DOES MAGNESIUM SULFATE ATTENUATE HYPERTENSIVE RESPONSE TO LARYNGOSCOPY AND INTUBATION?

M.R. Pipelzadeh, Z. Poormehdi and M. Mobasher

Department of Anesthesia, Ahwaz University of Medical Sciences, Ahwaz, Iran

Abstract - Endotrachial intubation can have serious deleterious hemodynamic changes. Hypertension and tachycardia are especially dangerous in coronary artery disease, intracranial hypertension. Magnesium sulfate is known for its vasodilating properties. Several studies have reported favorable protective hemodynamic properties of magnesium when used as a premedicant in cardiac or elderly patients. Our aim was to evaluate whether magnesium had the same properties if used just before laryngoscopy in young healthy subjects undergoing elective non-cardiac surgery. This double-blind study was done in Ahwaz University during a 6-month period starting at November 2000. 90 American Society of Anesthesia class I and II patients scheduled for elective surgery under general anesthesia were included. Premedication was excluded and patients were anesthetized with sodium thiopental 5 mg/kg, morphine 0.1 mg/kg, atracurium 0.6 mg/kg. Patients then received either 50 mg/kg (not exceeding 4 g) magnesium sulfate (n=45), or normal saline as placebo (n=45) in a double-blind setting. Systolic and diastolic blood pressures and heart rate were recorded at 6 times: before induction, after induction 30 seconds and 1,2,3 minutes after intubation. Magnesium ion plasma levels were measured before induction and 5 minutes after intubation. Only diastolic pressure at 30 second after intubation had a significant lower value in magnesium group (P < 0.002) and other variables were statistically nonsignificant. In the magnesium group Mg^{++} plasma concentration was 5.936 ± 1.009 meq/l (mean ±SD) at 5 minutes after induction. We concluded that magnesium sulfate had a very limited usefulness in the attenuation of blood pressure and heart rate in young healthy patients if given during induction of anesthesia.

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INTRODUCTION

During light general anesthesia, direct laryngoscopy and intubation cause an increase in heart rate and arterial pressure and dysrhythmia in up to 90% of patients because of afferent stimulation of the vagus and a sympatho-adrenal reflex. Hypertensive subjects show an exaggerated response (1). These changes are particularly serious in patients with coronary artery disease, raised intracranial pressure, intracranial aneurysm or open eye surgery (2). Several drug regiments can minimize these responses, including narcotics (fentanyl and alfentanyl), lidocaine, vasodilators (nitroprusside, alpha blockers, nitroglycerine), alpha 2 agonits (clonidine), calcium channel blockers (verapmil, nifedipine), and ACE inhibitors (captopril) (2). Magnesium is considered nature's physiologic calcium blocker (4). Use of magnesium sulfate has been advocated due to its direct vasodilating effects, potent anti-arrhythmic properties and inhibition of catecholamine release from adrenal medulla (5). Magnesium acts mainly on resistance vessels rather than capacitance vessels (3) To utilize its vasodilating properties, James et al suggested a dose of 40-60 mg/kg as an IV bolus dose followed by infusion of 80 mg/kg/h in pheochromocytoma cases. They showed a dose related reduction in systemic vascular resistance with magnesium sulfate (6). Previous studies carried out under various surgical procedures showed magnesium sulfate to have temporary hypotensive effects, and increased stroke volume with an increase or no change in cardiac output (3). When compared with lidocaine, magnesium sulfate induced more marked control of blood pressure in elective coronary artery bypass graft (7) and cataract surgery (8,9). After intravenous injection of 4-6 g magnesium sulfate, blood level rapidly rises to 2.1-3.8 meq/l and returns to 1.3-1.7 meq/l within 60 minutes (6). Therapeutic range is considered at blood levels between 4-6 meq. Side effects of hypermagnesemia depend on magnesium plasma level and vary from nausea, flushing, and diplopia at therapeutic levels to respiratory depression and cardiac arrest at very high levels (12.5-14.6 meq/l) (6). Previous studies focused on magnesium sulfate as a premedicant in cardiac or senile patients.

We studied the effect of intravenous magnesium sulfate given during induction of anesthesia on hemodynamic changes due to laryngoscopy and intubation in young healthy American Society of Anesthesia (ASA) class 1 and 11 patients undergoing non-cardiac surgery under general anesthesia.

MATERIALS AND METHODS

This prospective clinical trial was done during a 6-month period from November 2000 in Ahwaz University teaching centers (Golestan and Emam Khomieni hospitals). Ninety American Society of Anesthesia (ASA) class 1,11 patients candidates for elective surgery, were randomly selected. Age of patients was between 20-40 years. We excluded all pregnant patients and those known for cardio-vascular disease. Patients received no premedications. All patients were monitored with ECG, pulse oxymetry and sphygmomanometer. Blood samples were drawn to measure plasma Mg^{++} levels before and 5 minutes after induction. Anesthesia was induced with sodium thiopental 5 mg/kg, morphine 0.1 mg/kg, and atracurium 0.6 mg/kg. Patients double-blindly received either magnesium sulfate (Daru-Pakhsh Iran,) 50 mg/kg, not exceeding 4 g or placebo (equal volume of normal saline). Injection was made within 60 seconds. In both groups heart rate and systolic and diastolic blood pressures were recorded at 6 intervals: before induction, before laryngoscopy and 30 seconds and 1,2,3 minutes after laryngoscopy. In order to assess the effect of magnesium on duration of atracurium, the time interval between the first and second dose of atracurium was recorded. Magnesium level was measured by atomic absorption using Carl Zeis FAA, with acetylene gas at a wavelength of 285.2 nm. To attain accuracy each sample was measured twice. Data were analyzed using single factor ANOVA test followed by Duncan's statistical analysis.

RESULTS

The magnesium treated group (25 female, 20 male), and the saline group (20 female, 25 male) had a mean age of 28.89 and 29.89 years respectively. Mean duration of repeated dose of Atracurium was 27.8 mg (magnesium group) and 29.7 mg (saline group), P=0.498. Side effects were similar in both groups. Urticaria was observed in both magnesium and saline groups as 22.2 and 28.8% respectively . One case in magnesium group developed arrhythmia (a few PVC's) that terminated as the rate of injection was reduced (Table 1). Systolic pressures were similar in both groups (Table 2).

Table 1. Side effects in both groups

Side effect	Magnesium	Saline
Flushing	4 (8.9%)	5 (11.1%)
Sweating	6 (12.3%)	2 (4.4%)
Arrythmia	1 (2.2%)	None
Urticaria	10 (22.2%)	13 (28.9%)

Table 2. Average diastolic blood pressure changes (mmHg)

Time event	Saline	Magnesium
Before induction	75.7	74.7
Before laryngoscopy	68.9	68.3
30 seconds	97.2**	90.4**
1 min	88.5	86.6
2 min	83.9	81
3 min	79.3	79

** magnesium group has significant change in 3rd measurement (30 sec. after laryngoscopy) with P = 0.002. Other values are related and not significant

Diastolic pressures were similar except for 30 seconds after intubation whereas the magnesium group showed lower diastolic pressures; p = 0.002. This difference was not seen at other stages (Table 3). Average heart rates were similar and statistically nonsignificant (Table 4).

Magnesium ion plasma concentration before and 5 minutes after intubation was 2.45 ± 0.21 meq/l and 5.93 ± 1.009 meq/l respectively.

Table 3. Average systolic pressure in both groups (mmHg) (P> 0.05)

Time event	Magnesium	Saline
Before induction	119.2	120.3
Before intubation	110.4	106.1
30 seconds	142.8	148.9
1 min	133.5	136.1
2 min	123.4	127.3
3 min	119.7	122.2

Table 4. Average heart rate changes in both groups (beat/minute) (P> 0.05)

Time event	Magnesium	Saline
Before induction	90	92.8
Before laryngoscopy	91.3	98.4
30 seconds	103	111.2
1 min	103.5	106.4
2 min	97	98.3
3 min	91	92.5

DISCUSSION

We did not see any serious side effect with the dose of 50 mg/kg injected in about 60 seconds. Most patients fell within therapeutic range (4-6 meq/l) except for one patient whose [Mg^{++}] was more than 8 meq/l. He did not show any ECG changes. About 20 % in each group had urticaria, which was probably due to histamine release by other induction drugs. Only one patient developed arrhythmia; 3-4 isolated PVC's appeared but disappeared as we reduced the speed of injection. No patient developed a heart block. Muscular weakness and respiratory depression was not a problem in the immediate postoperative period. Magnesium sulfate failed to have a significant effect in controlling systolic or diastolic pressures and heart rate in most events except at 30 seconds after intubation ($P = 0.002$), Table 2. But this advantage was offset in next measurement at 1 minute. This is contrary to previous studies by James (5), Pun (7), and Naghibi (9) who found significant lower heart rates and pressures in magnesium treated groups. The main difference was timing of injection. They gave magnesium sulfate along with premedication rather than at time of induction. We conclude that intravenous magnesium sulfate in a dose of 50 mg/kg does not effectively prevent unwanted hemodynamic consequences of laryngoscopy and intubation if given during induction of anesthesia. Magnesium sulfate does not affect the need of a second dose of atracurium. It seems that magnesium sulfate can be safely used with atracurium. Magnesium has several pharmacologic actions that make it attractive for the anesthetist. Its main toxic effect is neuromuscular paralysis and the route of elimination is renal, so that prolonged action may be anticipated in renal failure. Magnesium should be regarded a calcium antagonist with antiadrenergic properties accompanied by minimal cardiac depression.

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