenous fentanyl 0.5mcg/kg and intravenous vecuronium 1mg when needed.

Dose of anesthetic agents were based on standard clinical sign and hemodynamic measurements. During perioperative period both groups received intravenous fluid at rate of 5ml/kg/hr. Patient positioned in prone position and vitals recorded after 10 min, 30 min, 60 min, 90 min & 120 min even if surgery took longer time. Intraoperative bleeding was measured by collection in suction bottle, drapes, gowns and soaked pads.

Intraoperative bleeding was measured by collecting blood with the pump graded with the precision of 25 ml. Forevaluation of the visibility of the operative field during surgery, qualityscale proposed by Fromm and Boezaart was used:

- 0: no bleeding.
- 1: slight bleeding—blood evacuation not necessary.
- 2: slight bleeding—sometimes blood has to be evacuated.
- 3: low bleeding—blood has to be often evacuated. Operativefield is visible for some seconds after evacuation.
- 4: average bleeding—blood has to be often evacuated. Operative field is visible only right after evacuation.

- 5: high bleeding—constant blood evacuation is needed. Sometimes bleeding exceeds evacuation. Surgery is hardly possible.

All infusions stopped either at the end of surgery or after 2 hr, Reversal of anesthesia was done with intravenous neostigmin 0.05mg/kg and intravenous glycopyrolate 0.02mg/kg. Patients were monitored in recovery area for any adverse effect.

Statistical analysis:

Sample size depended upon number of patients who were ready for giving consent and available for surgery, calculated with help of SPSS (IBM). Results are shown in tabulated form as mean and graphical representation of site of surgery as ordinal data showing categorical representation. Comparisons between both groups were conducted by using, t- test, one-way ANOVA test and Mann- Whitney-Wilcoxon test for failed normal tests. A P-value below 0.05 was considered significant.

RESULTS

The groups were comparable with respect to age, duration of surgery. All patients underwent the same type of surgery, performed by the same surgeon.

Variable	Group M	Group C	p-Value
Age(years)	44.43±1.57	44.50±1.50	0.86
Duration of Surgery (minutes)	170±40	180±45	0.367

Table-1: Demographic data

The preanesthetic and preoperative MAP were not significantly different (Table 2), between the groups, but after induction and at 10, 30, 60, 90, 120 min and at end of surgery they were significantly lower in the magnesium group ($P < 0.005$). A similar pattern was seen with heart rate.

Sixty patients ASA physical status I and II, undergoing elective spine surgery were included in two groups control ($n=30$); & magnesium ($n=30$).

The groups were comparable with respect to age, duration of surgery. All patients underwent the same type of surgery, performed by the same surgeon. There was a significant reduction in surgical time and the anesthetic requirements in the magnesium group. The preanesthetic mean arterial pressure were similar between the two groups initially, but after induction and at 10, 30, 60, 90 & 120 min of surgery were significantly lower in the magnesium group. Similar pattern was seen with heart rate. There were no episodes of hypotension ($MAP < 50$ mmHg), arrhythmia or reflex tachycardia during mag-

Time	Group M	Group C	p-Value
Preoperative	84.33±1.35	85.67±2.71	0.18
1 min before induction	83.33±1.68	84.17±2.99	0.18
1 min after induction	81.03±1.55	83.5±2.24	0.0001
1 min after intubation	82.70±1.34	90.67±1.37	0.0001
10 min	82.33±1.26	86.33±1.79	0.0001
30 min	81.63±1.30	88.16±2.40	0.0001
60 min	79.33±1.86	87.17±1.31	0.0001
90 min	77.00±1.09	90.67±1.09	0.0001
120 min	76.33±1.39	91.17±1.26	0.0001

Table-2: Mean Arterial Pressure (Data are presented as mean ± standard deviation)

Time	group C	group M	p-Value
preoperative	80.24±4.66	82.16±5.22	>0.05
1 min before induction	82.14±5.46	84.16±4.88	>0.05
1 min after induction	86.36±6.20	84.26±4.44	<0.05
1 min after intubation	89.52±4.20	84.20±4.29	<0.05
10 min	92.46±5.38	82.46±6.43	<0.05
30 min	82.46±4.64	75.68±4.12	<0.05
60 min	78.18±4.86	72.44±3.88	<0.05
90 min	76.44±3.96	70.46±4.22	<0.05
120 min	74.16±4.64	70.12±4.18	<0.05

Table-3: Mean heart rate (Data are presented as mean ± standard deviation)

nesium sulphate infusion, and no episode of rebound hypertension was seen on discontinuing magnesium sulphate infusion.

The condition at site of surgery were significantly better in the magnesium group than control group. The mean blood loss in the magnesium group was reduced. Discussion:

Magnesium sulphate is used as a hypotensive anesthesia technique in the magnesium group, there was an objectively better operative field, reduction in the duration of surgery and reduced blood loss.¹ In our study, the magnesium group received magnesium sulphate 40 mg./kg as a slow i.v. bolus in a 15 min period before the induction of anesthesia and 10 mg/ kg/ h by continuous i.v. infusion during the operation.

There was steady and smooth reduction in MAP and heart rate, with no episodes of severe hypotension.

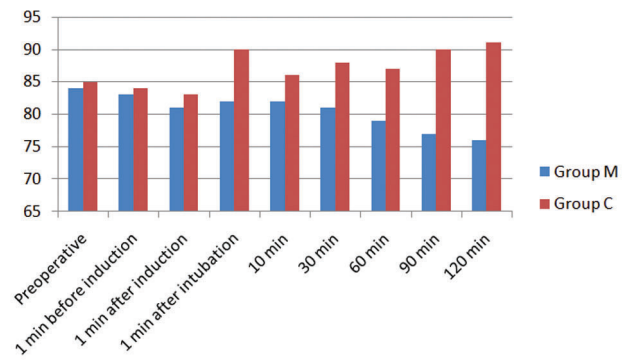


Figure-1: Mean Arterial pressure

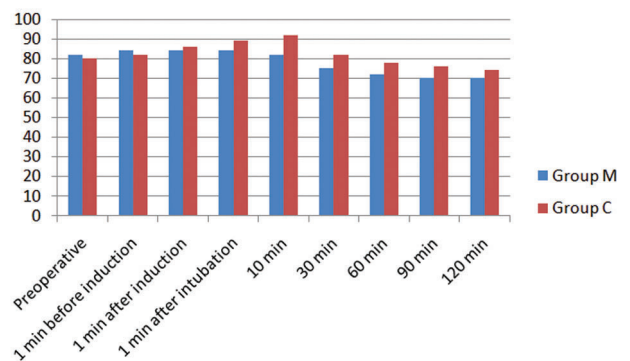


Figure-2: Mean Heart Rate

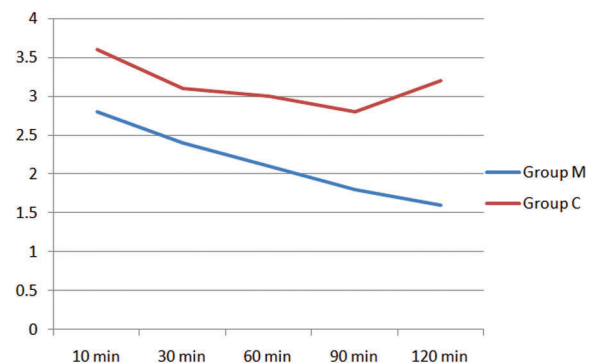


Figure-3: This graph represents score for site of surgery which shows better visual of site in group M.

Magnesium sulphate had been found to cause a reduction in arterial pressure, heart rate, blood loss and duration of surgery. To reduce bleeding intraoperatively, by producing deliberate hypotension, magnesium sulphate was used successfully. Non-Invasive arterial pressure monitoring was done and i.v. fluid was given, in both groups, at 5 ml /kg/h. No hemodynamic instability was seen in recovery when infusion was stopped. Magnesium sulphate was chosen as it is a vasodilator with minimal myocardial depression. Magnesium produces vasodilation by a direct action, as well indirectly by sympathetic blockade and inhibition of catecholamine release. The reduction in arterial pressure associated with a greater decrease in SVR is consistent with a

predominant peripheral vasodilator action of magnesium. It also exerts dose dependent depressant effect on cardiac contractility and offset by reducing peripheral vascular resistance.

A study using magnesium sulphate during sevoflurane anesthesia at doses of 30, 60 and even more than 120 mg kg⁻¹ concluded that magnesium did not have a deleterious effect on atrioventricular (AV) conduction time and surface ECG during 1 minimal alveolar concentration (MAC) of sevoflurane; and recommended the use of high doses of magnesium sulphate in patients with cardiac arrhythmia and hypertension during sevoflurane anesthesia.³

Magnesium helps in hypotensive anesthesia technique by causing an increase in cerebral blood flow velocity. The role of magnesium, in controlling intraoperative hypertension has been studied in hypertensive patients undergoing cataract surgery with local anesthesia, and was shown to reduce the intraoperative variability in arterial pressure.⁴

In a study of patients undergoing cerebral-aneurysm clipping, an initial magnesium sulphate dose of 40 gram/hour was used until MAP of 70 mm Hg was reached, followed by 20 gram/hour until the target MAP was reached, and then 10 gram/hour for the remaining time. The serum magnesium levels were high, and there was prolonged postoperative sedation; however, anesthesia had been maintained with a total intravenous technique using fentanyl and midazolam.⁷

Lower serum magnesium concentrations were found in a study of patients undergoing major oral and maxillofacial surgery, where magnesium was given at a rate of 40 gram/hour after induction until the MAP reached 55 mm Hg, followed by a maintenance dose of 5 gram/hour until 30 min before the end of surgery, without loss of the hypotensive effect. The total magnesium sulphate dose was 20–51.5 g (mean 33.5 g).³

This study also compared deliberate hypotension induced by sodium nitroprusside with that produced by magnesium sulphate. The magnesium sulphate infusion showed poorer control of arterial pressure and had a slower onset, taking 28 (12) min to reach the desired MAP.

As in our study, there was no rebound hypertension or reflex tachycardia associated with magnesium sulphate infusion. This may be attributed to hypermagnesemia producing inhibition of angiotensin-converting enzyme activity, sympathetic blockade, slowing of sino-atrial node transmission, depression of carotid baroreceptors, and diminished release of adrenal catecholamines.⁶

CONCLUSION

We conclude that Magnesium sulfate, used as an infusion led to reduction in MAP, heart rate and duration of surgery. Its use also reduced anaesthetic dose requirements and time of emergence. Further studies of this technique are warranted.

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