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*Original Article*

# Serum magnesium levels as a predictor of coronary artery stenosis and their association with oxidative stress biomarkers

Hasidaer Midilibieke[1*], Ying Gao[1*], Juledezi Hailati[1], Yunchun Yang[1], Lei Zhang[1], Madina Mahesutihan[1], Jiao Wang[1], Meijuan Zheng[1], Muhuyati[2], Zhiqiang Liu[1]

[1]*Department of General Cardiology, The First Affiliated Hospital of Xinjiang Medical University, Urumqi, Xinjiang Uygur Autonomous Region, China;* [2]*Xinjiang Medical University, Urumqi, Xinjiang Uygur Autonomous Region, China.* [*]*Equal contributors and co-first authors.*

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**Abstract:** Background: To investigate the association between serum magnesium (Mg) levels and the presence of coronary artery stenosis, as well as their correlation with oxidative stress biomarkers malondialdehyde (MDA) and superoxide dismutase (SOD). Methods: In this retrospective study, a total of 42 patients diagnosed with coronary artery stenosis at the First Affiliated Hospital of Xinjiang Medical University between January 2022 and October 2024 were included as the coronary artery experimental group, while another 42 cases with no significant stenosis confirmed by coronary angiography during the same period served as the control group. Demographic and clinical characteristics, as well as serum Mg levels and oxidative stress markers were compared between the two groups. The correlations between serum Mg levels and both coronary artery stenosis and oxidative stress factors (MDA and SOD) were analyzed using the Spearman rank correlation coefficient. Receiver operating characteristic (ROC) curve was used to analyze the diagnostic performance of serum Mg levels on coronary artery stenosis. Results: There were no significant differences in the baseline demographic characteristics between the two groups (all P>0.05), including age, gender distribution, body mass index (BMI), educational background, smoking and alcohol consumption history, systolic blood pressure (SBP), diastolic blood pressure (DBP), DM course, fasting plasma glucose (FPG), glycated hemoglobin (GHb), triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), creatinine (Cr), and blood urea nitrogen (BUN). The stenosis group had significantly lower serum Mg levels (1.57±0.37 vs. 2.02±0.33 mmol/L, P<0.001) and higher SOD (86.11±21.59 vs. 59.02±17.36 U/mL, P<0.001), while MDA was elevated in controls (24.52±8.33 vs. 18.82±6.37 nmol/mL, P=0.001). The ROC analysis of serum Mg levels for predicting coronary artery stenosis yielded an area under the curve (AUC) of 0.825 (95% CI: 0.757-0.923), with an optimal cutoff value of 1.88 mmol/L (standard error: 0.3265), achieving a sensitivity of 80% and specificity of 75%. Spearman correlation analysis demonstrated a negative association between Mg and MDA (r=-0.506, P=0.041) and a positive association with SOD (r=0.288, P=0.008) in patients with coronary stenosis. Conclusions: Lower serum magnesium levels are significantly associated with an increased risk of coronary artery stenosis. Moreover, reduced serum Mg correlates with elevated MDA levels and decreased SOD activity, indicating enhanced oxidative stress.

**Keywords:** Magnesium, angina pectoris, coronary artery stenosis, coronary heart disease

## Introduction

In recent years, the importance of magnesium ions in cardiovascular physiology and pathology has attracted increasing attention [1]. Clinical studies have demonstrated a negative correlation between serum magnesium (Mg) levels and the severity of coronary artery stenosis; individuals with lower magnesium concentrations are at higher risk of both the occurrence and severity of stenosis [2-4]. Hypomagnesemia may contribute to coronary artery stenosis by impairing vascular endothelial function, promoting vascular smooth muscle cell proliferation and migration, and disrupting cellular ion homeostasis [1, 5]. For example, reduced Mg levels may affect the release of nitric oxide (NO), an important vasodilator pro-

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duced by vascular endothelial cells, leading to impaired vasodilation and subsequently promoting the development of coronary artery stenosis [6, 7].

Evidence from oxidative stress studies further indicates that oxidative stress plays a key role in the development and progression of coronary artery stenosis. Increased oxidative stress leads to excessive production of reactive oxygen species (ROS), resulting in lipid peroxidation, protein oxidation, and DNA damage. These processes trigger vascular inflammation and apoptosis, promote the formation and instability of atherosclerotic plaques, and ultimately exacerbate coronary artery stenosis [8-11]. Simultaneously, the body's antioxidant system, including enzymes such as superoxide dismutase (SOD), and glutathione peroxidase (GSH-Px), plays an important role in resisting oxidative stress. When antioxidant capacity declines, oxidative damage is further exacerbated.

Further research has shown an intrinsic link between Mg ions and oxidative stress [12, 13]. Some experimental evidence suggests that Mg ions are able to affect intracellular redox homeostasis, possibly by modulating the activity of antioxidant enzymes or directly participating in the scavenging of ROS. However, the precise mechanisms underlying this relationship remain incompletely understood [14, 15].

Given the current research status, further investigations are warranted. At the basic research level, it is necessary to elucidate the specific molecular mechanisms by which magnesium regulates oxidative stress, including its effects on key antioxidant enzymes and oxidative stress-related signaling pathways, as well as its interactions with other ions in cellular redox regulation. Clinically, long-term prospective studies are needed to clarify the effects of magnesium supplementation on oxidative stress and the progression of coronary artery stenosis, and to evaluate its therapeutic efficacy and safety [16]. In addition, individual differences, such as genders, ages, genetic backgrounds, and comorbidities, should be considered to better guide clinical treatment and provide more targeted intervention strategies for the prevention and treatment of cardiovascular diseases.

Building on the existing evidence, these research directions may help to clarify the complex relationships among Mg, coronary artery stenosis, and oxidative stress, and open new avenues for improving the diagnosis and treatment of coronary artery diseases. In this study, 42 patients diagnosed with coronary artery stenosis due to angina pectoris and 42 patients with no significant stenosis confirmed by coronary angiography in the same period were included to explore the correlation between serum Mg levels and coronary artery stenosis. For the first time, the predictive value of serum Mg levels for coronary artery stenosis was evaluated using the receiver operating characteristic (ROC) curve analysis. Furthermore, the bidirectional relationships between serum Mg level and oxidative stress markers were investigated, offering a novel perspective for clinical diagnosis.

## Materials and methods

### *Case selection*

This retrospective observational study included 42 patients diagnosed with coronary artery stenosis due to angina pectoris who were admitted to the First Affiliated Hospital of Xinjiang Medical University between January 2022 and October 2024, comprising the stenosis group. A control group of 42 patients with no significant coronary artery stenosis, confirmed by coronary angiography (CAG) during the same period, were included as the control group.

In the stenosis group, there were 27 males and 15 females, aged 18-75 years, with an average age of (61.54±9.81) years. In the control group, there were 24 males and 18 females, aged 18-75 years, with an average age of (64.51±8.75) years. There were no significant differences in baseline demographic data between the two groups ($P>0.05$), indicating comparability. All procedures involving human participants were conducted in accordance with the Declaration of Helsinki (2013) and were approved by the Ethics Committee of The First Affiliated Hospital of Xinjiang Medical University.

### *Inclusion criteria*

*Inclusion criteria for stenosis group:* (1) Coronary artery stenosis confirmed by CAG,

defined as ≥50% luminal narrowing in at least one major coronary artery (left main, left anterior descending, left circumflex, or right coronary artery) according to the American Heart Association classification. (2) Age 18-75 years old, regardless of gender. (3) Hospitalized for angina pectoris or suspected coronary heart disease.

*Inclusion criteria for control group:* (1) No significant coronary artery stenosis confirmed by CAG, defined as <50% luminal narrowing in all coronary arteries. (2) Age 18-75 years old, matched to the stenosis group by age (±5 years) and gender. (3) Underwent CAG for clinical indications (e.g., chest pain, abnormal electrocardiogram) but showed no significant stenosis.

*Exclusion criteria*

(1) Active malignancies (e.g., hematological malignancies or solid tumors) diagnosed within the past 5 years. (2) Severe immune system disorders, including AIDS, active systemic lupus erythematosus (SLE) (Disease Activity Index ≥10), or congenital immunodeficiency. (3) Chronic kidney disease (CKD) stage 3 or higher (estimated glomerular filtration rate [eGFR] <60 mL/min/1.73 $m^2$, calculated by the CKD-EPI formula); Chronic liver disease, including cirrhosis confirmed by imaging or liver biopsy, or acute hepatitis (positive hepatitis B/C virus replication or alanine transaminase >5× upper limit of normal). (4) Severe hematological disorders (hemoglobin <90 g/L in males, <80 g/L in females) or thrombocytopenia (<100×$10^9$/L). (5) Endocrine disorders affecting magnesium metabolism, including primary hyperparathyroidism, thyrotoxicosis (serum thyroxine >14 μg/dL), or type 1 diabetes mellitus with a course of >10 years and glycated hemoglobin >10%. (6) Pregnancy or lactation. (7) History of organ transplantation due to immunosuppressive therapy potentially affecting oxidative stress markers. (8) Incomplete clinical data, including coronary angiography reports, key laboratory data (e.g., serum Mg level, malondialdehyde (MDA), or SOD). (9) Use of magnesium supplements or medications affecting magnesium metabolism within 4 weeks prior to blood sampling (e.g., intravenous magnesium sulfate, oral magnesium oxide, loop diuretics [furosemide], or proton pump inhibitors [omeprazole]).

*Coronary stenosis severity and lower serum magnesium levels definition*

The severity of coronary artery stenosis was determined angiographically and categorized based on the percentage reduction in luminal diameter relative to the adjacent normal reference segment, consistent with established clinical and angiographic standards [17]. Stenosis was classified into the following groups: Mild: <50% diameter narrowing (non-significant stenosis). Moderate: 50-70% diameter narrowing. Severe: >70% diameter narrowing (hemodynamically significant stenosis).

Stenoses ≥50% were collectively considered obstructive coronary artery disease. This classification defines the stenosis severity groups used for subsequent analyses. Lower serum magnesium levels are inversely associated with the severity of coronary artery stenosis. Mechanistically, magnesium deficiency promotes endothelial dysfunction, vascular inflammation, and accelerated atherosclerosis progression, culminating in significant luminal narrowing. Clinically, patients with severe stenosis (>70% luminal occlusion) exhibit significantly lower Mg levels compared to those with mild-to-moderate disease, independent of traditional cardiovascular risk factors [5, 18, 19].

*Data collection*

Baseline data collected for both groups included sex, age, body mass index (BMI), education level, smoking and alcohol consumption history, systolic blood pressure (SBP), diastolic blood pressure (DBP), FPG, GHb, TG, TC, LDL-C, HDL-C, Cr, BUN levels and comorbidities (e.g., hypertension, diabetes mellitus).

Fasting venous blood samples were collected in the morning. Following centrifugation, the supernatant was isolated for biochemical analyses: (1) Serum Mg level detection: Serum magnesium concentration was measured using the methyl thymol blue colorimetric method on an ADVIA RCentaur XPT automated chemiluminescence analyzer (Siemens Healthineers, Germany), with manufacturer provided reagents. (2) Oxidative stress markers: Serum MDA levels were assayed by the thiobarbituric acid reactive substances (TBARS) method using a commercial kit (Nanjing Jiancheng Bioengineering Institute, China; catalog num-

**Table 1.** Comparison of baseline data between the two groups

| | Stenosis group (n=42) | Control group (n=42) | $t/\chi^2$ | P |
|---|---|---|---|---|
| Age | 61.54±9.81 | 64.51±8.75 | 1.466 | 0.146 |
| <60 | 19 | 17 | 0.194 | 0.413 |
| ≥60 | 23 | 25 | | |
| BMI | 23.49±3.23 | 23.06±3.29 | -0.602 | 0.549 |
| SBP | 131.27±14.88 | 136.22±15.4 | 1.498 | 0.138 |
| DBP | 82.21±11.91 | 83.17±11.19 | 0.380 | 0.705 |
| DM_COURSE | 10.78±4.97 | 11.01±6.77 | 2.492 | 0.115 |
| FPG | 8.19±3.22 | 8.94±3.68 | 0.994 | 0.323 |
| GHb | 9.48±2.17 | 10.26±2.16 | 1.638 | 0.105 |
| TG | 2.14±1.14 | 1.97±1.17 | -0.657 | 0.513 |
| TC | 4.01±1.10 | 3.89±1.00 | -0.490 | 0.625 |
| LDL_C | 2.17±0.80 | 2.01±0.63 | -1.053 | 0.296 |
| HDL_C | 1.24±0.35 | 1.16±0.39 | -1.003 | 0.319 |
| Cr | 68.62±15.91 | 74.77±25.37 | 1.331 | 0.187 |
| BUN | 5.94±1.47 | 6.09±1.62 | 0.440 | 0.661 |
| Gender | | | | |
| Male | 27 | 24 | 0.449 | 0.328 |
| Female | 15 | 18 | | |
| Hypertension | | | | |
| Yes | 15 | 22 | 2.367 | 0.093 |
| No | 27 | 20 | | |
| Smoking | | | | |
| Yes | 20 | 23 | 0.429 | 0.331 |
| No | 22 | 19 | | |
| DM | | | | |
| Yes | 22 | 25 | 0.435 | 0.330 |
| No | 20 | 17 | | |
| EDU | | | | |
| Under Bachelor | 24 | 23 | 0.048 | 0.500 |
| Bachelor and above | 18 | 19 | | |

Note: SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; DM, Diabetes Mellitus; FPG, Fasting Plasma Glucose; GHb, Glycated Hemoglobin; TG, Triglycerides; TC, Total Cholesterol; LDL_C, Low-Density Lipoprotein Cholesterol; HDL_C, High-Density Lipoprotein Cholesterol; Cr, Creatinine; BUN, Blood Urea Nitrogen; DM_COURSE, Diabetes Mellitus COURSE; EDU, education.

ber: A003-1). Serum SOD activity was measured by the xanthine oxidase method using a corresponding kit from the same supplier (Nanjing Jiancheng Bioengineering Institute, China; catalog number: A001-3). All procedures followed the manufacturers' instructions for reagent preparation and operation.

*Statistical analysis*

All statistical analyses were conducted using SPSS 26.0 (Chicago, IL, USA). For normally distributed data, group comparisons were conducted using the independent samples t-test for two groups, with post-hoc Tukey's test performed when significant differences were detected. Non-normally distributed continuous variables were analyzed using non-parametric tests: the Mann-Whitney U-test for two-group comparisons. For categorical data, comparisons between groups were performed using the chi-square test, with Fisher's exact test applied where expected cell counts were <5. Multivariate analysis was used to identify independent risk factors for coronary artery stenosis. The correlation between serum Mg levels and the occurrence of coronary artery stenosis as well as the MDA and SOD levels was further analyzed using Spearman rank correlation coefficient test. ROC curve analysis was performed to evaluate the predictive value of serum Mg levels for coronary artery stenosis. A *P*-value <0.05 was considered statistically significant.

## Results

*Comparison of baseline data between the two groups*

No significant differences were observed between the two groups in terms of age, sex, BMI, SBP, DBP, DM course, FPG, GHb, TG, TC, LDL-C, HDL-C, Cr, or BUN levels (all P>0.05) (**Table 1**).

*Comparison of Mg, MDA and SOD levels between the two groups*

The stenosis group exhibited significantly lower serum Mg levels compared to the control group (t=-5.997, P<0.001). Conversely, MDA level, an oxidative stress marker, was higher in the control group than in the stenosis group (t=3.521,

**Table 2.** Comparison of Mg, MDA, and SOD levels between the two groups

| | Stenosis group (n=42) | Control group (n=42) | t | P |
|---|---|---|---|---|
| Mg | 1.57±0.37 | 2.02±0.33 | -5.997 | 0.000 |
| MDA | 24.52±8.33 | 18.82±6.37 | 3.521 | 0.001 |
| SOD | 59.02±17.36 | 86.11±21.59 | -6.334 | 0.000 |

Note: Mg, Magnesium; MDA, Malondialdehyde; SOD, Superoxide Dismutase.

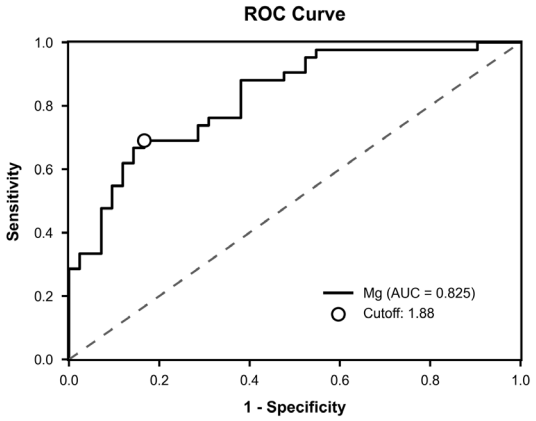


**Figure 1.** ROC curve for serum magnesium level in predicting coronary artery stenosis.

P=0.001). SOD, an antioxidant enzyme, showed a significantly higher level in the stenosis group compared to the control group (t= -6.334, P<0.001) (**Table 2**).

*Predictive value of serum Mg levels for coronary artery stenosis*

ROC curve analysis demonstrated that serum Mg levels had a high discriminative ability for predicting coronary artery stenosis, with an area under the curve (AUC) of 0.825 (95% CI: 0.757-0.923). The optimal cutoff value was 1.88 mmol/L (standard error: 0.3265), with a sensitivity of 80% and specificity of 75% (**Figure 1**).

*Correlation between serum Mg levels and coronary artery stenosis*

Spearman correlation analysis revealed a significant negative correlation between serum Mg levels and the severity of coronary artery stenosis (r=-0.506, P<0.01) (**Table 3**).

**Table 3.** Spearman correlation analysis of serum magnesium level with stenosis severity and oxidative stress factors MDA, and SOD

| | Mg | |
|---|---|---|
| | R | P |
| Stenosis severity | -0.506 | 0.001 |
| MDA | -0.224 | 0.041 |
| SOD | 0.288 | 0.008 |

Note: Mg, Magnesium; MDA, Malondialdehyde; SOD, Superoxide Dismutase.

*Correlation between serum Mg levels and oxidative stress markers*

Spearman correlation analysis revealed that serum Mg levels were negatively associated with MDA (r=-0.224, P=0.041) but positively associated with SOD (r=0.288, P=0.008), indicating that lower magnesium levels were associated with higher oxidative stress, while higher magnesium levels were associated with enhanced antioxidant capacity (**Table 3**).

## Discussion

The clinical manifestations of coronary artery stenosis are heterogeneous and depend on the location and severity of the stenosis, as well as the development of collateral circulation, making the evaluation of treatment outcomes and prognosis challenging. Serum magnesium plays an essential role in maintaining cellular function and regulating vascular tone and blood pressure [20, 21]. Magnesium deficiency exacerbates endothelial dysfunction by increasing vascular permeability and impairing vasodilation, thereby accelerating the progression of coronary stenosis through oxidative stress pathways [22, 23]. In this context, this study investigated the correlation between serum magnesium levels and coronary artery stenosis. The findings highlight the potential of serum magnesium as a prognostic biomarker for coronary artery stenosis, providing valuable insights into disease evaluation and management.

The results of this study showed that the proportions of patients aged over 60 years, and those with hypertension, a history of smoking and alcohol consumption, or DM were significantly higher in the coronary artery stenosis

group compared to the control group. Additionally, serum magnesium levels were significantly lower in the stenosis group, suggesting that age, hypertension, smoking and alcohol consumption, DM, and serum Mg levels are important factors influencing the development of coronary artery stenosis. Older individuals have a higher risk of coronary atherosclerosis. Aging is associated with vascular dysfunction, characterized by increased vascular stiffness and elevated inflammatory responses, which contribute to the progression of coronary artery stenosis [24, 25]. Hypertension can induce vascular endothelial damage, reduce SOD synthesis, and impair endothelium-dependent vasodilation, thereby promoting the development of coronary artery stenosis [26]. Smoking introduces large amounts of nicotine, which triggers inflammatory responses and oxidative stress, leading to endothelial cell damage and dysfunction, ultimately increasing the risk of coronary artery stenosis [27]. Alcohol consumption affects prostacyclin levels, enhances hyperreactivity of platelets, and its metabolite acetaldehyde reduces guanylate cyclase activity, all of which facilitate the pathogenesis of coronary artery stenosis [28-30]. Diabetes mellitus, characterized by abnormal glucose and lipid metabolism, contributes to atherosclerosis and endothelial dysfunction. Furthermore, abnormal lipid metabolism aggravates oxidative stress, making diabetes mellitus an independent risk factor for coronary artery stenosis [31, 32].

Magnesium plays a regulatory role in intracellular free calcium concentrations, which is closely related to vascular contractility. Previous studies have confirmed that magnesium deficiency can cause myocardial ischemia and coronary heart disease and promote the development of atherosclerosis. Khatun et al. [24] demonstrated that magnesium enhances prostacyclin synthesis in vascular endothelial cells, thereby modulating vasospasm, which is consistent with the results of this study. The ROC curve provides a graphical representation of sensitivity and specificity across various thresholds, avoiding reliance on a fixed cutoff and accommodating intermediate diagnostic states. This facilitates clinical decision making by allowing physicians to balance the risks of false positives and false negatives based on clinical context. In this study, ROC analysis showed that serum Mg levels had an AUC of 0.825 for predicting coronary artery stenosis, indicating good discriminative performance.

This study adds to the growing body of evidence supporting Mg as a potential biomarker for coronary artery stenosis, demonstrating robust discriminative capacity (AUC=0.825), compared to conventional electrolytes such as sodium and potassium (AUC<0.65), and novel inflammatory marker (e.g., GlycA) (AUC=0.74) [18, 33]. Multicenter studies in CKD populations have also validated the predictive value of Mg for clinical outcomes, further supporting its pathophysiological relevance [34, 35].

This study has several limitations. First, the small sample size (n=84) may limit the statistical power and generalizability of the findings. Second, the retrospective design introduces potential selection bias. Third, the lack of longitudinal follow-up precludes the evaluation of temporal associations between magnesium levels and disease progression. Additionally, direct comparisons with emerging biomarkers such as fibroblast growth factor-23 were not performed. Future research should incorporate Mg into multivariate models and investigate its mechanistic role in the pathogenesis of coronary artery stenosis, as highlighted in recent systematic reviews.

To address the current limitations and enhance translational relevance, future research should prioritize large-scale, multicenter cohort studies to improve the generalizability of findings. The development of multivariate predictive models that integrate novel biomarkers and clinical variables is warranted. Mechanistic investigations, including both *in vitro* and *in vivo* studies, are essential to delineate the biological pathways through which magnesium modulates coronary pathophysiology, bridging associative findings with causal mechanisms. Furthermore, the incorporation of magnesium levels into comprehensive clinical frameworks alongside established diagnostic and prognostic indicators, could facilitate precision medicine approaches for risk stratification and therapeutic optimization in coronary artery disease. These concerted efforts will not only refine predictive accuracy but also deepen the mechanistic understanding of magnesium's role in vascular pathology.

In summary, serum magnesium levels are significantly associated with the occurrence of coronary artery stenosis. Changes in serum magnesium concentrations may serve as a valuable predictor for the development of coronary artery stenosis, providing novel insights for clinical diagnosis and risk assessment.

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## Disclosure of conflict of interest

None.

**Address correspondence to:** Muhuyati, Xinjiang Medical University, Urumqi, Xinjiang Uygur Autonomous Region, China. Tel: +86-0991-2110666; E-mail: hasidaer@163.com; Zhiqiang Liu, Department of General Cardiology, The First Affiliated Hospital of Xinjiang Medical University, Urumqi, Xinjiang Uygur Autonomous Region, China. Tel: +86-0991-4364780; E-mail: liuzhiqiang1320132@163.com

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