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RESEARCH ARTICLE

# Efficacy of intravenous magnesium sulphate in prevention of contrast-inducec nephropathy after primary percutaneous coronary intervention

**Mohamed El-Hosanie Omar[a], Mohamad Wafaei Abou-Eleinin[b], Ismail Mohamed Ibrahim Elshaimaa A. Seaoud[b]***

*[a]Division of Cardiovascular Medicine, National Heart Institute, Cairo, Egypt*
*[b]Division of Cardiovascular Medicine, Zagazig University School of Medicine, Zagazig, Egypt*

***Corresponding author:** Elshaimaa A. Seaoud, Zagazig University, Faculty of Human Medicine. Orcid ID: https://orcid.org/0000-0003-2082-2389, Email: cardio_eg21@hotmail.com

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## Abstract

*Background:* Contrast induced nephropathy (CIN) is considered to be one of the most common major adverse effects of cardiac catheterization and is associated with short and long-term morbidity and mortality. CIN pathogenesis is still not completely understood although it is clear that the root concept is medullary hypoxia-induced renal tubular damage.

*Objective:* The study aimed to assess the efficacy of magnesium sulphate in prevention of CIN in patients undergoing primary coronary intervention.

*Methodology:* Our study was conducted on 100 patients who were randomly assigned to a study group IV hydration and 2 grams of intravenous magnesium sulphate group (n=50) and a control group (n=50) who were given only IV hydration Patients serum creatinine and kinetic GFR were assessed at a baseline, 2 hours and 48 hours after PPCI GFR was assessed using kinetic GFR equation, The primary end point was the occurrence of CI-AKI within 48 hrs. CI-AKI was defined as 0.5 mg/dl or more increase in serum creatinine or 25% or more increases above base line serum creatinine or drop in GFR more than 25%.

*Results:* In the group of patients who received Magnesium sulphate before PPCI, there was a significantly lower incidence of CIN compared to the control group. A multilogistic regression model showed that magnesium sulphate had a protective role against CIN while presence of RWMA (regional wall motion abnormality) before PPCI can predict post-procedural CIN.

*Conclusion:* Magnesium Sulphate may have a protective value and can reduce the incidence of contrast induced nephropathy after PPCI.

**Keywords:** Intravenous magnesium sulfate, Contrast induced nephropathy, Primary PCI

## Introduction

Contrast-induced nephropathy (CIN) is an acute kidney injury that frequently occurs after administration of contrast media, it is a well-known complication of cardiac catheterization.[1] The reported incidence of CIN varies widely in different populations, ranging from 7% to 25%, depending on the presence of risk factors. Its development has been associated with increased in-hospital and

long-term morbidity and mortality, prolonged hospitalization, and long-term renal impairment.[2] The most common definition of CIN is either an increase in baseline serum creatinine 25 % or more above the baseline or an absolute increase in serum creatinine by greater than 0.5 mg/dl above baseline.[3] There is currently a general agreement that adequate pre procedural hydration constitutes the cornerstone of prevention, yet there are reports of the use of some other agents with various efficacies.[4]

Magnesium sulphate is one of the agents that showed promising results in prevention of renal impairment. Magnesium inhibits calcium-induced cell death. It is anti-apoptotic in mitochondrial permeability transition and antagonizes calcium overload-triggered apo-ptosis. Recent studies have proved that it plays a conspicuous role in the pathogenesis of cardiovascular diseases on the biochemical and cellular level.[5]

Low serum magnesium has been associated with inflammation and disturbances in the regulation of vascular tone and endothelial function, thus it is thought to contribute to the development and progression of atherosclerosis, potentially worsening coronary heart disease.[6] High plasma magnesium levels inhibit blood coagulation and thrombus formation in vivo, diminish platelet aggregation reduce synthesis of platelet agonist thromboxane A2, and inhibits thrombin-stimulated calcium influx. Additionally, studies have shown that magnesium can suppress platelet activation by either inhibiting platelet-stimulating factors, such as thromboxane A2, or by stimulating synthesis of platelet-inhibitory factors, such as prostacyclin (PGI2).[7]

## Patients and Methods

This interventional randomized control study included 100 patients who attended to the coronary care unit at Zagazig University Hospitals from Aug 2019 to March 2020 with diagnosis of STEMI according to the fourth universal definition of myocardial infarction.[8]

Patients were divided into two groups:

**Group 1 (control):** were 50 patients who were given normal saline 1–1.5 cc/kg from the time of procedure until 6 h after the procedure

**Group 2 (study):** given the same hydration protocol as group 1 and were also given IV magnesium sulfate 2 gram just before the procedure

The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki and has been approved by our institution's ethics committee. A written informed consent was taken from all enrolled patients.[9]

### Inclusion criteria

All patients admitted with acute myocardial infarction STEMI and older than 20 years old.

### Exclusion criteria

Patients were excluded from the study if one or more of the following criteria were present which may affect kidney function or blood flow which may affect our result, a baseline serum creatinine more than 2 mg/dl, patients presenting with cardiogenic shock or with an ejection fraction < 40 % before PPCI, History of end-stage renal failure or being on dialysis. History of intravenous contrast media administration within the previous 10 days. Finally, we excluded patients with a known metabolic disorder with impairment of serum magnesium level.

All patients underwent Complete history taking and full clinical examination and cardiac assessment.

12 leads ECG were done for each patient on admission. Calculation of ejection fraction was done using modified Simpson method and regional wall motion abnormality was assessed by two echocardiographers. Iso-osmolar non-ionic contrast iopromide was used for all patients.

## Biochemical evaluation

In all the patients, baseline serum creatinine was measured before angioplasty, 2 hours and 48 hours after the procedure using ***Beckman Coulter AU840 Biochemical Analyzer***. All the measurements were made in a single hospital-based laboratory, and the laboratory staffs were blinded to the study protocol and serum samples. Kinetic GFR was used to assess development of acute kidney injury (AKI) post PPCI. Due to the limited accuracy of GFR estimation using the Cockcroft-Gault, MDRD in assessment of acute changes in creatinine level. Kinetic GFR uses two serum creatinine measurements

(Cr1 and Cr2) , at two different time points (time 1 and time 2) in order to calculate the GFR thus it is essentially a dynamic creatinine clearance rate.[10]

## Measurement of kinetic GFR (kGFR)

Daily circulating creatinine is proportional the estimated GFR (derived from the MDRD equation) and initial creatinine concentration. A conversion factor was required to account for the difference in the units. Because creatinine is measured in μmol/L but GFR is expressed in mL/min/1.73 $m^2$, we must divide our calculation by 1000 to express the product correctly. To convert from mL/min to daily production, this is then multiplied by 1440 min/day. Overall, this gives a conversion factor of 1.44.[11]

The final formula used in calculating kinetic GFR is:

$$\mathrm{kGFR} = \frac{\text{daily circulating creatinine}}{\text{mean creatinine}} \times \left(1 - \frac{24 \times \text{change in creatinine}}{\Delta h \times \text{max potential change in creatinine per day}}\right)$$

$$\mathrm{kGFR} = \frac{\mathrm{Cr1} \times \mathrm{eGFR} \times 1.44}{(\mathrm{Cr1} + \mathrm{Cr2})/2} \times \left(1 - \frac{24 \times (\mathrm{Cr2} - \mathrm{Cr1})}{t \times (\mathrm{Cr1} \times \mathrm{eGFR} \times 1.44)(0.6 \times W)}\right)$$

Thus, daily circulating creatinine is creatinine concentration × eGFR × 1.44. The maximum change in 48 h will be proportional to both daily production and also the patient's body weight, which determines the volume of distribution of creatinine (0.6 × body weight in kg).[11]

### Statistical analysis

Data were analyzed using Statistical Program for Social Science (SPSS) version 24. Quantitative data were expressed as mean ± standard deviation (SD). Qualitative data were expressed as frequency and percentage. We used the following tests of significance: Independent-samples t-test, Chi-square ($\chi^2$) test. A multivariate logistic regression model to determine effect of independent variables on Kidney injury.

## Results

The study enrolled 100 acute myocardial infarction (STEMI) patients who underwent primary percutaneous coronary intervention.

Patients were divided into two groups:

Group I: (study) 50 patients.
Group II: (control) 50 patients.

Table 1 compares baseline characteristics biochemical, echocardiographic parameters and CI-AKI (contrast-induced - acute kidney injury) incidence of the studied groups. Mean age of group I was 56.78±12.40 while that of group II was 56.86 ± 11.00, no statistically significant difference was seen between the groups regarding age, gender, weight, incidence of smoking, hypertension, diabetes mellitus and amount of dye used (P > 0.05).

Group I had a higher mean WBCs count (Mean of 7.25 ± 2.03 in group I compared to 5.89 ± 1.83 in group II.) (P<0.001).Mean serum creatinine rose significantly in group II when compared to group I after PPCI (1.15 ± 0.42 in group I compared to 1.59 ± 0.9 in group II) (P=0.002). Similarly, mean GFR dropped significantly in group II compared to group I after PPCI. (Mean in group I = mean was 72.98 ± 20.34 compared to 56.46 ± 26.76 in group II.) (P < 0.001).

Incidence of CIN in group I was 18% compared to 42 % in group II, the difference was statistically significant (P = 0.013).

Patients also were divided into two groups based on incidence of CIN as follows:

Group A: no CIN included 70 patients.
Group B: CIN included 30 patients.

The baseline characteristics of the group A and B are shown in Table 2. The group which developed CIN (group B) included 30 patients whose mean age was 57.50 ± 11.46 years, 70% were males, 38% were hypertensive, 38 % were diabetic, 30% were smokers and mean weight was 85.83 ± 11.30. While group A (No CIN) were 70 patients whose mean age was 56.53 ± 11.82 years 71.4% were males, 44% were diabetic, 52% were hypertensive and 42% were smokers and mean weight was 85.16 ± 10.95.95. No statistically significant difference between the groups was seen regarding age, gender, risk factors of coronary artery disease/(hypertension, diabetes mellitus and smoking). Similarly no significant

**Table 1** Baseline characteristics , blood and lipid indices, kidney function tests biochemical, echocardiographic parameters, coronary angiographic data and CI-AKI incidence of the studied groups

| Items: (No.=100) | | All patients | Group I (Study group ) (N=50) | Group II (Control group) (N=50) | p-value |
|---|---|---|---|---|---|
| CIN | | 30 (30%) | 9 (18%) | 21 (42%) | 0.013 |
| Age (years) | | 56.82 ± 11.67 | 56.78 ± 12.40 | 56.86±11.00 | 0.973 |
| Sex | | | | | |
| Male | | 68 (68%) | 37 (74%) | 31 (62%) | 0.198 |
| Female | | 32 (32%) | 13 (26%) | 19 (38%) | |
| Weight (kg) | | 85.36 ± 11.01 | 86.92 ± 10.28 | 83.80 ± 11.58 | 0.157 |
| Risk factors | | | | | |
| Smoking | | 36 (36%) | 21 (42%) | 15 (30%) | 0.211 |
| HTN | | 45 (45%) | 26 (52%) | 19 (38%) | 0.159 |
| DM | | 41 (41%) | 22 (44%) | 19 (38%) | 0.542 |
| Dye Amount (CC) | | 261.48 ± 66.03 | 251.00 ± 67.76 | 271.96 ± 63.19 | 0.113 |
| Hb (mg/dl) | | 13.61 ± 1.84 | 13.75 ± 1.45 | 13.63 ± 1.35 | 0.680 |
| PLT (*1000/cc) | | 297.02 ± 76.26 | 283.34 ± 65.26 | 310.70 ± 84.3 | 0.073 |
| WBCS (*1000/cc) | | 6.57 ± 2.04 | 7.25 ± 2.03 | 5.89 ± 1.83 | <0.001* |
| Urea (mg/dL) | Before | 42.45 ± 9.22 | 43.86 ± 9.53 | 41.04 ± 8.76 | 0.127 |
| | After | 51.77 ± 16.60 | 51.60 ± 13.60 | 51.94 ± 19.29 | 0.919 |
| Creatinine (mg/dl) | Before | 1.02 ± 0.21 | 1.01 ± 0.22 | 1.04 ± 0.20 | 0.486 |
| | After | 1.37 ± 0.73 | 1.15 ± 0.42 | 1.59 ± 0.90 | 0.002* |
| GFR (mL/min) | Before | 80.78 ± 24.08 | 86.00 ± 23.53 | 75.55 ± 23.71 | 0.029* |
| | After | 64.72 ± 25.06 | 72.98 ± 20.34 | 56.46 ± 26.76 | <0.001** |
| EF | | 58.20 ± 5.69 | 57.68 ± 5.70 | 58.72 ± 5.67 | 0.642 |
| ECG | | | | | |
| ANT | | 63 (63%) | 33 (66%) | 30 (60%) | 0.654 |
| INF | | 26 (26%) | 11 (22%) | 15 (30%) | |
| Lateral | | 11 (11%) | 6 (12%) | 5 (10%) | |
| RWMA | | | | | |
| No | | 53 (53%) | 24 (48%) | 29 (58%) | 0.316 |
| Yes | | 47 (47%) | 26 (52%) | 21 (42%) | |
| Lesions | | | | | |
| LAD | | 62 (62%) | 33 (66%) | 29 (58%) | 0.579 |
| RCA | | 26 (26%) | 11 (22%) | 15 (30%) | |
| LCX | | 10 (10%) | 5 (10%) | 5 (10%) | |
| OM | | 1 (1%) | 1 (2%) | 0 (0%) | |
| DIAG | | 1 (1%) | 0 (0%) | 1 (2%) | |

difference was seen between both groups in the type of coronary lesions, amount of dye, platelet, WBCs count or hemoglobin level (P = 0.7, 0.188, 0.579, 0.299, 0329 respectively). There was a significantly higher number of patients with regional wall motion detected at admission in group A compared to group B (45.7% compared to 70% respectively with a P = 0.026). Furthermore, 41 patients (58.6%) of group A received magnesium sulphate compared to 9 patients (30%) of group B.The difference was statistically significant (P = 0.009)

Table (3) shows A multivariate logistic regression model performed (crude and adjusted) to ascertain the effects of RWMA as a predictor and role of magnesium in protection against development of CIN. The result showed that magnesium had a protective role against CIN (p 0.011) with OR (0.317 and P-value < 0.05). While RWMA has a predictive role for development of CIN (OR > 1, and P value < 0.05).

## Discussion

Contrast-induced nephropathy (CIN) is an acute kidney injury that frequently occurs after administration of contrast media, it is a well-known complication of cardiac catheterization.[1] The reported incidence of CIN varies widely in different populations, ranging from 7% to 25%, depending on the

**Table 2** Comparison between baseline characteristics of group A (no CIN) and group B (CIN)

| Items: (No. = 100) | All patients | Group A No CIN (n=70) | Group B CIN (No=30) | p-value |
|---|---|---|---|---|
| Age (years) | 56.82 ± 11.67 | 56.53 ± 11.82 | 57.50 ± 11.46 | 0.705 |
| Sex | | | | |
| Male | 68 (68%) | 50 (71.4%) | 21 (70%) | 0.885 |
| Female | 32 (32%) | 20 (28.6%) | 9 (30%) | |
| Weight (kg) | 85.36 ± 11.01 | 85.16 ± 10.95 | 85.83 ± 11.30 | 0.780 |
| Risk factors | | | | |
| Smoking | 36 (36%) | 25 (42%) | 11 (30%) | 0.928 |
| HTN | 45 (45%) | 33 (52%) | 12 (38%) | 0.511 |
| DM | 41 (41%) | 32 (44%) | 9 (38%) | 0.143 |
| Hb (g/dL) | 13.61 ± 1.84 | 13.73 ± 1.39 | 13.33 ± 2.61 | 0.329 |
| RWMA | | | | |
| No | 53 (53%) | 38 (54.3%) | 9 (30.0) | 0.026* |
| Yes | 47 (47%) | 32 (45.7%) | 21 (70.0%) | |
| Lesions | | | | 0.707 |
| LAD | 62 (62%) | 43 (61.4%) | 19 (63.3%) | |
| RCA | 26 (26%) | 19 (27.1%) | 7 (23.3%) | |
| LCX | 10 (10%) | 7 (10% | 3 (10%) | |
| OM | 1 (1%) | 1 (2%) | 0 (0%) | |
| DIAG | 1 (1%) | 0 (0%) | 1 (2%) | |
| PLT (*1000/cc) | 297.02 ± 76.26 | 294.23 ± 76.96 | 303.53 ± 75.50 | 0.579 |
| WBCs (*1000/cc) | 6.57 ± 2.04 | 6.71 ± 1.96 | 6.24 ± 2.21 | 0.299 |
| Dye amount (cc) | 261 ± 66.03 | 64.8 ± 248.17 | 248.17 ± 67.9 | 0.188 |
| Magnesium | 100 (100%) | | | 0.009* |
| Mg | | 41 (58.6%) | 9 (30%) | |
| No MG | | 29 (41.4%) | 21 (70%) | |

**Table 3** Multivariate logistic regression (crude and adjusted) to detect independent predictors of CIN

| Variable | Crude | | Adjusted | |
|---|---|---|---|---|
| | Odds ratio (95% CI) | P-value | Odds ratio (95% CI) | P-value |
| Magnesium | 0.317 (0.125-0.805) | 0.016* | 3.057 (1.139-8.2) | 0.027* |
| RWMA | 2.631 (0.149-0.972) | 0.383 | 0.307 (0.113-0.833) | 0.020 |

* Adjusted: for age , gender, smoking, diabetes mellitus and hypertension.

presence of risk factors. Its development has been associated with increased in-hospital and long-term morbidity and mortality, prolonged hospitalization, and long-term renal impairment.[2]

Magnesium sulphate is one of the agents that showed promising results in prevention of renal impairment. Magnesium inhibits calcium-induced cell death. It is anti-apoptotic in mitochondrial permeability transition and antagonizes calcium overload-triggered apoptosis. Studies have proved that it plays a conspicuous role in the pathogenesis of cardiovascular diseases on the biochemical and cellular.[5]

Our study was conducted on 100 patients presenting to the ED with acute myocardial infarction all of which had a normal baseline KFT (kidney function tests). All of our study population underwent primary PCI. The aim of the study was to detect the effect of intravenous magnesium sulfate in prevention of contrast-induced nephropathy after PPCI.

Patients were divided into two equal groups (group I study): received MgSo4 before PPCI and control group II who didn't receive $MgSO_4$).

Our study showed that in patients who didn't receive $MgSO_4$, the mean of serum creatinine rose significantly, mean GFR dropped significantly and incidence of CIN was significantly higher than the other group.

Multilogistic regression done to test for predictors of CIN amongst our study groups showed that

RWMA could help predict post PPCI CIN while administration of $MgSO_4$ could help protect against CIN in this group of patients.

This was in agreement with Firouzi et al.[12] who studied the effect of Intravenous magnesium sulfate in prevention of contrast-induced nephropathy in primary percutaneous coronary intervention and estimated creatinine level before and after the procedure and found that Magnesium Sulphate has a protective role in prevention of CI-AKI. Prevalence of CIN was 17 (26.6 %) patients in the control group and nine (14.5 %) patients in the study group; there was a significant reduction in CI-AKI in the study group (P = 0.01).

Also, a large retrospective study by Oh et al. in 2019[13] studied the role of preoperative intravenous magnesium sulphate for prevention of CIN they conducted a multivariable logistic regression analysis that showed that magnesium infusion was associated with a significant decrease (63%) in postoperative AKI.

The multivariate logistic regression model showed that magnesium had a protective role against CIN (OR 0.317) while RWMA was a good predictor of CIN (OR 2.631).

This was in agreement with Mavrakanas and colleagues in 2019[14] they studied Echocardiographic parameters and renal outcomes in patients with preserved renal function receiving contrast media and found that patients with worse renal function had lower LV ejection fraction, higher prevalence of RV systolic impairment, higher LV and left atrium diameter and more regional wall motion abnormalities.

Regarding the role of magnesium sulphate in protecting against CIN and acute kidney injury this can be explained by the anti-inflammatory anti-apoptotic, antioxidant effect as $MgSO_4$ significantly inhibited endotoxin-induced up-regulation of inflammatory molecules and NF-κB activation in activated RAW 264.7 cells. The effects of $MgSO_4$ on inflammatory molecules and NF-κB may involve antagonizing calcium, inhibiting the L-type calcium channels, or both,[15] Also Magnesium Deficiency promotes endothelial dysfunction by stimulating the production of aldosterone and potentiates vascular inflammatory response, while expression/activity of various antioxidant enzymes (glutathione peroxidase, superoxide dismutase, and catalase) and the levels of important antioxidants (vitamin C, vitamin E, and selenium) also decreased due to $MgSO_4$ deficiency.[6]

So, in light of our results, we believe that the prophylactic use of intravenous Mg can help in lowering the incidence of CIN after Primary percutaneous coronary intervention.

## Conclusion

In primary PCI patients, the prophylactic use of intravenous Mg can be recommended to be added to traditional hydration for CI-AKI prevention.

The administration of IV Mg to patients with low-to-moderate risk of CIN scheduled for primary angioplasty in acute myocardial infarction may reduce the occurrence of CI-AKI.

## Recommendation

Larger sample size studies are recommended to consolidate our findings.

This study recommends using IV Mg to patients with low-to-moderate risk of CIN scheduled for primary angioplasty in acute myocardial infarction may reduce the occurrence of CI-AKI.

This study recommends considering Kinetic-GFR as a medical practice for estimating possibility for incidence of CI-AKI after PPCI.

## Limitations

This study is a single-center study with a limited sample size, we did not include all the patients with a significant CI-AKI with a higher potential for morbidity and mortality.

High-risk patients such as those in cardiogenic shock or severe ventricular dysfunction were not included in this study.

## Statement of Ethics

The study was approved by Zagazig University Ethical Committee of Zagazig, Egypt. All patients provided written informed consent.

## Disclosure Statement

The authors declare that they have no conflicts of interest to disclose.

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