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ORIGINAL ARTICLE

Association Between Admission Serum Magnesium Level and Clinical End Points in Patients with ST-Elevation Myocardial Infarction that Treated with Primary Angioplasty

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ABSTRACT

Background: Although serum magnesium (Mg) levels have been linked to atherogenesis and cardiovascular disease in the general population, evidence regarding their association with clinical outcomes in patients with acute myocardial infarction (MI) is inconsistent.

Aim: This study investigated the relationship between admission serum Mg levels and both in-hospital and long-term clinical outcomes in patients undergoing primary percutaneous coronary intervention.

Study Design: Retrospective cohort study.

Methods: This study included patients with ST-segment elevation MI (STEMI) who presented within 12 hours of symptom onset and underwent primary percutaneous coronary intervention between February 2011 and April 2015. After predefined exclusions, 1,119 of 1,270 patients were included. STEMI was diagnosed according to European Society of Cardiology/American College of Cardiology Foundation/American Heart Association criteria, and ethics approval was obtained with waiver of written informed consent. Admission clinical data and serum Mg levels were collected from medical records, with Mg measured before coronary angiography and grouped into five categories. All patients underwent femoral coronary angiography/stenting and received guideline-based medical therapy, with echocardiographic and follow-up data obtained from hospital records and telephone interviews.

Results: We included 1,119 patients with STEMI. The median follow-up duration was 25±16 months. An admission serum Mg cut-off of 1.83 mg/dL predicted acute stent thrombosis with 76% sensitivity and 65% specificity (area under the curve: 0.781; 95% confidence interval: 0.543–0.920; p=0.024). Apart from acute stent thrombosis, all other clinical endpoints were comparable across different serum Mg level groups.

Conclusion: Low admission serum Mg was significantly associated with acute stent thrombosis. However, no significant association was observed between serum Mg levels and in-hospital or long-term major adverse cardiovascular events, including cardiovascular mortality, target vessel revascularization, and stroke.

Keywords: Magnesium, myocardial infarction, percutaneous coronary intervention

INTRODUCTION

Since the advent of primary percutaneous coronary intervention (PCI), the treatment of ST-segment elevation myocardial infarction (STEMI) has become substantially more effective.[1,2] Primary PCI is now the default treatment strategy for eligible STEMI patients.[1,2] Despite this advancement, STEMI patients remain at risk for both in-hospital and long-term adverse events.[3,4] Considerable efforts have been made to identify potential contributors to adverse outcomes in patients with acute myocardial infarction (MI). Clinical and laboratory parameters associated with unfavorable outcomes have been investigated, though many show inconsistent or controversial results.[5] Magnesium (Mg^{2+}) is the second most abundant intracellular cation and participates in over 325 enzymatic reactions, including lipid peroxidation, blood pressure regulation, and glucose metabolism.[5] Meta-analyses of prospective cohort studies indicate that Mg plays a critical role in cardiovascular, electrical, and metabolic homeostasis, with an inverse relationship observed between serum Mg levels and cardiovascular disease risk.[6,7] Mg also influences atherogenesis by modulating inflammation and oxidative processes.[6-8]

During the acute phase of MI, serum Mg levels have been reported to decrease transiently.[9] Some studies suggest that low serum Mg is associated with major adverse cardiac events (MACEs), in-hospital stent thrombosis, electrocardiographic no-reflow, and long-term mortality in acute MI patients.[10-12] Conversely, large-scale studies have found no association between low serum Mg levels and either in-hospital

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or long-term adverse events in MI patients.[13,14] Additionally, Naksuk et al.[14] demonstrated a potential adverse effect of higher serum Mg levels ≥2.4 mg/dL in a large cohort of acute MI patients. Given these conflicting findings, we conducted this study to evaluate the potential role of admission serum Mg levels in predicting in-hospital and long-term adverse events in STEMI patients undergoing primary PCI.

METHODS

Study Population

This retrospective study included 1,270 patients recruited between February 2011 and April 2015. All consecutive patients meeting the inclusion criteria were considered. The exclusion criteria were: missing admission Mg levels (n=28), history of end-stage renal disease (n=20), use of Mg supplements (n=5), active cancer (n=12), infection (n=7), and cirrhosis (n=5). Additionally, patients with STEMI who were treated medically (n=29) or underwent coronary artery bypass grafting [(CABG), n=45] were excluded. After applying these criteria, a total of 1,119 patients were included in the analysis. Eligible patients presented within 12 hours of symptom onset and were diagnosed with STEMI, for which primary PCI was performed. STEMI diagnosis was based on the criteria proposed by the European Society of Cardiology/American College of Cardiology Foundation/American Heart Association (ESC/ACCF/AHA) committee.[15] The study was approved by Bezmialem Vakıf University Ethics Committee (approval no: 15, date: 08.10.2025). As this was a retrospective study, written informed consent was not obtained from patients.

Data Collection

Patients' demographic data, medical history, and in-hospital information were obtained from digital medical records. After STEMI diagnosis in the emergency department, venous blood samples were collected from the antecubital vein prior to coronary angiography. Mg levels were categorized as <1.8, 1.8 to <2.0, 2.0 to <2.2, 2.2 to <2.4, and ≥2.4 mg/dL. Laboratory analysis of Mg was performed using a Roche/Hitachi Modular Analyzer (Roche Diagnostics International Ltd., Rotkreuz, Switzerland) with a xylidyl blue reaction. Mg levels are expressed in milligrams per deciliter (mg/dL). Following primary PCI, patients were admitted to the coronary care unit (CCU). Bedside transthoracic echocardiography was performed on the day of PCI. Left ventricular ejection fraction (LVEF) was determined using a modified Simpson's method. Patients' follow-up data were obtained through telephone interviews and/or review of hospital records.

Coronary Angiography and Medication

Coronary angiography and stenting were performed via the femoral approach using a 7-F guiding catheter. At the emergency department, all patients received a loading dose of 300 mg of acetylsalicylic acid and 600 mg of clopidogrel, regardless of prior medication. The procedure employed non-ionic, low-osmolality contrast media. The use of tirofiban was at the discretion of the operator in the catheterization laboratory. After the procedure, patients were transferred to the CCU, where angiotensin-converting enzyme inhibitors, beta-blockers, and statins were administered according to the ESC PCI guidelines.[16]

Definitions

STEMI was defined according to the criteria proposed by the ESC/ACCF/AHA committee.[15] Diabetes mellitus (DM) was defined as a prior diagnosis of DM or a fasting blood glucose level ≥126 mg/dL during hospitalization. Hypertension and hyperlipidemia were defined by a previous diagnosis and/or use of relevant medications. MACEs included cardiovascular death, stroke, and reinfarction. Cardiovascular death encompassed death from any cardiovascular cause, stroke, or sudden unexplained death. Target vessel revascularization (TVR) was defined as the need for coronary stenting or bypass surgery after the initial procedure. Acute stent thrombosis was diagnosed according to the definitions of the Academic Research Consortium.[16,17]

Statistical Analysis

Categorical data are expressed as numbers, and continuous data as mean±standard deviation. One-way ANOVA and chi-square tests were used for univariate comparisons of baseline characteristics among Mg groups. Variables that were statistically significant in bivariate correlations with Mg levels ($p<0.05$) or considered clinically important were included in the final multivariable linear regression model, which was conducted using a forward stepwise approach. A p value <0.05 was considered statistically significant. Data were analyzed using IBM SPSS Statistics version 20 software (IBM, Armonk, NY, USA). The predictive ability of variables was evaluated using receiver operating characteristic (ROC) curves, with the area under the curve (AUC) calculated for each variable.

RESULTS

The study included 1,119 patients with STEMI who were treated with primary PCI. Most patients were male (90%), and the mean age was 54 years. The overall mean follow-up period was 25±16 months. Baseline clinical characteristics of the patients are presented in Table 1. Groups were comparable in terms of age, creatinine, glomerular filtration rate, hypertension, hyperlipidemia, smoking, and family history of coronary artery disease. LVEFs following primary PCI were similar between groups (p=0.424). Admission serum glucose levels and the prevalence of type 2 diabetes mellitus (T2DM) differed significantly between groups (both p<0.001). Stent length and diameter were comparable across all groups (p=0.891 and p=0.691, respectively).

Table 2 summarizes the in-hospital and follow-up events. Clinical endpoints, including cardiogenic shock, cardiac death, reinfarction, TVR, MACE, stroke, cardiopulmonary resuscitation, ventricular arrhythmias, heart failure, inotropic usage, and atrial fibrillation (AF), were similar among groups, except for acute stent thrombosis (p=0.046). In-hospital analysis revealed a nominally significant difference in acute stent thrombosis across serum Mg groups (p=0.046); however, this association lost significance after correction for multiple comparisons. ROC curve analysis for predicting acute stent thrombosis is shown in Figure 1. The optimal admission serum Mg cut-off value was 1.83 mg/dL, with 76% sensitivity and 65% specificity [AUC: 0.781; 95% confidence interval (CI): 0.543–0.920; p=0.024]. Long-term follow-up clinical endpoints are presented in Table 3. Groups were similar in terms of cardiac death, late stent thrombosis,

Table 1. Baseline demographic and clinical characteristics

Variables	<1.8 (n=268)	1.8–2.0 (n=381)	2.0–2.2 (n=319)	>2.2 (n=151)	p value
Gender (female/male)	44 (16.4%)/224 (83.6%)	29 (7.6%)/352 (92.4%)	14 (4.4%)/305 (95.6%)	17 (11.3%)/134 (88.7%)	<0.001
Age (years)	53±9	53±9	53±9	55±10	0.387
Magnesium (mg/dL)	1.7±0.1	2±0.1	2.1±0.1	2.5±0.3	<0.001
Potassium (mmol/L)	4.1±0.52	4.06±0.48	4.06±0.47	4.16±0.65	0.223
Glucose (mg/dL)	168±80	157±77	144±61	155±74	0.001
Creatinine (mg/dL)	1±0.3	1.1±0.5	1.1±0.2	1.2±0.8	<0.001
Cholesterol (mg/dL)	187±40	193±44	191±40	193±44	0.381
Triglyceride (mg/dL)	142±101	156±145	162±101	154±84	0.278
LDL (mg/dL)	117±36	119±34	120±35	118±39	0.874
HDL (mg/dL)	41±8	42±10	40±9	41±9	0.181
CK-MB (μg/L)	224±166	241±174	218±179	220±176	0.334
Hematocrit (%)	40.9±3.9	41.6±4.2	41.7±3.6	42.1±4.8	0.055
Leukocyte (10^3/μL)	12.9±3.7	13.2±3.9	12.7±3.8	12.9±3.2	0.402
Platelet (10^3/μL)	262±65.1	256±58	263±72	271±76.2	0.142
Stent length (mm)	19±7	19±6	19±7	20±7	0.891
Stent diameter (mm)	3.1±0.4	3.1±0.3	3.1±0.3	3.1±0.4	0.691
Ejection fraction (%)	48±9	47±11	49±10	48±13	0.424
Glomerular filtration rate	93.9±22.8	90.9±22	91.4±21.9	88.9±24.3	0.066
Follow-up period (months)	25±15	24±16	22±16	21±14	0.081
Diabetes mellitus	90 (34.0%)/175 (66.0%)	75 (19.8%)/303 (80.2%)	61 (19.2%)/256 (80.8%)	31 (20.8%)/118 (79.2%)	<0.001
Hypertension	95 (38.3%)/153 (61.7%)	137 (38.7%)/217 (61.3%)	117 (38.5%)/187 (61.5%)	60 (42.0%)/83 (58.0%)	0.891
Family CAD history	54 (22.4%)/187 (77.6%)	61 (17.8%)/282 (82.2%)	58 (20.0%)/232 (80.0%)	29 (21.0%)/109 (79.0%)	0.569
Hyperlipidemia	113 (43.6%)/146 (56.4%)	133 (37.7%)/220 (62.3%)	123 (40.7%)/179 (59.3%)	55 (40.4%)/81 (59.6%)	0.528
Smoking	157 (65.4%)/83 (34.6%)	234 (68.0%)/110 (32.0%)	195 (66.1%)/100 (33.9%)	80 (60.6%)/52 (39.4%)	0.511

All data were expressed as mean±standard deviation
LDL: Low-density lipoproteins, HDL: High-density lipoproteins, CK-MB: Creatine kinase-MB, CAD: Coronary artery disease

Table 2. Angiographic/procedural and in-hospital cardiac findings

Findings	<1.8 (n=268)	1.8–2.0 (n=381)	2.0–2.2 (n=319)	>2.2 (n=151)	p value
In hospital					
Shock	7 (2.6%)	6 (1.6%)	11 (3.4%)	2 (1.3%)	0.324
Pre-TIMI flow 1–3	228 (85.4%)/23 (8.6%)/16 (6.0%)	339 (89.0%)/27 (7.1%)/15 (3.9%)	267 (84.2%)/35 (11.0%)/15 (4.7%)	128 (85.3%)/16 (10.7%)/6 (4.0%)	0.469
Post-TIMI flow 1–3	29 (11.2%)/13 (5.0%)/217 (83.8%)	37 (9.9%)/24 (6.5%)/311 (83.6%)	23 (7.3%)/16 (5.1%)/275 (87.6%)	11 (7.4%)/12 (8.1%)/126 (84.6%)	0.481
Tirofiban usage	113 (42.2%)	189 (49.6%)	153 (48.0%)	82 (54.3%)	0.121
Sudden death	8 (3.0%)	8 (2.1%)	3 (0.9%)	4 (2.6%)	0.336
Reinfarction	8 (3.0%)	5 (1.3%)	6 (1.9%)	2 (1.3%)	0.441
TVR	13 (4.9%)	13 (3.4%)	11 (3.4%)	8 (5.3%)	0.625
MACE	20 (7.5%)	20 (5.2%)	14 (4.4%)	11 (7.3%)	0.343
Stroke	1 (0.4%)	5 (1.3%)	1 (0.3%)	1 (0.7%)	0.378
CPR	9 (3.4%)	9 (2.4%)	8 (2.5%)	4 (2.6%)	0.883
VT/VF	16 (6.0%)	15 (3.9%)	11 (3.4%)	5 (3.3%)	0.408
Congestive heart failure	43 (16.0%)	46 (12.1%)	36 (11.3%)	16 (10.6%)	0.258
Inotrope agent	23 (8.6%)	24 (6.3%)	26 (8.2%)	8 (5.3%)	0.482

Table 2. Continued

Findings	<1.8 (n=268)	1.8–2.0 (n=381)	2.0–2.2 (n=319)	>2.2 (n=151)	p value
Atrial fibrillation	5 (1.9%)	6 (1.6%)	6 (1.9%)	4 (2.6%)	0.878
A-V blockage	6 (2.2%)	11 (2.9%)	7 (2.2%)	4 (2.6%)	0.931
Late pacemaker	6 (2.2%)	9 (2.4%)	11 (3.4%)	4 (2.6%)	0.784
Acute thrombosis (0–1 days)	5 (1.9%)	1 (0.3%)	2 (0.6%)	0 (0.0%)	0.046
Transfusion	5 (1.9%)	13 (3.4%)	8 (2.5%)	1 (0.7%)	0.269
In follow-up period					
Cardiac death	11 (4.1%)	18 (4.7%)	8 (2.5%)	2 (1.3%)	0.153
Late thrombosis (>30 days)	4 (1.5%)	6 (1.6%)	6 (1.9%)	2 (1.3%)	0.969
Re-infarction	21 (7.8%)	31 (8.1%)	21 (6.6%)	15 (9.9%)	0.578
TVR	51 (19.0%)	78 (20.5%)	49 (15.4%)	25 (16.6%)	0.165
MACE	63 (23.5%)	95 (24.9%)	58 (18.2%)	31 (20.5%)	0.056

All categorical data except pre (0.1.2) and post-TIMI (0.1.2) were expressed as X/Y that X means not exist while Y means exist
TVR: Target vessel revascularization, MACE: Major adverse cardiac events, VT-VF: Ventricular fibrillation ventricular tachycardia, CPR: Cardiopulmonary resuscitation
TIMI: Thrombolysis in myocardial infarction

Table 3. Long-term clinical outcomes

Variables	Unstandardized coefficients		Standardized coefficients	p value
	B	Std. error	Beta	
(Constant)	2.210	0.041	-	<0.001
Follow-up (months)	-0.003	0.001	-0.164	<0.001
GFR	-0.001	0.0001	-0.121	<0.001
DM	-0.066	0.023	-0.105	<0.001
Cardiac death	-0.143	0.056	-0.095	0.011
Acute (0-1 days) thrombosis	-0.235	0.109	-0.078	0.032

The regression model has 11.2 R square value with a significance at p<0.0001
DM: Diabetes mellitus, GFR: Glomerular filtration rate, Std: Standard

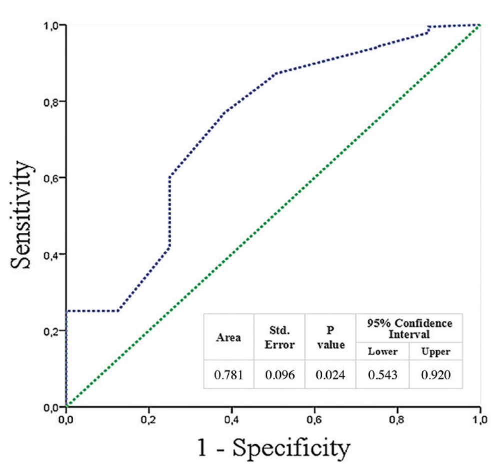

Figure 1. ROC curve of Mg^{+2} levels for acute stent thrombosis
ROC: Receiver operating characteristic, Std: Standard, Mg: Magnesium

reinfarction, TVR, and MACE. Stepwise linear regression analysis of admission serum Mg levels showed a significant relationship with acute stent thrombosis (p=0.032), as detailed in Table 3.

DISCUSSION

The present study demonstrated that low admission serum Mg levels were significantly associated with acute stent thrombosis. However, no significant association was found between admission serum Mg levels and in-hospital or long-term major adverse cardiovascular events, cardiovascular mortality, TVR, or stroke. It has been shown that during the acute phase of MI, transient hypomagnesemia occurs due to a shift from the extracellular to the intracellular space.[9] Therefore, serum Mg levels at the acute phase may not accurately reflect intracellular Mg concentrations. Mg functions correlate better with intracellular levels than with serum levels.[18] The amount of Mg within erythrocytes and lymphocytes may provide a more accurate reflection of intramyocardial Mg.[19] Mg, as an intracellular cation, plays various important roles in atherogenesis and cardiovascular diseases.[7] Under normal circumstances, Mg prolongs the refractory period of the atria and ventricles and stabilizes proarrhythmic substrates.[20] Mg also acts as a physiological calcium antagonist by binding to calcium sites and modifying membrane potential. Guo et al.[21] demonstrated that low Mg levels are associated with variant angina. As a result, Mg could limit coronary vasospasm and oxidative damage during MI and ischemia, which in turn may reduce infarct size.[22] Following cardiac surgery, $MgSO_4$ administration reduces the incidence of AF.[23,24] The Framingham Heart Study revealed that low Mg levels are associated with an increased risk of AF development in people without cardiovascular disease.[25] In the present study, we did not find any relationship between low serum Mg levels and the development of AF or ventricular arrhythmias. This could be due to a transient drop in serum Mg rather than an absolute decrease. The patient population in this study was different from those in the aforementioned studies.[23-25] We suggest that serum Mg levels should be evaluated differently in acute MI patients compared with the general population and patients undergoing CABG.

Mg improves glucose and insulin metabolism and reduces the risk of developing T2DM and metabolic syndrome.[26,27] Hypomagnesemia, through lipoprotein peroxidation, causes dyslipidemia, which decreases high-density lipoprotein levels and plasma apolipoprotein B, while increasing triglyceride-rich lipoproteins.[28] As a result of impaired glucose and lipid metabolism, hypomagnesemia plays an important role in cardiovascular health.[28] In the present study, we did not find any relationship between serum lipid levels and admission serum Mg levels. This may be due to a transient decrease in serum Mg rather than an absolute deficiency. On the other hand, the presence of DM was less frequent in patients with low serum Mg levels, consistent with previous reports. We suggest that because the low serum Mg levels were transient, no correlation was observed between DM and serum lipid levels. This finding may have occurred by chance.

Mg reduces platelet activation by inhibiting thromboxane A2 and increasing prostacyclin.[29,30] Thus, Mg plays an important role in platelet aggregation and adhesion. Additionally, Mg, acting as a calcium antagonist, exerts a platelet-inhibitory effect.[31] In a large-scale retrospective study, Çiçek et al.[10] showed that a serum Mg level <1.91 mg/dL significantly predicts acute stent thrombosis in STEMI patients treated with primary PCI. In a more recent, relatively small-scale study, patients with STEMI who underwent primary PCI were evaluated by Yuksel et al.,[11] who showed that a serum Mg level ≤1.87 mg/dL predicts electrocardiographic no-reflow and long-term mortality independently. In accordance with these studies, An et al.,[12] in a prospective study, revealed that a low level of serum Mg could be a predictor of MACEs in patients with acute MI treated with drug-eluting stents. In the present study, we detected that a serum Mg level <1.83 mg/dL was significantly associated with acute stent thrombosis. These results are consistent with the findings of Çiçek et al.[10] as both studies were performed in similar patient groups—namely, STEMI patients undergoing primary PCI. Because serum Mg levels may transiently decrease during the acute phase of STEMI, this reduction may alter platelet activity and increase platelet aggregation, thereby contributing to stent thrombosis. We also did not find any correlation between chronic stent thrombosis and admission serum Mg level. These findings suggest that, in the acute phase of STEMI, low serum Mg levels may be associated with increased platelet activity and stent thrombosis. However, although acute thrombosis appeared to differ between groups in the unadjusted analysis, this finding should be interpreted with caution because multiple outcome comparisons were performed. After adjustment for multiple testing, the association was attenuated, suggesting that the result may represent a chance finding rather than a robust signal. Coronary no-reflow is a complex condition with several proposed causes. One of the most important proposed causes of no-reflow is increased platelet reactivity. The results of Yuksel et al.[11] support this theory. The results of the aforementioned study showed that even though angiographic no-reflow (post-TIMI <3) was similar between groups, a low serum Mg level was associated with electrocardiographic no-reflow. In the present study, similarly, we did not find any significant correlation between serum Mg level and post-TIMI flow. We did not evaluate electrocardiographic no-reflow.

On the other hand, studies have demonstrated contrary results. In a moderate-scale study, Vassalle et al.[13] did not find any significant relationship between low serum Mg level and adverse cardiac events (non-fatal MI and all-cause mortality) in acute MI patients. In concordance with this study, a large retrospective study conducted at Mayo Clinic Hospital in patients admitted to the intensive cardiac care unit—mostly composed of acute MI patients—did not reveal any association between serum Mg levels and sudden cardiac death or corrected QT interval.[14] They also found that both admission and post-admission serum Mg level ≥2.4 mg/dL were associated with increased hospital mortality.[14] In a similar context, the Mg on Coronaries Trial Investigators did not find any beneficial effect of early Mg administration on 30-day mortality in high-risk STEMI patients.[32] In our study, No significant association was observed between admission serum Mg level and in-hospital or long-term mortality, reinfarction, TVR, or MACEs. These results are consistent with the aforementioned studies.[13,14,32] These endpoints could be affected by absolute Mg levels rather than a transient decrease in serum Mg.

These findings suggest that the serum Mg level during the acute phase of MI and the Mg level in the general population without cardiovascular disease should be evaluated separately. In this perspective, serum Mg levels in the acute phase of MI are probably transient and may be associated with a temporary increase in platelet reactivity and acute stent thrombosis. Therefore, after the acute phase of MI, serum Mg levels should be re-checked and re-evaluated, with special attention paid to the acute phase of STEMI.

Study Limitations

The present study had several limitations. First, this was a retrospective study and therefore had the inherent limitations of such a design. The causative relationship of all confounders cannot be confirmed. Second, we did not measure serial serum Mg levels; therefore, we could not determine subsequent Mg levels or their effects on outcomes. Third, the study was conducted at a single center, which limits the generalizability of the results. Stent thrombosis may result from multiple factors, and in the present study, we did not evaluate all potential confounders. Finally, electrocardiographic no-reflow was not assessed. Although ROC analysis was performed, the wide CI (AUC 0.781; 95% CI: 0.543–0.920) indicates limited precision of the findings. In addition to the retrospective design and the lack of serial Mg measurements, several other methodological limitations should be acknowledged. No a priori power analysis was performed, which may limit the study’s ability to reliably detect clinically meaningful differences. Furthermore, the statistical modeling strategy was not fully aligned with the nature of the outcomes, as binary endpoints were not evaluated using multivariable logistic regression analysis. This may have limited adequate adjustment for potential confounding factors. Finally, no correction for multiple comparisons was applied despite the evaluation of numerous endpoints, increasing the risk of type I error.

CONCLUSION

In conclusion, this large-scale retrospective study demonstrated that low admission serum Mg levels were significantly associated with stent thrombosis. However, no significant association was observed between serum Mg levels and in-hospital or long-term cardiovascular mortality, stroke, reinfarction, TVR, or major adverse cardiovascular events. When the findings of the present study are considered alongside those of previous reports, it is evident that randomized clinical trials are needed to draw more robust and definitive conclusions.

Ethics Committee Approval: The study was approved by Bezmialem Vakıf University Ethics Committee (approval no: 15, date: 08.10.2025).

Informed Consent: As this was a retrospective study, written informed consent was not obtained from patients.

Authorship Contributions: Concept: C.A., M.K., Design: C.A., M.K., Data Collection or Processing: B.H.Ş.U., Analysis or Interpretation: C.A., S.U., Literature Search: M.U., Writing: C.A., E.D.

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