Original Article

# The Perioperative Effect of Magnesium sulfate in Patients with Concentric Left Ventricular Hypertrophy Undergoing Cardiac Surgery: A Double-Blinded Randomized Study

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## Abstract

**Objective**: The objective of this study was to assess the cardioprotective effect of magnesium sulfate in patients with left ventricular concentric hypertrophy undergoing cardiac surgery. **Design:** The study was a double-blinded randomized study. **Setting:** This study was conducted at a cardiac center. **Patients:** The study included 250 patients. **Intervention:** The study included two groups (each = 125): Group M – the patients who received magnesium sulfate infusion (15 mg/kg/h). The infusion was started 20 min before induction, during surgery, and the first postoperative 24 h. Group C – the patients who received an equal amount of normal saline. **Measurements:** The variables included troponin I level, creatinine kinase-MB (CK-MB) level, electrocardiograph (ECG) with automatic ST-segment analysis (leads II and V), E/A peak ratio, end-diastolic volume, cardiac index (CI), heart rate, mean arterial blood pressure (MAP), mean arterial pulmonary pressure (mPAP), pulmonary and systemic vascular resistances, and pharmacological and mechanical support. **Main Results:** The troponin I level, CK-MB, and ECG changes were lower in Group M than Group C ($P < 0.05$). The E/A peak ratio and end-diastolic volume increased in Group M than Group C ($P < 0.05$). There was a significant increase in the CI and a decrease in the heart rate, mPAP, pulmonary vascular resistances, and pharmacological and mechanical support in Group M compared to Group C ($P < 0.05$). There were minimal changes in the MAP and systemic vascular resistance in Group M compared to Group C ($P < 0.05$). **Conclusion:** The magnesium sulfate provides a cardioprotective effect in patients with concentric ventricular hypertrophy undergoing cardiac surgery. It decreases the incidence of perioperative myocardial infarction and arrhythmia. Furthermore, it decreases the requirement of pharmacological and mechanical support.

**Keywords:** *Adult cardiac surgery, concentric ventricular hypertrophy, creatinine kinase-MB isoenzyme, magnesium sulfate, myocardial protection, troponin I*

## Introduction

Concentric myocardial hypertrophy is the physiological myocardial response to the increased cardiac afterload such as patients with systemic hypertension and aortic valve stenosis.[1,2] The physiological oxygen supply–demand ratio in hypertrophied myocardium is disturbed, and therefore, it is vulnerable to ischemic damage.[2] Hypertrophy also decreases the myocardial compliance and thereby impairs diastolic filling.[3] Furthermore, it is associated with increased myocardial oxygen consumption during cardiac surgery[4,5] and more liable for atrial fibrillation or flutter.[6,7] Ventricular hypertrophy can compromise the subendocardial blood flow by increasing coronary resistance and the increased filling pressures.[2]

The intraoperative goals for patients with ventricular hypertrophy are as follows: (1) to minimize the stressful stimuli and (2) to maintain the sinus rhythm and the hemodynamics stability.[8]

Magnesium plays an important role in preserving cardiac rhythm,[9,10] reducing the coronary microvascular injury during reperfusion, and preserving coronary microvascular function.[11] Hypomagnesemia can induce cardiac arrhythmias, especially in patients with ischemic heart disease and left ventricular hypertrophy.[12]

It was hypothesized in the present study, the perioperative magnesium will improve the cardiac outcome in patients with left ventricular hypertrophy, and therefore, this study was done to assess the perioperative effect of magnesium sulfate in patients with

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left ventricular concentric hypertrophy undergoing cardiac surgery.

## Methods

### Outcomes

The primary outcome was the myocardial protective effect which was assessed by the serum troponin I level, creatinine kinase-MB (CK-MB) level, arrhythmia, electrocardiograph (ECG) changes, development of postoperative new regional wall motion abnormalities, and improvement the diastolic dysfunction. A secondary outcome was the stability of postoperative hemodynamics and the requirement for pharmacological and mechanical cardiac support.

### Patients

After obtaining informed consent and approval of local ethics and research committee, a double-blinded randomized study included 250 patients with concentric left ventricular hypertrophy undergoing coronary artery bypass grafting (CABG) surgery or aortic valve replacement under cardiopulmonary bypass (CBP), diastolic dysfunction (impaired relaxation), coronary artery disease (patients with ischemic heart disease or percutaneous transluminal coronary angioplasty, previous CABG), or ventricular function (ejection fraction ≥40%). Exclusion criteria included patients with congestive heart failure, acute myocardial infarction, mitral or tricuspid valve surgery, malfunctioning artificial heart valve, obstructive cardiomyopathy, pericardial disease, and renal or hepatic impairment. The patients were assessed using New York Heart Association (NYHA), American Society of Anesthesiologists (ASA) Physical Status Score, and Euroscore. The patients were classified into two equal groups ($n$ = 125 each), and the concealment of allocation was done using random numbers generated through Excel.

- Group M: (magnesium sulfate group). The patients received a continuous infusion of magnesium sulfate (without a loading dose) at 15 mg/kg/h. The infusion was started 20 min before induction and maintained during surgery and the first postoperative 24 h. The medication was prepared by adding 5 g magnesium sulfate in 50 ml syringe
- Group C: (control group). The patients received an equal amount of normal saline.

### Anesthetic technique

For all patients and under local anesthesia before induction, a radial arterial cannula and central venous line were inserted; after induction, a pulmonary artery catheter was inserted to enable continuous hemodynamic monitoring. Induction was done by intravenous fentanyl (3-5 μg/kg), etomidate (0.3 mg/kg), and rocuronium (0.8 mg/kg). The anesthesia was maintained with oxygen/air (50%), sevoflurane (1%–3%), fentanyl infusion (1–3 μg/kg/h), and cisatracurium (1–2 μg/kg/min). At the end of surgical intervention, the patients were prepared for weaning from cardiopulmonary bypass (CPB). If there was difficulty to wean from CPB, pharmacological support (dopamine, epinephrine, norepinephrine, or nitroglycerine) or mechanical support (intra-aortic balloon pump and pacemaker) were started. At the end of surgery, the patients were transferred to cardiac surgery Intensive Care Unit (ICU) with full monitoring.

### Cardiopulmonary bypass

CBP was established with cannulation of the ascending aorta and right atrium. The patients received cold blood cardioplegia in the standard ratio (4:1); four parts of blood from the CBP circuit and one part potassium-rich crystalloid named Plegisol (Hospira Inc., Lake Forest, IL, USA). The initial dose was 30 ml/kg body weight, and subsequent doses were 20 ml/kg given every 20 min. The temperature was reduced to 28°C while maintaining a perfusion pressure of 100–125 mmHg. In the two groups, cardioplegia solution was given as two-thirds through the antegrade and one-third through the retrograde route, and a hotshot (warmblood) antegrade dose was given just before the myocardium reperfusion.

### Monitoring of patients

Hemodynamic monitoring included the heart rate, mean arterial blood pressure (MAP), a continuous electrocardiograph (ECG) with automatic ST-segment analysis (leads II and V), central venous pressure (CVP), cardiac index (CI), systemic vascular resistances (SVRs), pulmonary vascular resistances (PVRs), mean arterial pulmonary pressure (mPAP), pulmonary capillary wedge pressure (PCWP), urine output, troponin I level, and CK-MB level, required pharmacological and mechanical support in addition to the blood level of magnesium. Derived cardiovascular variables, namely CI, pulmonary, and SVR, were calculated using standard formulae, and the measurements were based on the bolus thermodilution technique using the mean of three consecutive 10 ml injectates of 5% glucose through the Swan-Ganz catheter. Transesophageal echocardiography (TEE) was done to obtain a standard sequence of cardiac images during surgery. Baseline and postoperative values were obtained by a cardiologist. Global left ventricular systolic function was evaluated by measuring the CI. The left ventricular diastolic function was evaluated by measuring the E/A peak ratio and end-diastolic volume. Furthermore, TEE assessed the right ventricle and valvular functions. In patients with postoperative ischemic changes in the ECG and elevated cardiac biomarkers, transthoracic echocardiography was done to diagnose the development of new regional wall motion abnormalities. Postoperative coronary angiography was done for patients with elevated cardiac biomarkers to assess the patency of the coronary grafts.

Hemodynamic values were serially collected at the following time points: T0: Baseline reading; T1: 15 min after induction; T2: Before CBP; T3: 30 min after CBP; T4: On ICU admission; T5: 6th h after ICU admission; T6: 12th h after ICU admission; and T7: 24th h after ICU admission. The troponin level was checked before surgery, before CPB, at the time of ICU admission, and at the 6th, 12th, 24th, 48th, and 72nd postoperative hours.

### Sample size calculation

Power analysis was performed using the Chi-square test for independent samples on the perioperative blood levels of troponin I and CK-MB because they were the main outcome variables in the present study. A pilot study was done before starting this study because there were no available data in the literature for the myocardial protective effect of magnesium sulfate in patients with concentric left ventricular hypertrophy undergoing cardiac surgery. The results of the pilot study (20 patients in each group) showed that the perioperative blood levels of troponin I and CK-MB increased in 35% of Group M and 50% of Group C. Taking power 0.8, alpha error 0.05, and beta 0.2, a minimum sample size of 125 patients was calculated for each group.

### Statistical analysis

Data were statistically described in terms of mean ± standard deviation or frequencies (number of cases) and percentages when appropriate. Comparison of numerical variables between the study groups was done using the Student's *t*-test for independent samples. Repeated measures analysis of variance test was used to see the effect of magnesium sulfate on hemodynamics (heart rate, MAP, CVP, CI, SVR, PVR, mPAP, and PCWP) at different follow-up intervals. For comparing categorical data, Chi-square test was performed. Fisher's exact test was used instead when the expected frequency is <5. $P < 0.05$ was considered statistically significant. All statistical calculations were done using computer program SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) version 20 for Microsoft Windows.

## Results

Table 1 shows no significant differences regarding the demographic data, comorbidities, preoperative medications, NYHA class, Euroscore, and the ASA physical status score ($P > 0.05$).

Figure 1 shows the changes in the hemodynamics of patients during the procedure and through the first 24 h in the ICU. There was no significant difference in the preoperative hemodynamics of patients of the two groups ($P > 0.05$). After magnesium sulfate infusion, the heart rate decreased in Group M and increased in patients of Group C, and the difference between the two groups was statistically significant ($P < 0.05$) [Figure 1a].

**Table 1: Preoperative data of patients' data are presented as mean±standard deviation, *n* (%)**

| Variable | Group M (*n*=125) | Group C (*n*=125) | *P* |
|---|---|---|---|
| Age (year) | 56.29±12.47 | 54.69±11.94 | 0.3011 |
| Weight (kg) | 89.17±15.40 | 86.18±13.76 | 0.106 |
| Gender | | | |
| Male:female | 75:50 | 66:59 | 0.307 |
| Diabetes mellitus | 107 | 98 | 0.187 |
| Hypertension | 113 | 117 | 0.485 |
| Ischemic heart diseases | 106 | 111 | 0.455 |
| Aortic stenosis | 67 | 61 | 0.447 |
| Atrial fibrillation | 42 | 38 | 0.684 |
| Pulmonary hypertension | 58 | 53 | 0.610 |
| Ejection fraction (%) | 44.35±4.10 | 45.25±4.63 | 0.105 |
| Angiotensin-converting enzyme inhibitors | 55 | 63 | 0.375 |
| Beta-blockers | 96 | 103 | 0.346 |
| Calcium channel blockers | 64 | 57 | 0.447 |
| Aspirin | 106 | 111 | 0.455 |
| Statins | 75 | 70 | 0.608 |
| Stroke | 7 | 4 | 0.539 |
| Carotid stenosis | | | |
| <50% | 30 | 24 | 0.442 |
| Unilateral | 12 | 8 | 0.485 |
| Bilateral | 18 | 16 | 0.853 |
| Smoking | | | |
| Current smokers | 78 | 86 | 0.351 |
| Ex-smokers | 24 | 15 | 0.397 |
| NYHA | | | |
| II | 13 | 9 | 0.633 |
| III | 88 | 95 | 0.391 |
| IV | 24 | 21 | 0.742 |
| ASA | | | |
| III: IV | 58: 67 | 69: 56 | 0.205 |
| Euroscore (%) | 15.40±6.28 | 14.68±5.85 | 0.349 |
| Blood sugar (mmol/L) | 7.58±1.44 | 7.36±1.39 | 0.220 |
| Body surface area (m²) | 1.78±0.19 | 1.77±0.17 | 0.661 |
| CABG | 71 | 63 | 0.374 |
| Aortic valve replacement | 19 | 14 | 0.455 |
| CABG and aortic valve replacement | 35 | 48 | 0.106 |

NYHA: New York Heart Association, ASA: American Society of Anesthesiologists Physical Status Score, CABG: Coronary artery bypasses graftiwwng, Group M: Magnesium sulfate group, Group C: Control group

There were minimal changes in the MAP in patients of the Group M and an increase in the MAP in patients of Group C, and the difference between the two groups was significant ($P < 0.05$) [Figure 1b]. The CI increased in patients of the Group M than Group C ($P < 0.05$) [Figure 1c]. There were minimal changes in the systemic vascular resistance in patients of Group M and an increase in patients of Group C ($P < 0.05$) [Figure 1d]. Regarding the mean pulmonary arterial blood pressure and PVRs, there was

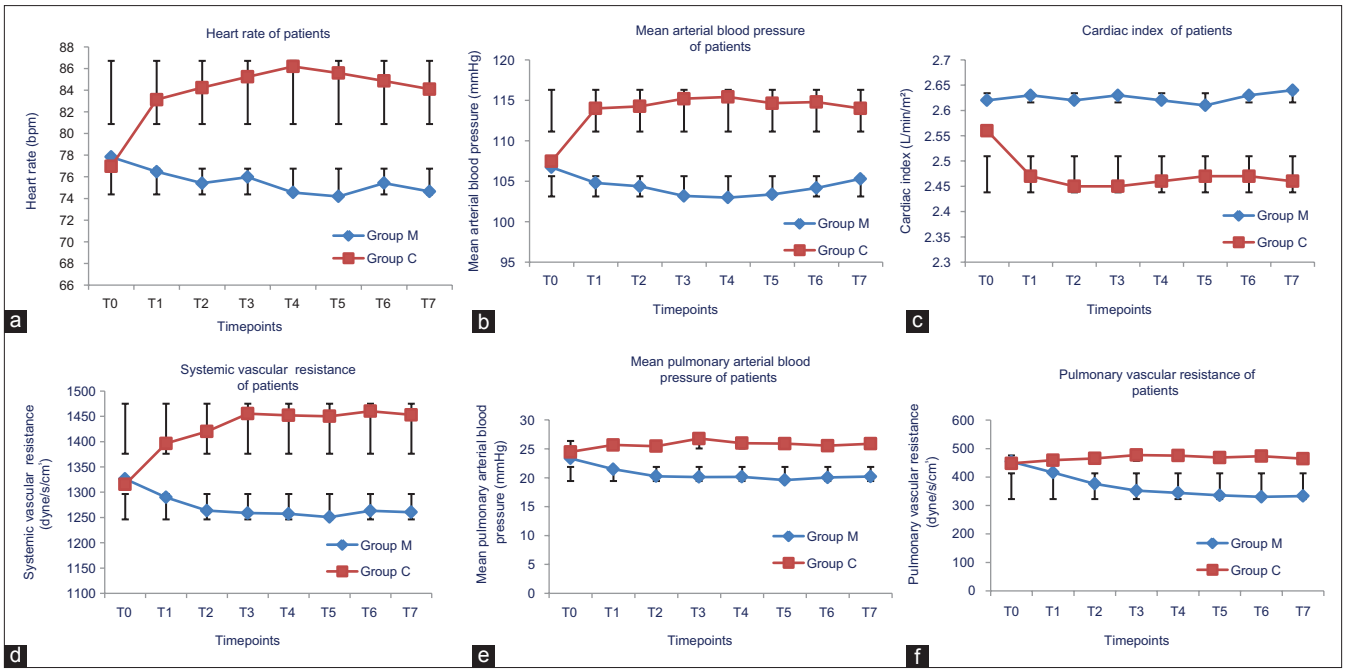


**Figure 1: The changes in hemodynamics of patients. a: Heart rate; b: Mean arterial blood pressure; c: Cardiac index; d: Systemic vascular resistance; e: Mean pulmonary arterial blood pressure; f: Pulmonary vascular resistance. T0: Baseline reading, T1: 15 min after induction, T2: Before cardiopulmonary bypass, T3: 30 min after cardiopulmonary bypass, T4: On ICU admission, T5: 6th h after ICU admission, T6: 12th h after ICU admission, T7: 24th h after ICU admission. ICU: Intensive Care Unit**

a decrease in patients of Group M and minimal increase in patients of Group C ($P < 0.05$) [Figure 1e and f]. There were no significant changes in the central venous pressure of patients or PCWP of patients between the two groups ($P > 0.05$).

Table 2 shows the changes in the left ventricular end-diastolic volume and E/A peak ratio. There was no significant difference in the preoperative left ventricular end-diastolic volume and E/A peak ratio between the two groups ($P > 0.05$), but after magnesium sulfate infusion, there was a significant increase in the left ventricular end-diastolic volume and E/A peak ratio in the Group M more than Group C ($P < 0.05$).

Figure 2 shows the changes in the blood level of magnesium. There was no significant difference in the preoperative blood magnesium level between the two groups ($P > 0.05$), but after magnesium infusion, the blood magnesium level was higher during and after surgery in the patients of Group M compared to patients of Group C ($P < 0.05$).

Figure 3 shows the changes in the blood level of troponin I and CK-MB. There was no significant difference between the two groups in the blood levels of troponin I and CK-MB before surgery or CPB ($P > 0.05$). After CPB, the troponin I and CK-MB increased in patients of the two groups; the increase was lower in patients of the Group M than patients of Group C through the first 24 postoperative hours, then the levels decreased through the second 24 postoperative hours, and the decrease continued through the third 24 postoperative hours and the difference between the two groups was statistically significant ($P < 0.05$) [Figure 3a and b].

Table 3 shows the intraoperative data and the outcomes of patients of the two groups. There was no difference in the CBP time, cross-clamping time, blood loss, transfused packed red blood cells, hematocrit value, neurological, and renal complications between the two groups ($P > 0.05$). The weaning from CPB was easier in patients of the Group M than the Group C. Patients of Group M needed smaller doses of pharmacological support (dopamine, epinephrine, norepinephrine, and nitroglycerine) than the Group C ($P < 0.05$), and the requirement for mechanical support intra-aortic balloon pump and pacemaker was lower in patients of Group M than the Group C ($P < 0.05$). The incidence of postoperative arrhythmias (atrial fibrillation and ventricular extrasystole) was lower in the Group M than the Group C ($P = 0.039$, $P = 0.033$, respectively). The number of patients associated with ECG changes (ST-segment changes) was 22 patients in the Group M and 37 patients in the Group C ($P = 0.036$). The number of patients suffered from postoperative myocardial infarction was 12 patients in Group M and 25 patients in the Group C ($P = 0.031$). The number of patients associated with postoperative new regional wall motion abnormalities was 12 patients in Group M and 25 patients in the Group C ($P = 0.031$). The number of patients suffered from postoperative myocardial infarction and associated occluded coronary grafts was 6 patients in Group M and 13 patients in the Group C ($P = 0.150$). The amount of transfused fluids and

**Table 2: End-diastolic volume of left ventricle and E/A peak ratio (data are presented as mean±standard devaition)**

| Variable | Group M (*n*=125) | Group C (*n*=125) | *P* |
|---|---|---|---|
| Left ventricle end-diastolic volume (ml) | | | |
| T0 | 59.36±12.20 | 61.15±13.45 | 0.271 |
| T1 | 69.38±15.35 | 64.50±14.77 | 0.011* |
| T2 | 71.57±15.90 | 67.14±14.10 | 0.020* |
| T3 | 75.17±16.75 | 69.88±15.60 | 0.010* |
| T4 | 78.45±16.80 | 73.93±16.25 | 0.031* |
| T5 | 78.68±16.46 | 74.13±15.69 | 0.026* |
| T6 | 80.17±16.98 | 75.30±15.85 | 0.019* |
| T7 | 87.10±18.44 | 75.30±16.70 | 0.002* |
| E/A peak ratio | | | |
| T0 | 0.65±0.25 | 0.71±0.32 | 0.099 |
| T1 | 1.02±0.40 | 0.90±0.36 | 0.013* |
| T2 | 1.12±0.56 | 0.95±0.53 | 0.014* |
| T3 | 1.18±0.50 | 1.00±0.52 | 0.010* |
| T4 | 1.18±0.41 | 1.07±0.34 | 0.021 |
| T5 | 1.22±0.64 | 1.03±0.56 | 0.013* |
| T6 | 1.21±0.45 | 1.09±0.38 | 0.023 |
| T7 | 1.20±0.43 | 1.08±0.39 | 0.021 |

*$P$<0.05 significant comparison between the two groups. Group M: Magnesium sulfate group, Group C: Control group, T0: Baseline reading, T1: 15 min after induction, T2: before cardiopulmonary bypass, T3: 30 min after cardiopulmonary bypass, T4: On ICU admission, T5: 6th h after ICU admission, T6: 12th h after ICU admission, T7: 24th h after ICU admission, ICU: Intensive Care Unit

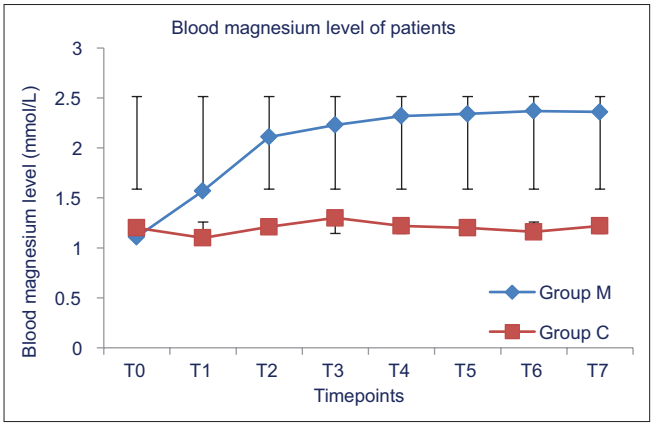


**Figure 2: Blood magnesium level of patients. T0: Baseline reading, T1: 15 min after induction, T2: Before cardiopulmonary bypass, T3: 30 min after cardiopulmonary bypass, T4: On ICU admission, T5: 6th h after ICU admission, T6: 12th h after ICU admission, T7: 24th h after ICU admission. Group M: Magnesium sulfate group, Group C: Control group. ICU: Intensive Care Unit**

intraoperative urine output was higher in Group M than Group C ($P$ < 0.05). The blood glucose increased in patients of the two groups, but the increase was lower in Group M than Group C ($P$ = 0.013). There was no difference in the incidence of stroke between the two groups ($P$ = 0.701), but the incidence of cognitive dysfunction (delirium) was lower in Group M than Group C ($P$ = 0.011). The incidence of new renal dysfunction (renal impairment or acute renal failure) was insignificant between the two groups ($P$ = 0.078, $P$ = 0.748, respectively). The ICU and hospital lengths of stay were shorter in patients of the Group M than the Group C ($P$ < 0.05). The incidence of mortality was lower in patients of the Group M than the Group C, but the difference was statistically insignificant ($P$ = 0.570).

## Discussion

The present study showed that the perioperative hemodynamics were controlled well by the magnesium sulfate; therefore, the outcome was better in Group M than Group C. The ECG changes (ST-segment) and cardiac markers (troponin I and CK-MB isoenzyme) were lower in Group M than Group C. The incidence of arrhythmias and myocardial ischemia was lower in Group M than Group C. The weaning from CPB was easier in Group M than Group C and the requirement for pharmacological or mechanical support was lower in Group M than Group C. The magnesium sulfate decreased the heart rate, so the oxygen demand decreased, and the incidence of ischemia decreased in Group M compared to the Group C. The diastolic dysfunction as monitored by the E/A ration and left ventricular end-diastolic volume improved with magnesium in Group M than Group C. The improvement of the diastolic dysfunction increased the end-diastolic ventricular volume, therefore, increasing the cardiac output.

The magnesium sulfate can induce relaxation of the stiff ventricle through the direct effect of magnesium sulfate on the myocardial contractility,[13] calcium-antagonist effect,[14,15] and the inhibition of catecholamines release.[16]

The results of the present study correlate with other studies that showed the cardioprotective effect of magnesium. Naghipour *et al*.[17] showed that magnesium sulfate significantly decreased the incidence of all types of postoperative arrhythmia after cardiac surgery. Gries *et al*.[18] reported that magnesium inhibits the platelet function and also, it has an effect on the clotting cascade as a calcium antagonist;[19] therefore, magnesium may produce a cardiac protective effect through the anticoagulant effect and patency of the coronary arteries or the grafts.[20] In an experimental study, Nakayama *et al*.[21] showed that magnesium increased the lusitropy and reversed the diastolic dysfunction even in the presence of higher levels of catecholamines.

Resatoglu *et al*.[22] documented that the addition of magnesium to the cardioplegia significantly decreased the postoperative concentrations of cardiac troponin I, creatinine phosphokinase, creatinine phosphokinase-MB, C-reactive protein, and lactate dehydrogenase compared with the control group and the same results were shown by other studies.[23,24]

Magnesium sulfate was used as an adjunct to primary coronary intervention in patients with acute myocardial

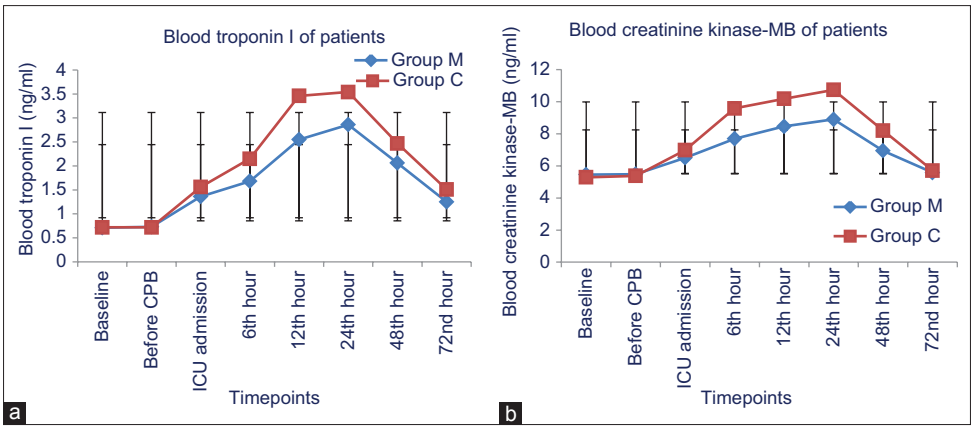


**Figure 3: (a) Blood troponin I, (b) Blood creatinine kinase-MB. ICU admission: Reading at ICU admission; 6th h: 6th h after ICU admission; 12th h: 12th postoperative hour; 24th h: 24th postoperative hour; 48th h: 48th postoperative hour; 72nd h: 72nd postoperative hour. Group M: Magnesium sulfate group, Group C: Control group. ICU: Intensive Care Unit**

infarction, and it minimized the reperfusion injury and led to a significant improvement of the left ventricular systolic function and decreased the mortality compared to patients who received normal saline.[25]

Magnesium plays an important role in preserving cardiac rhythm by stabilizing membrane function and prolongation of atrial and atrioventricular nodal refractory periods, which may facilitate rate and rhythm control.[9,10,26] Furthermore, the magnesium reduces the coronary microvascular injury during reperfusion and preserves coronary microvascular function.[11] It was shown that magnesium significantly improved the left ventricular systolic function and clinical outcome after acute myocardial infarction.[19] The cardioprotective effect of magnesium through its effect on calcium transport and minimizing the intracellular calcium, therefore, inhibits a variety of cellular enzymes, transport systems, and mitochondrial function and preserving the cellular adenosine triphosphatase and energy-dependent cellular processes.[27] Magnesium induces coronary vasodilatation and improves the coronary microcirculation and increases the coronary oxygen supply.[10,13,28] Furthermore, it reduces the myocardial oxygen consumption by lowering the heart rate, contractility, systemic afterload, and attenuating catecholamine-induced elevated oxygen demand,[13,29] and it reduces the production of free radicals, thereby protecting the postischemic myocardium from oxidative damage,[30,31] inhibits the catecholamine release from the adrenal glands.[23,24] Magnesium has an anti-inflammatory effect that may protect and minimize myocardial injury during and after CPB.[1] Honarmand *et al.*[32] showed that the preoperative administration of magnesium sulfate minimized the changes in the heart rate, systolic, diastolic, and mean arterial pressures related to laryngoscopy and tracheal intubation by inhibition of the catecholamine release from the adrenal glands[33] and its calcium antagonist effect.[15]

The amount of transfused fluids and intraoperative urine output was higher in Group M than Group C, and this may reflect the improvement of diastolic dysfunction and increasing the cardiac output. The magnesium inhibits the catecholamines and vasopressin release, therefore, producing renal vasodilatation and increasing the renal blood flow and urine output.[34,35]

In the present study, the blood glucose was better controlled in Group M than Group C, as the magnesium increases the cellular sensitivity to insulin and glucose.[36] One study showed that hyperglycemia at the time of myocardial ischemia is associated with increased mortality in diabetic and nondiabetic patients,[37] and also, the acute hyperglycemia is associated with increased platelet and leukocyte activation[38,39] that may lead to clot formation or an increase in the thrombus size and myocardial injury.[40,41]

The neurological outcome was better in Group M than Group C, and this may reflect the neuroprotective effect of magnesium and the same finding was shown by another study.[42]

Magnesium sulfate decreased the ICU and hospital length of stay and Naghipour *et al.*[17] showed the same results.

There are limitations to the present study. First, it was done in a single-center; and second, limited researchers talking about the same topics as the present study to discuss these findings in details.

## Conclusion

The magnesium sulfate induces a better cardioprotective effect in patients with concentric left ventricular hypertrophy undergoing cardiac surgery. It decreases the incidence of perioperative myocardial infarction and arrhythmia. Furthermore, it decreases the requirement of pharmacological and mechanical support, the ICU, and hospital length of stay.

### Acknowledgments

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**Table 3: Intraoperative data and outcome of patients data are presented as mean±standard deviation, *n* (%)**

| Variable | Group M (*n*=125) | Group C (*n*=125) | *P* |
|---|---|---|---|
| CPB time (min) | 114.19±28.60 | 111.76±25.95 | 0.482 |
| Cross-clamping time (min) | 91.55±18.38 | 93.90±19.48 | 0.327 |
| Dopamine | 6.67±1.55 | 7.18±1.62 | 0.011 |
| Epinephrine (µg/kg/min) | 0.05±0.02 | 0.07±0.04 | 0.001* |
| Norepinephrine (µg/kg/min) | 0.06±0.04 | 0.08±0.06 | 0.002* |
| Nitroglycerine (µg/kg/min) | 0.68±0.47 | 0.83±0.55 | 0.021* |
| Intra-aortic balloon pump | 13 | 29 | 0.011* |
| Pacing | 25 | 46 | 0.043* |
| Transfused P-RBC (unit) | 3.26±0.50 | 3.40±0.63 | 0.212 |
| Hematocrit (%) | 36.80±2.70 | 37.20±2.95 | 0.264 |
| Blood loss (ml) | | | |
| Intraoperative (ml) | 2169.90±210.83 | 2205.60±230.18 | 0.202 |
| Postoperative (ml/24 h) | 516.37±124.15 | 540.80±130.10 | 0.634 |
| Intraoperative fluids | | | |
| Crystalloids (ml) | 3250.70±563.25 | 3030.40±520.17 | 0.002* |
| Hesteril 6% | 740.27±12.740 | 630.60±115.90 | 0.001* |
| Postoperative fluids 24 h | | | |
| Crystalloids (ml) | 4453.70±990.24 | 4210.40±860.55 | 0.039* |
| Hesteril 6% | 1270.62±355.15 | 988.34±298.42 | 0.001* |
| Intraoperative urine output (ml) | 2220.75±286.14 | 2105.30±234.93 | 0.004* |
| Blood sugar (mmol/L) | 9.15±2.34 | 10.00±2.42 | 0.013* |
| Atrial fibrillation | 14 | 27 | 0.039* |
| Ventricular extrasystole | 16 | 30 | 0.033* |
| ECG changes (ST-segment changes) | 22 | 37 | 0.036* |
| Myocardial infarction | 12 | 25 | 0.031* |
| Regional wall motion abnormalities | 12 | 25 | 0.031* |
| Coronary angiography (occluded grafts) | 6 | 13 | 0.150 |
| Neurological complications | | | |
| Stroke | 3 | 4 | 0.701 |
| Cognitive dysfunction | 26 | 45 | 0.011* |
| New acute renal impairment | 7 | 16 | 0.078 |
| New renal failure | 4 | 6 | 0.748 |
| ICU length of stay (days) | 4.78±1.37 | 5.23±1.45 | 0.012* |
| Hospital length of stay (days) | 9.76±2.53 | 10.55±3.15 | 0.029* |
| Mortality | 5 | 8 | 0.570 |

*$P$<0.05 significant comparison between the two groups. Group M: Magnesium sulfate group; Group C: Control group, CPB: Cardiopulmonary bypass, P-RBC: Packed-red blood cells; ICU: Intensive Care Unit, Group M: Magnesium sulfate group; Group C: Control group, ECG: Electrocardiograph

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**Conflicts of interest**

There are no conflicts of interest.

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