

Serum Magnesium Levels and the Risk of Arrhythmias in Acute Myocardial Infarction

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Received: 05-08-2026 / Revised: 02-09-2026 / Accepted: 03-10-2026

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Conflict of interest: Nil

Abstract:

Background: Magnesium plays an important role in myocardial electrical stability, and reduced serum magnesium may predispose patients with acute myocardial infarction (AMI) to arrhythmias. This study evaluated the association between serum magnesium levels and arrhythmias in patients with AMI.

Methods: A hospital-based observational study was conducted among 112 patients with AMI over one year. Patients presenting within 12 hours of symptom onset underwent clinical evaluation, ECG, routine investigations, and echocardiography. Serum magnesium was measured on Days 1 and 5. Patients were monitored for arrhythmias during hospitalization.

Results: The mean serum magnesium level increased from 1.88 ± 0.26 mg/dL on Day 1 to 1.98 ± 0.47 mg/dL on Day 5. Arrhythmias occurred in 63 (56.3%) patients. Magnesium levels were significantly lower in patients with arrhythmias than in those without on Day 1 (1.70 ± 0.16 vs. 2.10 ± 0.19 mg/dL) and Day 5 (1.76 ± 0.51 vs. 2.26 ± 0.20 mg/dL; $p < 0.001$).

Conclusion: Lower serum magnesium was significantly associated with arrhythmias in AMI.

Keywords: Acute myocardial infarction, Magnesium, Arrhythmia, Hypomagnesemia, Ventricular arrhythmia.

DOI: 10.25258/ijcpr.18.10.19

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Introduction

Minerals are essential for normal cellular growth, physiological function, and maintenance of fluid and electrolyte balance. In the human body, mineral homeostasis is regulated through complex interactions involving hemodynamic, electrolyte, and other physiological mechanisms [1, 2]. Although the importance of macronutrients and vitamins has long been recognized, increasing evidence has highlighted the critical role of minerals, including trace elements, in maintaining normal biological functions [3, 4].

Magnesium is an important intracellular cation that contributes to numerous physiological processes, including myocardial contractility, membrane stability, electrolyte homeostasis, and cardiac conduction. It has therefore been implicated in the pathophysiology of cardiovascular diseases, particularly acute myocardial infarction (AMI) [5, 6]. Adequate magnesium availability is considered important for maintaining the functional and electrical integrity of the myocardium, and disturbances in magnesium homeostasis may increase cardiac electrical instability and predispose patients to arrhythmias [7, 8].

Previous investigations have reported alterations in serum magnesium concentrations following AMI, with levels tending to decrease during the initial 48 hours and gradually returning toward normal over the subsequent weeks. Reduced myocardial magnesium concentrations have also been observed following myocardial infarction and have been associated with the development of complications, particularly cardiac arrhythmias. Magnesium deficiency may promote myocardial electrical instability and has been suggested as a contributing factor in ventricular fibrillation and sudden cardiac death among patients with ischemic heart disease. Additionally, magnesium deficiency has been associated with coronary vasospasm and metabolic abnormalities that may further contribute to cardiovascular risk [9-11].

Acute myocardial infarction remains a major cause of morbidity and mortality, with its prognosis influenced by several factors, including the occurrence of potentially life-threatening arrhythmias. Despite advances in the management of AMI, the mechanisms underlying arrhythmic complications are not completely understood. Since

magnesium plays an important role in myocardial electrical stability, alterations in its serum concentration may be associated with the development of arrhythmias following AMI. Therefore, the present study was undertaken to assess the association between serum magnesium levels and arrhythmias in patients with acute myocardial infarction.

Materials and Methods

Study Design and Study Population: A hospital-based observational study was conducted over a period of one year and included 112 patients diagnosed with acute myocardial infarction (AMI). Patients presenting to the hospital within 12 hours of onset of symptoms were considered for inclusion. The diagnosis of AMI was established based on a compatible history of chest discomfort, characteristic electrocardiographic (ECG) findings, and elevated cardiac enzyme levels.

Clinical Assessment and Investigations: All enrolled patients underwent detailed clinical evaluation, including history-taking and comprehensive physical examination. Baseline demographic characteristics and cardiovascular risk factors, including smoking, family history, obesity, hypertension, diabetes mellitus, and dyslipidaemia, were recorded. Routine laboratory investigations included haemoglobin, complete blood count, urine examination, blood glucose, blood urea, serum creatinine, serum electrolytes, fasting lipid profile, and cardiac enzyme estimation. Echocardiography was performed in all patients.

Electrocardiographic Assessment and Arrhythmia Monitoring: A 12-lead ECG was performed to confirm the diagnosis and classify the infarction according to the anatomical location, including anterior wall, inferior wall, and anteroseptal myocardial infarction. Patients were monitored during their hospital stay for the

development of cardiac arrhythmias. Arrhythmic events, particularly ventricular premature contractions (VPCs) and ventricular tachycardia, were documented during the five-day observation period. Patients were subsequently categorized into groups with and without arrhythmias.

Serum Magnesium Estimation: Serum magnesium concentrations were assessed at two predefined time points, Day 1 and Day 5 of hospitalization. The magnesium values were compared between patients who developed arrhythmias and those who remained free of arrhythmic complications during the observation period.

Statistical Analysis: Continuous variables were expressed as mean $\pm$ standard deviation (SD), while categorical variables were presented as frequencies and percentages. Serum magnesium levels were compared between patients with and without arrhythmias at Days 1 and 5. A p-value <0.05 was considered statistically significant. The association between serum magnesium levels and the occurrence of arrhythmias was evaluated using appropriate statistical tests.

Results

A total of 112 patients with acute myocardial infarction were included in the study. The majority of participants were aged 50 years and above, followed by those in the 40–49-year age group. Males constituted a greater proportion of the study population than females, with an approximate male-to-female ratio of 2:1.

Among the evaluated cardiovascular risk factors, hypertension was the most frequently observed, followed by dyslipidaemia, diabetes mellitus, family history, and obesity. Smoking was reported in approximately two-fifths of the study population (Table 1).

Table 1: Demographic Characteristics and Risk Factors of the Study Participants

Characteristic	No. of Patients	Percentage (%)
Age group (years)		
20–29	2	1.8
30–39	18	16.1
40–49	40	35.7
≥ 50	52	46.4
Sex		
Male	74	66.1
Female	38	33.9
Risk factors		
Smoking	45	40.2
Family history	67	59.8
Obesity	63	56.3
Hypertension	76	67.9
Diabetes mellitus	67	59.8
Dyslipidaemia	72	64.3

Regarding the type of myocardial infarction, anterior wall MI was the most frequently observed ECG pattern, followed by inferior wall MI and anteroseptal MI. Of the 112 patients, approximately 63 patients (56.3%) developed arrhythmias during

the five-day observation period. The recorded arrhythmias consisted mainly of ventricular premature contractions (VPCs) and ventricular tachycardia (Table 2).

Table 2: Clinical and ECG Characteristics of Acute Myocardial Infarction

Parameter	No. of Patients	Percentage (%)
ECG diagnosis		
Anterior wall MI	49	43.8
Anteroseptal MI	16	14.3
Inferior wall MI	47	42.0
Arrhythmia during hospital stay		
Present	63	56.3
Absent	49	43.8

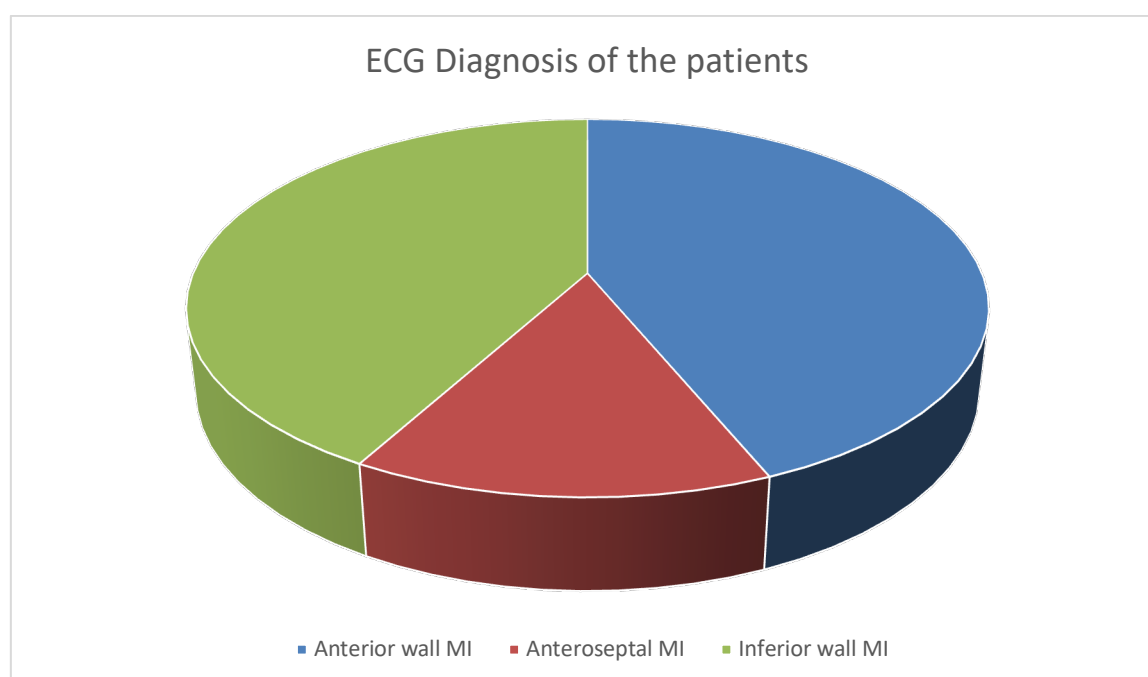


Figure 1: Clinical and ECG Characteristics of Acute Myocardial Infarction

The mean serum magnesium concentration on Day 1 was lower among patients who subsequently developed arrhythmias compared with those who remained free of arrhythmic complications (1.70 ± 0.16 vs. 2.10 ± 0.19). A similar difference was observed on Day 5, with lower magnesium levels among patients with arrhythmias (1.76 ± 0.51 vs.

2.26 ± 0.20). The differences between the two groups were statistically significant ($p < 0.001$), indicating an association between lower serum magnesium concentrations and the occurrence of arrhythmias following acute myocardial infarction (Table 3).

Table 3: Association of Serum Magnesium Levels with Arrhythmias

Time point	Arrhythmia status	No. of Patients	Mean Serum Magnesium $\pm$ SD	p-value
Day 1	With arrhythmias	63	1.70 ± 0.16	<0.001
	Without arrhythmias	49	2.10 ± 0.19	<0.001
Day 5	With arrhythmias	63	1.76 ± 0.51	<0.001
	Without arrhythmias	49	2.26 ± 0.20	<0.001

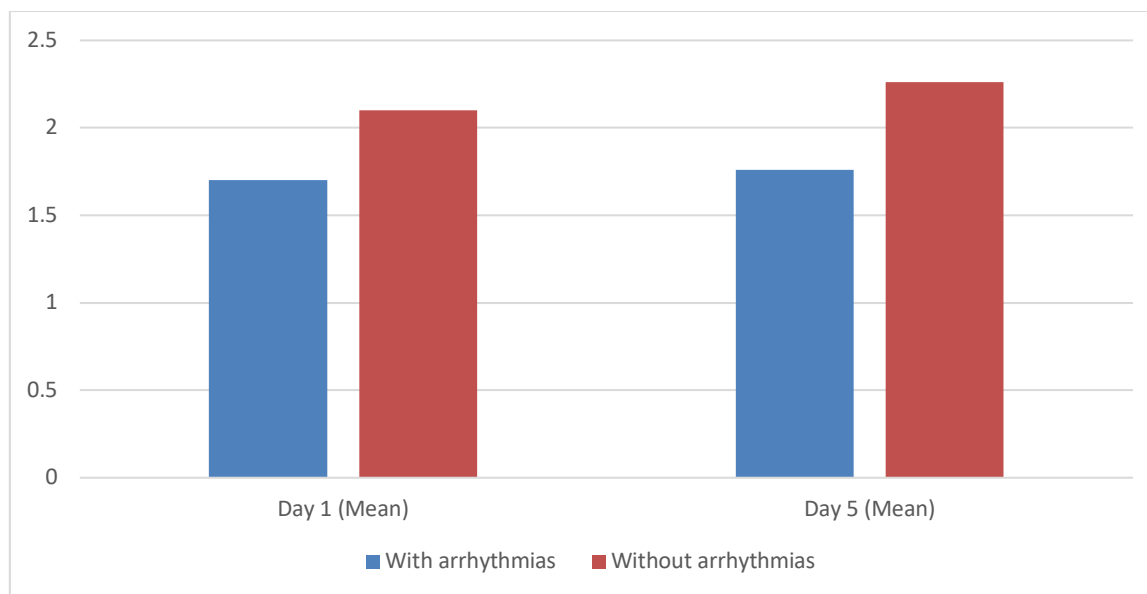


Figure 2: Association of Serum Magnesium Levels with Arrhythmias

Discussion

Magnesium plays a crucial role in cardiovascular physiology as an essential divalent cation involved in membrane stabilization, cellular ion transport, and regulation of calcium-mediated processes. Disturbances in magnesium balance can alter myocardial excitability and conduction and may consequently increase susceptibility to cardiac rhythm disturbances in patients with acute myocardial infarction (AMI). In the present study involving 112 patients, the majority were males, and patients aged 50 years and above represented the predominant age group. This pattern may reflect the greater burden of conventional cardiovascular risk factors among older individuals and their established contribution to the development of AMI.

The present study demonstrated a gradual rise in mean serum magnesium concentration from 1.88 ± 0.26 mg/dL on Day 1 to 1.98 ± 0.47 mg/dL on Day 5. However, patients who developed arrhythmias consistently had lower magnesium concentrations than those without arrhythmias. On Day 1, the mean magnesium level was 1.70 ± 0.16 mg/dL in patients with arrhythmias compared with 2.10 ± 0.19 mg/dL in those without arrhythmias ($p < 0.001$). A similar difference was observed on Day 5, with concentrations of 1.76 ± 0.51 mg/dL and 2.26 ± 0.20 mg/dL, respectively ($p < 0.001$). The early reduction followed by a gradual recovery in serum magnesium is consistent with previously reported observations of decreased magnesium concentrations during the acute phase of AMI [12-15]. These studies have demonstrated that magnesium levels may fall during the initial days following myocardial infarction and subsequently show progressive normalization, supporting the temporal changes observed in the present study.

The significantly lower magnesium concentrations among patients who developed arrhythmias indicate a potential relationship between magnesium depletion and post-infarction electrical instability. Magnesium deficiency can influence myocardial membrane potential and ionic balance, thereby facilitating abnormal ventricular electrical activity. Evidence from previous therapeutic studies regarding magnesium supplementation, however, remains inconsistent. Some investigations have demonstrated a reduction in ventricular arrhythmias following intravenous magnesium administration, suggesting a possible protective effect [16]. A reduction in mortality has also been reported in patients receiving magnesium therapy, although the effect on treatment-requiring tachyarrhythmias was not statistically significant [17]. Conversely, other studies have failed to demonstrate significant differences in mortality or clinically important ventricular dysrhythmias between magnesium-treated and placebo groups [18]. These conflicting findings indicate that the clinical benefit of routine magnesium supplementation in AMI may depend on factors such as the degree of magnesium depletion, timing of treatment, and characteristics of the affected population.

The present findings are further supported by earlier evidence demonstrating lower magnesium concentrations in patients with AMI and a greater occurrence of serious ventricular premature beats, ventricular tachycardia, and ventricular fibrillation among those with hypomagnesemia [11]. In the current study, although serum magnesium concentrations increased from Day 1 to Day 5, patients who developed arrhythmias continued to have significantly lower levels than those who remained free of arrhythmic events. This persistent difference suggests that serum magnesium status

during the acute phase of AMI may have prognostic relevance for arrhythmic risk. Regular assessment of magnesium levels may therefore assist in identifying patients who require closer cardiac monitoring. Nevertheless, the observational nature of the study does not establish a causal relationship, and larger prospective investigations are needed to determine whether early correction of hypomagnesemia can effectively prevent or reduce arrhythmias in patients with AMI.

Conclusion

The present study demonstrates a significant association between lower serum magnesium concentrations and the development of cardiac arrhythmias in patients with acute myocardial infarction. Patients who developed arrhythmias had significantly lower serum magnesium levels on both the first and fifth days of hospitalization compared with those without arrhythmias, despite a gradual increase in magnesium levels over time. These findings suggest that hypomagnesemia may contribute to myocardial electrical instability during the early phase of acute myocardial infarction. Monitoring serum magnesium levels may therefore be useful in identifying patients at increased risk of arrhythmic complications and may have potential implications for their clinical management. Further larger-scale prospective studies are required to determine whether correction of low magnesium levels can reduce arrhythmia-related complications.

References

1. Razzaque MS, Wimalawansa SJ. Minerals and human health: from deficiency to toxicity. *Nutrients*. 2025 Jan 26;17(3):454.
2. Binesh A, Venkatachalam K, Ranjan A, Joy S, Prasannan A. Major Minerals (Na, K, Ca, Mg, P) in Physiological Function and Disease. In: *Functional Biochemistry of Metallic Elements* 2026 Feb 7 (pp. 355-382). Cham: Springer Nature Switzerland.
3. Soetan KO, Olaiya CO, Oyewole OE. The importance of mineral elements for humans, domestic animals and plants: A review. *African journal of food science*. 2010 May 31;4(5):200-22.
4. Quintaes KD, Diez-Garcia RW. The importance of minerals in the human diet. *Handbook of mineral elements in food*. 2015 Feb 20:1-21.
5. Tangvoraphonkchai K, Davenport A. Magnesium and cardiovascular disease. *Advances in chronic kidney disease*. 2018 May 1;25(3):251-60.
6. Fatima G, Dzupina A, Alhmadi HB, Magomedova A, Siddiqui Z, Mehdi A, Hadi N, RAZA AM. Magnesium matters: A comprehensive review of its vital role in health and diseases. *Cureus*. 2024 Oct 13;16(10).
7. Chakraborti S, Chakraborti T, Mandal M, Mandal A, Das S, Ghosh S. Protective role of magnesium in cardiovascular diseases: a review. *Molecular and cellular biochemistry*. 2002 Sep;238(1):163-79.
8. Liu M, Dudley Jr SC. Magnesium, oxidative stress, inflammation, and cardiovascular disease. *Antioxidants*. 2020 Sep 23;9(10):907.
9. Speich M, Gelot S, Arnaud P, Nicolas G. Changes in magnesium, zinc, calcium, potassium, cholesterol, and creatine kinase concentrations in patients from pre-infarction syndrome to fatal myocardial infarction. *Clinical chemistry*. 1988 Oct 1;34(10):2083-6.
10. Ceremuzynski L, Jurgiel R et al . Threatening arrhythmias in acute myocardial infarction are prevented by intravenous magnesium sulphate. *Am Heart J*. 1989; 118: 1333 -1334.
11. Dyckner T. Serum magnesium in acute myocardial infarction: relation to arrhythmias. *Acta Medica Scandinavica*. 1980 Jan 12;207(1-6):59-66.
12. Abraham AS, Rosenmann D. Magnesium in the prevention of lethal arrhythmias in acute myocardial infarction. *Arch. Intern Med*. 1987; 147:753-755.
13. Singh RB, Sircar AR et al . Magnesium and potassium administration in acute myocardial infarction. *Magnesium Trace Elem*. 1990; 9:198-204.
14. Gopalakrishnan T, Sivakumar J. A Study of Correlation Between Level of Serum Magnesium and Arrhythmias in Patients with Acute Myocardial Infarction. *International Journal of Medical and Pharmaceutical Research*. 2025 Oct 20;6:1759-63.
15. Baset M, Haque HA, Saha M, Barai L, Tareq Z. Comparison of serum magnesium level (1-5 day) in patients with and without arrhythmias in patients with acute myocardial infarction. *Eur J Pharm Med Res*. 2020;7(9):56-61.
16. Rasmussen HS, Suenson M, McNair P, Nørregård P, Balslev S. Magnesium infusion reduces the incidence of arrhythmias in acute myocardial infarction. A double-blind placebo-controlled study. *Clinical cardiology*. 1987 Jun;10(6):351-6.
17. Shechter M, Mark N et al . Beneficial effects of magnesium sulphate in acute myocardial infarction. *Am J Cardiol* . 1990; 66:271-274.
18. Smith LF, Heagerty AM, Bing RF, Barnett DB. Intravenous infusion of magnesium sulphate after acute myocardial infarction: effects on arrhythmias and mortality. *International journal of cardiology*. 1986 Aug 1;12(2):175-80.