INTERVENTIONAL CARDIOLOGY PERSPECTIVES

Inter Cardio Pers

DOI: 10.4274/intercardiopers.2025.2025-9-1

ORIGINAL ARTICLE

Effect of Magnesium Levels on the Progression of Contrast-induced Nephropathy in NSTEMI Patients Undergoing PCI

Sefa Erdi Ömür, Gülşen Genç Tapar, Çağrı Zorlu, Kayıhan Karaman

Department of Cardiology, Tokat Gaziosmanpaşa University Faculty of Medicine, Tokat, Türkiye

ABSTRACT

Background: Contrast-induced nephropathy (CIN) is the most common cause of hospital-acquired acute renal failure. The increased use of contrast media in diagnostic and interventional cardiac catheterization procedures and the increased frequency of these procedures have made CIN a common problem in clinical cardiology practice.

Aim: The aim of our study was to understand the role of magnesium (Mg^{2+}) levels in patients who developed CIN after non-ST elevation myocardial infarction (NSTEMI).

Study Design: The study was conducted retrospectively and a total of 4,324 patients who applied to the cardiology clinic with NSTEMI were included in the study.

Methods: This retrospective single-center study included 4,003 NSTEMI patients undergoing percutaneous coronary intervention (PCI). Patients with systemic inflammatory or rheumatologic diseases, hematologic disorders, renal or hepatic failure, infection, malignancy, or prior thrombolytic therapy were excluded. CIN was defined as an increase in serum creatinine ≥25% or ≥0.5 mg/dL within 48-72 hours after contrast exposure. Statistical analyses were performed using SPSS 26.0. Logistic regression identified independent predictors, and receiver operating characteristic (ROC) analysis determined the optimal magnesium cut-off for CIN prediction.

Results: CIN occurred in 1,062 patients (26.5%). Patients who developed CIN were older and had higher rates of diabetes mellitus, lower systolic blood pressure, and lower left ventricular ejection fraction. Serum magnesium levels were significantly lower in patients with CIN. ROC analysis identified an Mg^{2+} cut-off of 2.13 mg/dL [area under the curve=0.745, 95% confidence interval (CI): 0.681-0.809, p<0.001; sensitivity 78%, specificity 76%]. In multivariate analysis, age, diabetes, systolic blood pressure, preprocedural creatinine, estimated glomerular filtration rate, and Mg^{2+} <2.13 mg/dL (OR=2.02, 95% CI: 1.71-2.31, p<0.001) were independent predictors of CIN.

Conclusion: Low magnesium levels are independently associated with an increased risk of CIN in NSTEMI patients undergoing PCI. Routine assessment of Mg^{2+} before contrast exposure may help identify high-risk patients and improve prevention strategies.

Keywords: Magnesium, non-ST elevation myocardial infarction, contrast induced nephropathy

INTRODUCTION

Contrast-induced nephropathy (CIN), which is most often a reversible form of acute kidney injury, is a major complication of the coronary angiography (CAG) and percutaneous coronary procedures that is linked to adverse clinical outcomes.[1] Even when percutaneous revascularization is technically successful, the CIN occurrence in these patients has consistently been associated with prolonged hospitalization alongside higher mortality and morbidity rates.[2] Multiple determinants have been implicated in the CIN risk, including the type and the dose of the administered contrast, concurrent use of nephrotoxic medications, systemic inflammation, diabetes mellitus (DM), pre-existing renal impairment, heart failure (HF), advanced age, reduced hemoglobin concentrations, and female sex.[3] Coronary artery disease (CAD) is the most prevalent cardiovascular disorder that carries substantial mortality and morbidity. CAD may clinically manifest as silent ischemia, stable or unstable angina pectoris, acute myocardial infarction, HF, or even sudden cardiac death. When atherosclerosis underlies the presentation, CAD frequently appears as acute coronary syndrome (ACS). In contemporary practice, ACS is categorized into three groups based on the initial electrocardiogram and biochemical markers: ST-elevation myocardial infarction (STEMI); non-STEMI (NSTEMI), in which myocardial injury is verified by elevated damage markers despite no ST elevation; and unstable angina. For patients diagnosed with ACS, early therapy—thrombolytics in the coronary care setting and/or percutaneous coronary intervention (PCI) in the catheterization laboratory—is implemented to promptly restore patency and blood flow in a coronary artery that is acutely occluded by thrombus. PCI has markedly improved ACS management by reducing ischemic complications and improving survival; nevertheless, the

Address for Correspondence: Sefa Erdi Ömür MD, Department of Cardiology, Tokat Gaziosmanpaşa University Faculty of Medicine, Tokat, Türkiye
E-mail: sefaerdi61@gmail.com **ORCID ID:** orcid.org/0000-0002-6209-1732

Cite as: Ömür SE, Genç Tapar G, Zorlu Ç, Karaman K. Effect of magnesium levels on the progression of contrast-induced nephropathy in NSTEMI patients undergoing PCI. *Inter Cardio Pers*. [Epub Ahead of Print]

Received: 08.09.2025
Accepted: 20.10.2025
Epub: 03.11.2025

intraprocedural use of contrast agents heightens the likelihood of CIN characterized by an acute deterioration in the renal function.[4] The CIN pathophysiology remains incompletely clarified. Proposed mechanisms include perturbations in renal hemodynamics, injury mediated by oxygen-derived free radicals, direct tubular cytotoxicity of the contrast medium,[5] and vasoconstriction within the renal microvasculature.

The most widely used CIN definition is a serum creatinine increase exceeding 25% over baseline or an absolute rise greater than 0.5 mg/dL above the baseline level.[6] Clinically, CIN is considered an acute kidney injury typically emerging within 5-7 days after intravenous contrast exposure and cannot be explained by alternative etiologies;[7] however, the more severe CIN forms—those with a higher probability of requiring hemodialysis—most often present within the first 48 h following the procedure. There is broad expert agreement on the importance of adequate hydration and the avoidance of hypovolemia prior to contrast administration. In addition, several pharmacologic strategies have also been investigated in the literature for CIN prevention, including N-acetylcysteine,[8] theophylline,[9] fenoldopam,[10] sodium bicarbonate,[11] and ascorbic acid.[12] Among these, sodium bicarbonate did not demonstrate superiority over iso-osmolar saline infusion,[13] while the other agents have generally shown, at best, a marginal adjunctive efficacy when added to optimal hydration. The rationale for a magnesium-mediated protective effect in CIN draws on the following observations: CIN appears more frequent among patients with hypomagnesemia;[14] combining magnesium supplementation with N-acetylcysteine confers protection against post-ischemic acute renal failure;[15] and hypomagnesemia is implicated in the pathobiology of chronic cyclosporine nephropathy.[16] On the basis of these magnesium-related data in the CIN context, we sought to examine the association between the serum magnesium levels and the CIN development among patients with NSTEMI undergoing PCI.

METHODS

Study Design and Population

This retrospective, observational analysis included a total of 4,324 patients managed between August 2020 and August 2024. Due to insufficient or missing data, 321 individuals were excluded, leaving 4,003 patients for the final evaluation. Cases that were considered eligible were those who presented to the emergency department with an NSTEMI diagnosis and subsequently underwent CAG. Within this cohort, the patients were monitored for the CIN occurrence, and those who developed CIN were identified and assessed. A written informed consent was obtained from all the participants. The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Ethics Committee of Tokat Gaziosmanpaşa University Hospital (decision number: 25-MOBAEK-136; date: 24.04.2025). The study received no financial support from any institution or organization. Artificial intelligence-supported technologies, such as large language models, chatbots, or image generators, were not employed in the generation, processing, or analysis of the study data.

The serum magnesium levels were measured in patients with NSTEMI. Their relationship with the CIN development was also examined. The NSTEMI diagnosis was established according to the European Society of Cardiology Guideline criteria.[17] The patients were excluded if they received thrombolytic therapy prior to the invasive procedure, if an invasive procedure was not performed within 12 h of the symptom onset; or if they had systemic inflammatory or rheumatologic disease, storage disease, anemia, malignancy, were younger than 18 years, had any hematologic disease, including acute or chronic stroke, advanced renal and/or hepatic failure, a history of acute or chronic infection, blood transfusion within the prior 3 months, severe valvular disease, or prior valve surgery.

Laboratory Parameters and Demographic Data

The biochemical parameters were obtained using a Beckman Coulter LH-750 hematology analyzer (Beckman Coulter, Inc., Fullerton, California). All the blood samples were collected after an overnight fast with patients in a sitting or supine position. Routine laboratory tests, including serum magnesium, were drawn immediately prior to the PCI procedure. DM was considered as a fasting plasma glucose level >125 mg/dL, HbA1c >6.5%, or ongoing use of antidiabetic therapy (oral agents/insulin). The patients were considered to have hyperlipidemia if their total cholesterol and low-density lipoprotein cholesterol exceeded 200 mg/dL and 100 mg/dL, respectively, or if they used lipid-lowering medications. Hypertension (HT) was considered as the use of antihypertensive drugs or a systolic/diastolic blood pressure >140/90 mmHg. Individuals who smoked within the previous 6 months were classified as smokers. Serum creatinine was measured on hospital admission and again 48 to 72 h after exposure to the contrast agent. CIN was defined as a creatinine increase of >0.5 mg/dL or a ≥25% rise within 48 h following PCI.

Echocardiography, Coronary Angiography, and Risk Scoring

Prior to CAG, all patients underwent transthoracic echocardiography using the Vivid E7 (GE Vingmed Ultrasound) system with an MS5 (1.5-4.5 MHz) probe. The left ventricular ejection fraction (LVEF) was calculated using the Simpson method. All CAG and PCI procedures were performed on the Xper Allura FD-10 C-arm detector system (Philips Medical Systems International B.V., Best, Netherlands). The standard Judkins technique with a 6 Fr catheter was employed in all cases using either femoral or radial access. The procedure duration and the total volume of contrast administered were recorded. The PCI procedures were performed by two experienced interventional cardiologists. For each patient, the MEHRAN risk score (MRS) was calculated, incorporating eight clinical and procedural variables: age >75 years, hypotension, congestive HF, intra-aortic balloon pump, serum creatinine, DM, anemia, and contrast volume. The estimated glomerular filtration rate (eGFR) was calculated using the Cockcroft-Gault formula.

Statistical Analysis

All analyses and figure generation were executed in SPSS v26.0 (SPSS Inc., Chicago, IL, USA). For continuous measures, the distributional properties were evaluated both by formal testing with the Kolmogorov-Smirnov test and by a visual inspection of the histograms and quantile-quantile (Q-Q) plots. Based on these assessments, the continuous variables are described as mean±standard deviation when approximately normal or as median with interquartile range,

otherwise. The between-group contrasts used Student's t-test or the Mann-Whitney U test as appropriate. The categorical data were reported as counts and percentages and compared using Pearson's chi-square (χ^2) test. We identified the optimal cut-point of Mg^{2+} for predicting CIN by performing a receiver operating characteristic (ROC) analysis. The resulting threshold was then applied to dichotomize the Mg^{2+} levels. The CIN associations were first screened via univariable logistic regression. The variables meeting the significance criterion ($p<0.05$) were subsequently retained in a multivariable logistic regression model. The effect estimates are presented as odds ratios (OR) with corresponding 95% confidence intervals (CI). To aid the interpretation of the binary endpoint across the Mg^{2+} strata, we additionally plotted a stratified incidence graph and a model-based predicted-probability curve from the logistic regression. A two-sided $p<0.05$ was taken to denote the statistical significance throughout.

RESULTS

A total of 4,003 patients were included in the analysis. CIN was observed to develop in 1,062 individuals (26.53%). Table 1 summarizes the cohort's demographic, clinical, laboratory characteristics, and medications. The between-group comparisons demonstrated significant differences in age, DM, LVEF, systolic blood pressure, and MRS. In the laboratory profile, the preprocedural and postprocedural creatinine, hemoglobin and hematocrit, troponin, C-reactive protein, and Mg^{2+} levels were all statistically significant.

Among patients with NSTEMI, the ROC analysis identified an Mg^{2+} cut-off of 2.13 mg/dL with an area under the curve of 0.745 (95% CI 0.681-0.809, $p<0.001$), a sensitivity of 78%, and a specificity of 76% (Figure 1). In the multivariate logistic regression model, age, DM, systolic blood pressure, MRS, preprocedural creatinine, eGFR, and Mg^{2+} <2.13 mg/dL emerged as the independent predictors of CIN in the myocardial infarction population (OR: 2.019, 95% CI: 1.712-2.310, $p<0.001$; Table 2). Table 3 presents the CIN incidence distribution across the Mg^{2+} strata.

The graphical analyses in Figure 2 depict the association between the Mg^{2+} concentration and the CIN among 4,003 NSTEMI patients undergoing PCI. Figure 2a presents that 730 out of 1,920 (38%) patients with Mg^{2+} <2.13 mg/dL developed CIN, whereas 332 of 2,083 (17%)

Table 1. Baseline demographic, hematological, and angiographic characteristics, and medications of patients according to the presence or absence of contrast-induced nephropathy (CIN)

Variable	No-CIN (n=2941)	CIN (n=1062)	p
Age (mean±SD)	66.49±8.7	69.91±8.91	**0.003**
Gender (female, n%)	1323 (44.98)	477 (44.91)	0.883
BMI (mean±SD)	28.19±5.70	29.01±3.89	0.207
DM (n%)	1176 (39.98)	456 (42.93)	**0.013**
HT (n%)	1030 (35.02)	371 (34.93)	0.119
HL (n%)	852 (28.96)	297 (27.96)	0.503
COPD (n%)	529 (17.98)	192 (18.07)	0.294
Current smoker (n%)	1211 (41.17)	432 (40.67)	0.179
Previous myocardial infarction (n%)	812 (27.60)	290 (27.30)	0.209
HF (n%)	1128 (38.35)	413 (38.88)	0.228
LVEF (%)	50.21±5.78	47.54±6.33	**0.010**
Systolic blood pressure (mmHG)	112.20±15.36	83.69±10.30	**<0.001**
MEHRAN score	4.92±1.03	11.41±3.79	**<0.001**
Hematological results			
Pre-precedural creatinine (mg/dL)	0.95±0.23	1.23±0.43	**0.015**
Pre-precedural eGFR (mL/dk/1.73 m^2)	85.41±11.23	68.52±10.47	**<0.001**
Post-precedural creatinine (mg/dL)	1.02±0.47	1.84±0.70	**<0.001**
Post-precedural eGFR (mL/dk/1.73 m^2)	83.25±10.9	45.11±11.36	**<0.001**
Hemoglobin (g/dL)	12.28±3.79	9.56±3.41	**0.022**
Hematocrit value	40.11±5.39	33.40±5.71	**<0.001**
Platelet (x10^3/µL)	412.20±35.79	413.70±36.71	0.059
Total cholesterol (mg/dL)	218.20±19.44	221.70±18.61	0.843
LDL cholesterol(mg/dL)	128.36±15.3	129.28±14.8	0.597

Table 1. Continued

Variable	No-CIN (n=2941)	CIN (n=1062)	p
ALT (U/L)	41.23±9.3	42.13±8.52	0.491
AST (U/L)	31.57±8.20	32.47±7.90	0.145
Sodium (mmol/L)	134.41±8.70	135.09±7.07	0.555
Potassium(mmol/L)	4.57±1.08	4.45±1.12	0.637
Magnesium (mg/dL)	2.5±1.12	1.83±0.42	**<0.001**
CRP (mg/L)	13.54±3.54	25.47±5.31	**<0.001**
Troponin (ng/mL)	452.12±30.47	469.09±28.41	**0.035**
Coronary angiography procedure results			
LAD (n%)	882 (29.98)	318 (29.94)	0.761
LCX (n%)	891 (30.29)	323 (30.41)	0.593
RCA (n%)	1171 (39.81)	421 (39.62)	0.509
Amount of contrast medium (mL)	126.31±21.30	125.33±23.27	0.712
Procedure time (min)	42.20±10.41	41.39±9.39	0.831
TIMI flow grade	2.49±0.12	2.01±0.04	**<0.001**
Medications			
ACE inh/ARB (n%)	1852 (62.87)	659 (62.05)	0.887
Beta-blocker (n%)	1458 (49.57)	530 (49.90)	0.795
Statin medication (n%)	863 (29.34)	312 (29.37)	0.437
Antiagregan medication (n%)	896 (30.46)	329 (30.97)	0.913

ALT: Alanin aminotransferaz, AST: Aspartat aminotransferaz, LDL: Light density lipoprotein, TSH: Thyroid stimulating hormone, T4: Thyroxine, CRP: C-reactive protein, WBC: White blood cell, BMI: Body mass index, COPD: Chronic obstructive pulmonary disease, ACE: Angiotensin converting enzyme blocker, ARB: Angiotensin receptor blocker, SS: Syntaks score, DM. Diabetes mellitus, HT: Hypertension, HL: Hyperlipidemia, HF: Heart failure, LVEF: Left ventricular ejection fraction, eGFR: Estimated glomerular filtration rate, LAD: Left antrior desending artery, LCX: Circumflex artery, RCA: Right coronary artery, TIMI: Thrombolysis score in myocardial infarction, SD: standard deviation

Table 2. Univariate and multivariate regression analysis to identify independent predictors in contrast nephropathy patients

Variable	Odds ratio (95% CI)	p value	Odds ratio (95% CI)	p value
Age	1.156 (1.022-1.309)	0.018	1.221 (1.045-1.427)	0.011
Diabetes mellitus	1.342 (1.101-1.621)	0.007	1.287 (1.041-1.556)	0.019
LVEF (%)	1.052 (0.821-1.311)	0.321	0.893 (0.711-1.122)	0.274
Systolic blood pressure (mmHg)	1.189 (1.041-1.387)	0.014	1.165 (1.031-1.349)	0.021
MEHRAN score	1.952 (1.049-2.579)	0.021	1.876 (1.120-2.449)	0.008
Pre-procedural creatinine (mg/dL)	1.341 (1.112-1.615)	0.002	1.297 (1.076-1.559)	0.005
Pre-procedural eGFR (mL/min/1.73 m²)	1.298 (1.087-1.489)	0.009	1.244 (1.041-1.482)	0.017
Post-procedural creatinine (mg/dL)	1.118 (0.945-1.326)	0.172	1.083 (0.881-1.297)	0.282
Post-procedural eGFR (mL/min/1.73 m²)	1.097 (0.841-1.325)	0.411	1.061 (0.792-1.289)	0.503
Hemoglobin (g/dL)	1.032 (0.813-1.249)	0.672	0.943 (0.712-1.189)	0.461
Hematocrit value	1.153 (0.907-1.419)	0.247	1.098 (0.833-1.317)	0.391
CRP (mg/L)	1.221 (0.991-1.497)	0.061	1.093 (0.884-1.345)	0.337
Troponin (ng/mL)	1.137 (0.989-1.307)	0.071	0.913 (0.711-1.201)	0.511
TIMI flow grade	1.179 (0.974-1.417)	0.091	0.926 (0.742-1.165)	0.497
Magnesium <2.13 (mg/dL)	2.236 (1.821-2.419)	<0.001	2.019 (1.712-2.310)	<0.001

LVEF: Left ventricular ejection fraction, CRP: C-reactive protein, TIMI, Thrombolysis score in myocardial infarction, CI: Confidence interval, eGFR: Estimated glomerular filtration rate

Table 3. Relationship between magnesium levels and CIN development

Magnesium group	CIN (+) patients	CIN (-) patients	Total	CIN incidence
Mg <2.13 mg/dL	730	1190	1920	38%
Mg ≥2.13 mg/dL	332	1751	2083	17%
Total	1062	2941	4003	26.5%

CIN: Contrast-induced nephropathy

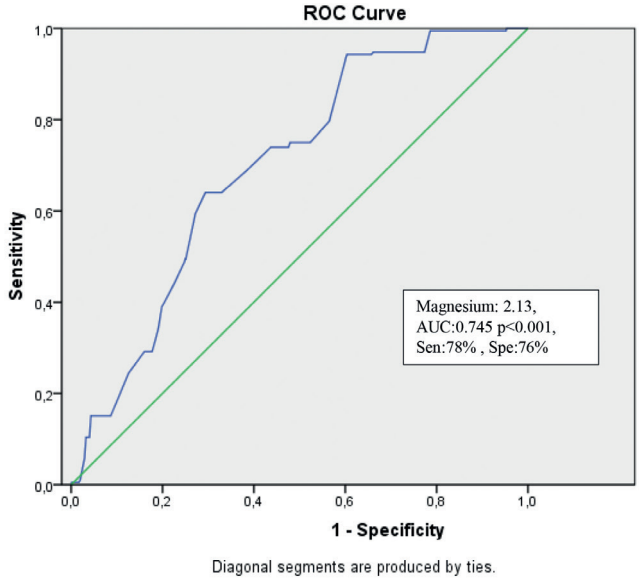

Figure 1. ROC curve showing magnesium levels in patients with non-STEMI CIN
ROC: Receiver operating characteristic, non-STEMI: Non-ST-elevation myocardial infarction, CIN: Contrast-induced nephropathy, AUC: Area under the curve

patients with Mg^{2+} ≥2.13 mg/dL experienced it. In Figure 2b, the logistic regression-derived probability curve demonstrates an inverse relationship between the Mg^{2+} level and the CIN risk, depicting a pronounced increase in the predicted probability below the 2.13 mg/dL threshold (red dashed line). The shaded green band represents a 95% CI.

DISCUSSION

We observed a robust association between the lower serum Mg^{2+} and the CIN development in this cohort of NSTEMI patients who underwent CAG and PCI. Beyond magnesium, several preprocedural clinical and biochemical characteristics were linked to the CIN risk, including reduced eGFR, lower systolic blood pressure, and higher creatinine concentrations before the procedure. Together, these findings underscore a multifactorial substrate for CIN in the NSTEMI population.

Pathobiologically, CIN reflects the interplay of intrarenal vasoconstriction, enhanced reactive oxygen species generation, and direct tubular injury.[18] Moreover, the abrupt vasoconstrictive response and the ensuing decline in the renal blood flow that follow contrast administration further catalyze the renal damage.[19] While the estimated CIN risk in the general population is approximately

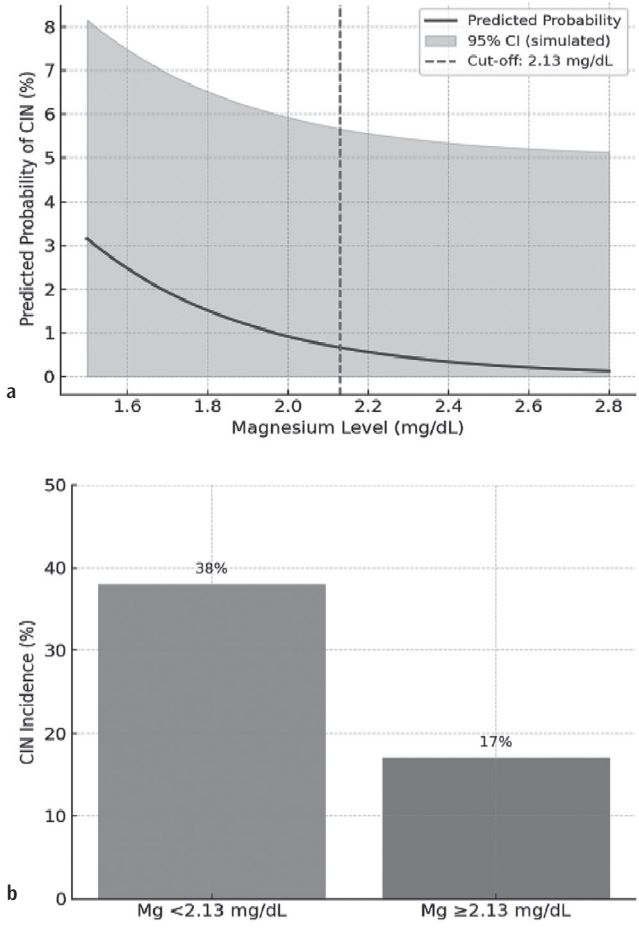

Figure 2. Relationship between magnesium levels and CIN development. (a) Stratified incidence of CIN by magnesium levels. Stratified incidence of CIN by magnesium levels. CIN occurred in 38% (730/1920) of patients with Mg <2.13 mg/dL and in 17% (332/2083) with Mg 22.13 mg/dL. (b) Predicted probability of CIN by magnesium level (logistic regression). Logistic regression curve showing predicted probability of CIN according to magnesium level. CIN risk increased sharply below 2.13 mg/dL (red dashed line, 95% CI shown in green).
CIN: Contrast-induced nephropathy, CI: Confidence interval

0.6-2.3%,[20] the incidence in patients undergoing PCI rises to roughly 15-35%.[21] Clinically, CIN is associated with longer hospitalization, higher mortality, and chronic renal failure.[7] It is also linked to an increase in major adverse cardiovascular events.[22] Consequently, current preventive practice emphasizes adequate hydration, iso-osmolar contrast selection, and intravenous isotonic saline. Although N-acetylcysteine can reduce the CIN occurrence, it does not appear to

influence hard outcomes like mortality or dialysis requirement.[23] From a mechanistic standpoint, magnesium is indispensable for cellular homeostasis and functions as a cofactor in a wide array of enzymatic reactions.[24] The experimental and clinical observations we obtained indicated that Mg^{2+} can dampen the macrophage activation and exert anti-inflammatory effects by lowering cytokines, such as interleukin (IL)-1β, IL-6, and IL-10,[25] while also mitigating the oxidative stress.[26] Therefore, hypomagnesemia is a plausible pathogenic contributor that promotes oxidative injury, reactive oxygen species accumulation, and endothelial dysfunction, all of which can facilitate the CIN evolution.

The pleiotropic vasodilatory, anti-inflammatory, anti-ischemic, and antiarrhythmic actions of magnesium suggest a protective role spanning both the cardiovascular and renal systems.[27] Consistent with this, prior literature has connected low Mg^{2+} with a greater cardiovascular risk[28] and with conditions like atherosclerosis, HT, insulin resistance, metabolic syndrome, and osteoporosis.[29] In the kidney, magnesium may prevent or reverse injury that is induced by nephrotoxic agents.[26,30] Among individuals with diabetic nephropathy, lower plasma Mg^{2+} is associated with a faster renal function decline and progression to end-stage renal disease.[31]The clinical data specific to the CIN prevention further support a role for magnesium. Firouzi et al.[32] reported that prophylactic magnesium reduced CIN in primary PCI patients, whereas Demirtola et al.[33] demonstrated a significant negative correlation existing between serum Mg^{2+} and CIN occurrence. Our results are in alignment with these observations and extend them by demonstrating—in a large NSTEMI cohort undergoing PCI—that low preprocedural Mg^{2+} is independently associated with a subsequent CIN risk, positioning Mg^{2+} as a potentially modifiable biomarker for risk stratification and targeted prevention.

Patients presenting with ACS are frequently hemodynamically unstable.[34] This instability can impair renal perfusion and limit the implementation of an adequate prophylactic hydration.[35] The urgency of CAG and the frequent need for diuretics may exacerbate medullary ischemia.[36,37] In our dataset, the systolic blood pressure was indeed significantly lower among patients who developed CIN, and this is consistent with these hemodynamic considerations.

From a practical perspective, the serum Mg^{2+} measurement is inexpensive, rapid, and universally available in hospital laboratories. Therefore, incorporating a routine pre-PCI Mg^{2+} assessment may offer a simple, low-cost means of identifying patients at a heightened risk. Furthermore, the magnesium supplementation in those with low levels can represent a feasible preventive strategy; nevertheless, prospective randomized trials are needed to establish the optimal dosing protocols and determine causality with greater certainty.

Study Limitations

This study has several limitations. First, its single-center, retrospective design restricts generalizability. Second, its retrospective nature did not allow us to fully control for all the potential confounders. Specifically, detailed information on the hydration status before and after the contrast exposure, use of nephrotoxic drugs, exact type and volume of contrast administered, and concomitant medications (e.g., ACE inhibitors and statins) were not systematically available. Each of these factors may materially affect the CIN risk and should be carefully captured in the future work. Additionally, we were also unable to evaluate other renal pathologies (e.g., proteinuria) that might have influenced the baseline renal function. To enhance the precision and the external validity of these findings, multicenter prospective studies incorporating a comprehensive assessment of these parameters are warranted.

CONCLUSION

This study demonstrates that the Mg^{+2} level is an important CIN determinant in patients with NSTEMI undergoing PCI. Considering the Mg^{+2} evaluation—and, where appropriate, preprocedural magnesium treatment—may be beneficial for the CIN prevention, particularly in individuals with low Mg^{+2} values.

Ethics Committee Approval: The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Ethics Committee of Tokat Gaziosmanpaşa University Hospital (decision number: 25-MOBAEK-136; date: 24.04.2025).

Informed Consent: Retrospective study.

Authorship Contributions: Surgical and Medical Practices: S.E.Ö., G.G.T., Concept: S.E.Ö., Design: S.E.Ö., Data Collection or Processing: S.E.Ö., Ç.Z., K.K., Analysis or Interpretation: S.E.Ö., Literature Search: S.E.Ö., Writing: S.E.Ö., G.G.T.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

REFERENCES

1. Pucelikova T, Dangas G, Mehran R. Contrast-induced nephropathy. *Catheter Cardiovasc Interv.* 2008;71:62-72.
2. Marenzi G, Lauri G, Assanelli E, et al. Contrast-induced nephropathy in patients undergoing primary angioplasty for acute myocardial infarction. *J Am Coll Cardiol.* 2004;44:1780-1785.
3. Yuan Y, Qiu H, Hu X, et al. Predictive value of inflammatory factors on contrast-induced acute kidney injury in patients who underwent an emergency percutaneous coronary intervention. Clin Cardiol. 2017;40:719-725.
4. He H, Chen XR, Chen YQ, Niu TS, Liao YM. Prevalence and predictors of contrast-ınduced nephropathy (CIN) in patients with st-segment elevation myocardial ınfarction (STEMI) undergoing percutaneous coronary ıntervention (PCI): a meta-analysis. *J Interv Cardiol.* 2019;2019:2750173.
5. Roozbeh J, Hamidian Jahromi A, Sharifian M, Pakfetrat M, Afshariani R. Protective effect of pentoxifylline on contrast induced nephropathy. *Saudi J Kidney Dis Transpl.* 2008;19:985-986.
6. McCullough P. "Renal complications of contrast media." *Interventional Cardiology. New York, NY: McGrawHill Medical* (2007).
7. Mehran R, Nikolsky E. Contrast-induced nephropathy: definition, epidemiology, and patients at risk. *Kidney Int Suppl.* 2006;(100):S11-15.
8. Briguori C, Colombo A, Airoldi F, et al. N-acetylcysteine versus fenoldopam mesylate to prevent contrast agent-associated nephrotoxicity. *J Am Coll Cardiol.* 2004;44:762-765.
9. Kapoor A, Kumar S, Gulati S, Gambhir S, Sethi RS, Sinha N. The role of theophylline in contrast-induced nephropathy: a case-control study. *Nephrol Dial Transplant.* 2002;17:1936-1941.

10. Walker PD, Brokering KL, Theobald JC. Fenoldopam and N-acetylcysteine for the prevention of radiographic contrast material-induced nephropathy: a review. *Pharmacotherapy*. 2003;23:1617-1626.
11. Kanbay M, Covic A, Coca SG, Turgut F, Akcay A, Parikh CR. Sodium bicarbonate for the prevention of contrast-induced nephropathy: a meta-analysis of 17 randomized trials. *Int Urol Nephrol*. 2009;41:617-627.
12. Spargias K, Alexopoulos E, Kyrzopoulos S, et al. Ascorbic acid prevents contrast-mediated nephropathy in patients with renal dysfunction undergoing coronary angiography or intervention. Erratum in: *Circulation*. 2005;111:379.
13. Kooiman J, Sijpkens YW, de Vries JP, et al. A randomized comparison of 1-h sodium bicarbonate hydration versus standard peri-procedural saline hydration in patients with chronic kidney disease undergoing intravenous contrast-enhanced computerized tomography. *Nephrol Dial Transplant*. 2014;29:1029-1036.
14. Staudinger T, Presterl E, Graninger W, et al. Influence of pentoxifylline on cytokine levels and inflammatory parameters in septic shock. *Intensive Care Med*. 1996;22:888-893.
15. Katholi RE, Woods WT Jr, Taylor GJ, et al. Oxygen free radicals and contrast nephropathy. *Am J Kidney Dis*. 1998;32:64-71.
16. Durham JD, Caputo C, Dokko J, et al. A randomized controlled trial of N-acetylcysteine to prevent contrast nephropathy in cardiac angiography. *Kidney Int*. 2002;62:2202-2207.
17. Byrne RA, Rossello X, Coughlan JJ, et al.; ESC Scientific Document Group. 2023 ESC Guidelines for the management of acute coronary syndromes. Erratum in: *Eur Heart J*. 2024;45:1145.
18. McCullough PA, Soman SS. Contrast-induced nephropathy. *Crit Care Clin*. 2005;21:261-280.
19. Sendeski MM. Pathophysiology of renal tissue damage by iodinated contrast media. *Clin Exp Pharmacol Physiol*. 2011;38:292-299.
20. Lasser EC, Lyon SG, Berry CC. Reports on contrast media reactions: analysis of data from reports to the U.S. Food and Drug Administration. Erratum in: *Radiology* 1997;204:876.
21. Silvain J, Nguyen LS, Spagnoli V, et al. Contrast-induced acute kidney injury and mortality in ST elevation myocardial infarction treated with primary percutaneous coronary intervention. *Heart*. 2018;104:767-772.
22. Ng AK, Ng PY, Ip A, et al. Impact of contrast-induced acute kidney injury on long-term major adverse cardiovascular events and kidney function after percutaneous coronary intervention: insights from a territory-wide cohort study in Hong Kong. *Clin Kidney J*. 2021;15:338-346.
23. Krzyżak D, Basiak M, Dec A, Hachuła M, Okopień B. The crucial question about contrast-ınduced nephropathy (CIN): should ıt affect clinical practice? *Pharmaceuticals (Basel)*. 2025;18:485.
24. Vormann J. Magnesium and kidney health - more on the 'forgotten electrolyte'. *Am J Nephrol*. 2016;44:379-380.
25. Tai Y, Qiu Y, Bao Z. Magnesium lithospermate B suppresses lipopolysaccharide-ınduced neuroinflammation in BV2 microglial cells and attenuates neurodegeneration in lipopolysaccharide-injected mice. *J Mol Neurosci*. 2018;64:80-92.
26. Long M, Zhu X, Wei X, et al. Magnesium in renal fibrosis. *Int Urol Nephrol*. 2022;54:1881-1889.
27. Muñoz-Castañeda JR, Pendón-Ruiz de Mier MV, Rodríguez M, Rodríguez-Ortiz ME. Magnesium replacement to protect cardiovascular and kidney damage? Lack of prospective clinical trials. *Int J Mol Sci*. 2018;19:664.
28. Chiuve SE, Korngold EC, Januzzi JL Jr, Gantzer ML, Albert CM. Plasma and dietary magnesium and risk of sudden cardiac death in women. *Am J Clin Nutr*. 2011;93:253-260.
29. Wyparło-Wszelaki M, Wąsik M, Machoń-Grecka A, et al. Blood magnesium level and selected oxidative stress ındices in lead-exposed workers. *Biol Trace Elem Res*. 2021;199:465-472.
30. Pere AK, Lindgren L, Tuomainen P, et al. Dietary potassium and magnesium supplementation in cyclosporine-induced hypertension and nephrotoxicity. *Kidney Int*. 2000;58:2462-2472.
31. Pham PC, Pham PM, Pham PA, et al. Lower serum magnesium levels are associated with more rapid decline of renal function in patients with diabetes mellitus type 2. Erratum in: *Clin Nephrol*. 2005;64:248.
32. Firouzi A, Maadani M, Kiani R, et al. Intravenous magnesium sulfate: new method in prevention of contrast-induced nephropathy in primary percutaneous coronary intervention. *Int Urol Nephrol*. 2015;47:521-525.
33. Demirtola Aİ, Mammadli A, Çiçek G. The role of magnesium levels in the progression of contrast-ınduced nephropathy in patients with STEMI undergoing primary PCI. *Angiology*. 2025;76:572-579.
34. Chong E, Poh KK, Liang S, Soon CY, Tan HC. Comparison of risks and clinical predictors of contrast-induced nephropathy in patients undergoing emergency versus nonemergency percutaneous coronary interventions. *J Interv Cardiol*. 2010;23:451-459.
35. Marenzi G, Assanelli E, Marana I, et al. N-acetylcysteine and contrast-induced nephropathy in primary angioplasty. *N Engl J Med*. 2006;354:2773-2782.
36. Sacha J, Gierlotka M, Feusette P, Dudek D. Ultra-low contrast coronary angiography and zero-contrast percutaneous coronary intervention for prevention of contrast-induced nephropathy: step-by-step approach and review. *Postepy Kardiol Interwencyjnej*. 2019;15:127-136.
37. Jurado-Román A, Hernández-Hernández F, García-Tejada J, et al. Role of hydration in contrast-induced nephropathy in patients who underwent primary percutaneous coronary intervention. *Am J Cardiol*. 2015;115:1174-1178.