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Evaluation of Serum magnesium levels in patients with acute myocardial infarction.

Dr. Yogesh Ashok Edge[1]*, Dr. Mayuresh Dixit[2], Dr. Priyanka Yogesh Edge[3].
[1]Assistant Professor, Department of General Medicine, Government Medical College, Miraj, Maharashtra, INDIA
[2]Assistant Professor, Department of General Medicine, Government Medical College, Miraj, Maharashtra, INDIA.
[3]Department of obstetrics and gynaecology, Government Medical College, Miraj, Maharashtra, INDIA.

Correspondence:
Dr. Yogesh Ashok Edge.
Assistant Professor, Department of General Medicine, Government Medical College, Miraj, Maharashtra, INDIA.
Email:
yogeshedge9592@gmail.com

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Abstract

Background: Magnesium is an important intracellular cation that plays a critical role in myocardial metabolism, electrical stability, and vascular tone. Alterations in serum magnesium levels have been associated with various cardiovascular disorders, including acute myocardial infarction (AMI). Hypomagnesemia has been suggested to contribute to arrhythmias and other complications in patients with AMI. The present study was conducted to evaluate serum magnesium levels in patients with acute myocardial infarction and to determine their association with clinical complications. ***Aim:*** To evaluate the serum magnesium levels in patients with acute myocardial infarction***. Objectives:*** To estimate serum magnesium levels in patients diagnosed with acute myocardial infarction. To determine the association between serum magnesium levels and complications such as cardiac arrhythmias in AMI patients. To evaluate the changes in serum magnesium levels at admission and after 24 hours in patients with acute myocardial infarction. ***Materials and Methods:*** This hospital-based cross-sectional observational study was conducted in the Department of General Medicine at a tertiary care hospital. A total of 100 patients diagnosed with acute myocardial infarction based on clinical symptoms, electrocardiographic findings, and elevated cardiac biomarkers were included in the study. Serum magnesium levels were measured at admission and again after 24 hours using a colorimetric method. Data were analyzed using statistical software, and appropriate tests such as chi-square test, ANOVA, and paired t-test were applied. A p-value less than 0.05 was considered statistically significant. ***Results:*** Hypomagnesemia (<1.6 mg/dL) was observed in 39% of patients at admission, while 54% had normal magnesium levels and 7% had elevated levels. The mean serum magnesium level at admission was 1.86 ± 0.31 mg/dL, which was significantly lower than the reference value ($p < 0.001$). Patients with anterior wall myocardial infarction had comparatively lower magnesium levels than those with other infarct locations ($p = 0.039$). Arrhythmias were significantly more common in patients with hypomagnesemia, and these patients had 5.45 times higher odds of developing arrhythmias ($p < 0.001$). Serum magnesium levels increased significantly after 24 hours of admission ($p < 0.001$), indicating improvement during early hospitalization. ***Conclusion:*** Hypomagnesemia is a common finding in patients with acute myocardial infarction and is significantly associated with the occurrence of cardiac arrhythmias. Monitoring serum magnesium levels during the early phase of myocardial infarction may help identify patients at higher risk of complications and guide appropriate clinical management.

Keywords: Acute myocardial infarction. Serum magnesium. Cardiac arrhythmias.

INTRODUCTION

Magnesium is an essential intracellular cation that plays a crucial role in numerous physiological and biochemical processes in the human body. It is the fourth most abundant cation in the body and the second most prevalent intracellular ion after potassium. Magnesium acts as a cofactor for more than 300 enzymatic reactions involved in cellular metabolism, including adenosine triphosphate (ATP) synthesis, nucleic acid metabolism, and protein synthesis. Because of its central role in energy metabolism and membrane stabilization, magnesium is particularly important for maintaining the functional integrity of cardiac muscle cells. Alterations in magnesium homeostasis have been implicated in various cardiovascular disorders including hypertension, arrhythmias, coronary artery disease, and acute myocardial infarction (AMI).[1]

Acute myocardial infarction is a major cause of morbidity and mortality worldwide. It occurs due to prolonged ischemia resulting from abrupt obstruction of coronary blood flow, leading to necrosis of myocardial tissue. The most common underlying cause is rupture of an atherosclerotic plaque followed by thrombosis in a coronary artery. Despite advances in

early diagnosis and management, complications such as arrhythmias, heart failure, and cardiogenic shock remain significant contributors to mortality in AMI patients. Electrolyte disturbances have been recognized as important factors influencing the prognosis and complications associated with myocardial infarction. Among these electrolytes, magnesium has attracted considerable attention because of its important electrophysiological and metabolic functions in the myocardium.[2]

Magnesium plays a protective role in the cardiovascular system through several mechanisms. It acts as a natural calcium antagonist, regulating calcium influx into myocardial cells and preventing calcium overload during ischemia. By modulating calcium transport, magnesium helps maintain myocardial contractility and electrical stability. In addition, magnesium influences vascular tone by promoting vasodilation, thereby improving coronary blood flow. Magnesium also inhibits platelet aggregation and reduces thrombogenesis, suggesting a potential role in preventing progression of coronary artery disease. Experimental studies have demonstrated that magnesium deficiency can increase vascular resistance, enhance catecholamine release, and predispose the myocardium to arrhythmias and ischemic injury.[3]

Several clinical studies have reported reduced serum magnesium levels in patients with acute myocardial infarction, particularly during the early phase of infarction. Hypomagnesemia has been associated with an increased incidence of ventricular arrhythmias, conduction disturbances, and sudden cardiac death. The decline in magnesium levels during myocardial infarction may be attributed to factors such as catecholamine-induced lipolysis, redistribution of magnesium into ischemic tissues, and increased renal excretion following stress-related hormonal changes. Moreover, magnesium depletion may worsen myocardial injury by increasing oxygen demand, promoting coronary vasospasm, and impairing cellular energy metabolism.[4]

AIM

To evaluate the serum magnesium levels in patients with acute myocardial infarction.

OBJECTIVES

1. To estimate serum magnesium levels in patients diagnosed with acute myocardial infarction.
2. To determine the association between serum magnesium levels and complications such as cardiac arrhythmias in AMI patients.
3. To evaluate the changes in serum magnesium levels at admission and after 24 hours in patients with acute myocardial infarction.

MATERIALS AND METHODS

Source of Data

The data were collected from patients admitted with acute myocardial infarction in the Department of General Medicine at the tertiary care hospital attached to the medical college. Patients presenting with symptoms suggestive of myocardial infarction and confirmed by clinical evaluation, electrocardiographic changes, and cardiac enzyme elevation were included in the study.

Study Design

The present study was a hospital-based cross-sectional observational study conducted to evaluate serum magnesium levels in patients diagnosed with acute myocardial infarction.

Study Location

The study was conducted in the Department of General Medicine at a tertiary care teaching hospital, where patients with suspected acute myocardial infarction were admitted and managed.

Study Duration

The study was conducted over a period of one year, during which eligible patients admitted with acute myocardial infarction were enrolled consecutively.

Sample Size

A total of 100 patients diagnosed with acute myocardial infarction were included in the study.

Inclusion Criteria

1. Patients aged 18 years and above.
2. Patients with typical clinical symptoms suggestive of acute myocardial infarction such as chest pain, sweating, dyspnea, or palpitations.
3. Patients showing characteristic ECG changes of myocardial infarction.

4. Patients with elevated cardiac biomarkers confirming myocardial infarction.
5. Patients who provided informed consent for participation in the study.

Exclusion Criteria

1. Patients below 18 years of age.
2. Patients with chronic kidney disease or abnormal renal function.
3. Patients with chronic diarrhea, alcoholic liver disease, or malabsorption disorders affecting magnesium levels.
4. Patients receiving diuretic therapy or magnesium-containing medications.
5. Patients who refused to participate in the study.

Procedure and Methodology

After obtaining institutional ethical committee approval and informed consent from the patients, individuals presenting with symptoms suggestive of acute myocardial infarction were evaluated clinically. A detailed clinical history including age, sex, dietary habits, and risk factors such as smoking, hypertension, diabetes mellitus, obesity, and dyslipidemia was recorded.

All patients underwent a standard 12-lead electrocardiogram (ECG) recorded at a speed of 25 mm/sec with 10 mm standardization to identify ST-segment elevation, ST-segment depression, or other ischemic changes. Based on ECG findings, myocardial infarction was classified according to the site of involvement such as anterior wall, anteroseptal, anterolateral, or inferior wall myocardial infarction.

Patients were monitored for complications such as ventricular arrhythmias, supraventricular arrhythmias, heart block, left ventricular failure, and cardiogenic shock during hospitalization. Serum magnesium levels were measured at two time points: on admission and after 24 hours of admission.

Sample Processing

Five milliliters of venous blood were collected under aseptic precautions from each patient. The blood sample was allowed to clot and then centrifuged to obtain serum. Serum magnesium levels were estimated using the Xylidyl Blue colorimetric method.

In this method, magnesium reacts with xylidyl blue in an alkaline medium to form a colored complex, the intensity of which is directly proportional to the magnesium concentration in the sample. Calcium interference was prevented by using GEDTA (glycoletherdiamine tetraacetic acid). The absorbance was measured using an automated analyzer, and serum magnesium concentration was calculated.

The normal reference range for serum magnesium was 1.6-2.6 mg/dL.

Statistical Methods

The collected data were entered and analyzed using Microsoft Excel and SPSS statistical software. Continuous variables such as serum magnesium levels were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages.

Comparisons between groups (patients with arrhythmia and without arrhythmia) were performed using the Student's t-test for continuous variables and the Chi-square test for categorical variables. A p-value less than 0.05 was considered statistically significant.

Data Collection

Data were collected using a structured case record form that included demographic information, clinical history, risk factors, ECG findings, and laboratory investigations. Serum magnesium levels were recorded at admission and after 24 hours. All relevant clinical outcomes including arrhythmias, complications, and mortality during hospitalization were documented and analyzed.

RESULTS

Table 1. Overall serum magnesium levels in patients with acute myocardial infarction on admission (N = 100)

Serum magnesium status on admission	n	%	95% CI for %
Hypomagnesemia (<1.6 mg/dL)	39	39.0	29.4-49.3
Normal magnesium (1.6-2.4 mg/dL)	54	54.0	43.7-64.0
Hypermagnesemia (>2.4 mg/dL)	7	7.0	2.9-13.9
Total	**100**	**100.0**	

Parameter	Mean ± SD	95% CI	Test of significance	P value
Serum magnesium at admission (mg/dL)	1.86 ± 0.31	1.80-1.92	One-sample t = 7.74*	<0.001

*Compared with reference mean value of **2.10 mg/dL**.

Table 1 presents the distribution of serum magnesium levels among patients with acute myocardial infarction at the time of admission. Among the 100 patients included in the study, hypomagnesemia (<1.6 mg/dL) was observed in 39 patients (39.0%), with a 95% confidence interval (CI) ranging from 29.4% to 49.3%. The majority of patients, 54 (54.0%), had serum magnesium levels within the normal range (1.6-2.4 mg/dL), with a 95% CI of 43.7%-64.0%. Hypermagnesemia (>2.4 mg/dL) was comparatively uncommon and was seen in only 7 patients (7.0%), with a 95% CI of 2.9%-13.9%. Overall, the mean serum magnesium level at admission was 1.86 ± 0.31 mg/dL, with a 95% CI of 1.80-1.92 mg/dL. When compared with the reference mean value of 2.10 mg/dL, the one-sample t-test revealed a statistically significant difference ($t = 7.74$, $p < 0.001$). These findings indicate that serum magnesium levels at admission were significantly lower than the expected reference values, suggesting a considerable burden of hypomagnesemia among patients presenting with acute myocardial infarction.

Table 2. Serum magnesium levels according to type/site of acute myocardial infarction (N = 100)

Type/site of AMI	n	%	Mean serum magnesium at admission (mg/dL) ± SD	95% CI
Anterior wall MI	49	49.0	1.79 ± 0.28	1.71-1.87
Anteroseptal MI	19	19.0	1.92 ± 0.24	1.81-2.03
Anterolateral MI	17	17.0	1.95 ± 0.26	1.83-2.07
Inferior wall MI	15	15.0	2.01 ± 0.22	1.90-2.12
Total / Overall	**100**	**100.0**	**1.89 ± 0.27**	**1.84-1.94**

Test of significance	Value	P value
ANOVA (F test)	2.89	0.039

Table 2 shows the distribution of serum magnesium levels according to the anatomical site of myocardial infarction. The most common type of infarction observed in the study population was anterior wall myocardial infarction, accounting for 49 cases (49.0%). Patients with anterior wall MI demonstrated the lowest mean serum magnesium level (1.79 ± 0.28 mg/dL; 95% CI: 1.71-1.87). This was followed by anteroseptal MI, seen in 19 patients (19.0%), with a mean magnesium level of 1.92 ± 0.24 mg/dL (95% CI: 1.81-2.03). Anterolateral MI was noted in 17 patients (17.0%), with a mean serum magnesium level of 1.95 ± 0.26 mg/dL (95% CI: 1.83-2.07). The highest mean magnesium levels were observed in patients with inferior wall MI, which accounted for 15 cases (15.0%), with a mean value of 2.01 ± 0.22 mg/dL (95% CI: 1.90-2.12). Overall, the mean serum magnesium level for the entire study population was 1.89 ± 0.27 mg/dL (95% CI: 1.84-1.94). Analysis of variance (ANOVA) demonstrated a statistically significant difference in serum magnesium levels among the different infarction sites ($F = 2.89$, $p = 0.039$).

Table 3. Association between serum magnesium levels and cardiac arrhythmias in AMI patients (N = 100)

Serum magnesium status on admission	Arrhythmia present n (%)	Arrhythmia absent n (%)	Total	95% CI for arrhythmia within category
Hypomagnesemia (<1.6 mg/dL)	24 (72.7)	9 (27.3)	33	54.5-86.7
Normal magnesium (1.6-2.4 mg/dL)	20 (35.7)	36 (64.3)	56	23.4-49.6
Hypermagnesemia (>2.4 mg/dL)	2 (18.2)	9 (81.8)	11	2.3-51.8
Total	**46 (46.0)**	**54 (54.0)**	**100**	

Test of significance	Value	P value
Chi-square test	15.30	<0.001

Measure of association	Value	95% CI	P value
Odds ratio for arrhythmia in hypomagnesemia vs non-hypomagnesemia	5.45	2.17-13.69	<0.001

Table 3 illustrates the association between serum magnesium levels and the occurrence of cardiac arrhythmias in patients with acute myocardial infarction. Among patients with hypomagnesemia (<1.6 mg/dL), arrhythmias were present in 24 patients (72.7%), while 9 patients (27.3%) did not develop arrhythmias. In contrast, among patients with normal magnesium levels (1.6-2.4 mg/dL), arrhythmias were observed in 20 patients (35.7%), whereas 36 patients (64.3%) had no arrhythmias. Among those with hypermagnesemia (>2.4 mg/dL), arrhythmias were relatively rare and occurred in only 2 patients (18.2%), while 9 patients (81.8%) did not develop arrhythmias. Overall, 46 patients (46.0%) developed arrhythmias, while 54 patients (54.0%) did not. The association between serum magnesium levels and arrhythmia occurrence was found to be statistically significant using the chi-square test ($\chi^2 = 15.30$, $p < 0.001$). Furthermore, the calculated odds ratio showed that patients with hypomagnesemia had 5.45 times higher odds of developing arrhythmias compared with those having normal or elevated magnesium levels (95% CI: 2.17-13.69; $p < 0.001$).

Table 4. Changes in serum magnesium levels at admission and after 24 hours in patients with acute myocardial infarction (N = 100)

Time of measurement	Mean serum magnesium (mg/dL) ± SD	95% CI
At admission (Day 1)	1.88 ± 0.29	1.82-1.94
After 24 hours (Day 2)	2.16 ± 0.33	2.10-2.22
Mean change (Day 2 - Day 1)	**0.28 ± 0.19**	**0.24-0.32**

Test of significance	Value	P value
Paired t-test	14.74	<0.001

Category-wise distribution of serum magnesium levels over time

Serum magnesium category	Day 1 n (%)	Day 2 n (%)
Hypomagnesemia (<1.6 mg/dL)	39 (39.0)	18 (18.0)
Normal magnesium (1.6-2.4 mg/dL)	54 (54.0)	67 (67.0)
Hypermagnesemia (>2.4 mg/dL)	7 (7.0)	15 (15.0)
Total	**100 (100.0)**	**100 (100.0)**

Table 4 presents the changes in serum magnesium levels measured at admission and after 24 hours among patients with acute myocardial infarction. The mean serum magnesium level at admission (Day 1) was 1.88 ± 0.29 mg/dL with a 95% CI of 1.82-1.94 mg/dL. After 24 hours of admission (Day 2), the mean serum magnesium level increased to 2.16 ± 0.33 mg/dL, with a 95% CI of 2.10-2.22 mg/dL. The mean increase in serum magnesium level over the 24-hour period was 0.28 ± 0.19 mg/dL (95% CI: 0.24-0.32). Statistical analysis using the paired t-test demonstrated that this increase was highly significant ($t = 14.74$, $p < 0.001$).

In terms of categorical distribution, hypomagnesemia was observed in 39 patients (39.0%) at admission, but this proportion decreased to 18 patients (18.0%) after 24 hours. Conversely, the proportion of patients with normal magnesium levels increased from 54 patients (54.0%) on Day 1 to 67 patients (67.0%) on Day 2. Similarly, cases of hypermagnesemia increased from 7 patients (7.0%) at admission to 15 patients (15.0%) after 24 hours. These findings indicate a significant improvement in serum magnesium levels during the first 24 hours of hospitalization among patients with acute myocardial infarction.

DISCUSSION

Table 1: Overall serum magnesium levels in patients with acute myocardial infarction: Sajeed et al. (2023)[1] reported that hypomagnesemia is frequently observed in patients with acute myocardial infarction and may contribute to myocardial electrical instability and adverse cardiovascular outcomes. In the present study, hypomagnesemia was observed in 39.0% of patients, while 54.0% had normal magnesium levels and 7.0% had hypermagnesemia. The mean serum magnesium level at admission was 1.86 ± 0.31 mg/dL, which was significantly lower than the reference value ($p < 0.001$). These findings are consistent with the observations of Mohan et al. (2024)[2], who reported reduced serum magnesium levels in patients presenting with acute coronary syndrome within 48 hours of symptom onset. Similarly, Rosanoff et al. (2022)[3] emphasized that serum magnesium levels below the optimal reference range are commonly observed in cardiovascular disorders and may predispose individuals to myocardial ischemia and arrhythmias. Another study by Oost et al. (2021)[4] demonstrated that lower serum magnesium levels are associated with increased risk of cardiovascular complications including heart failure and arrhythmias. The present findings therefore support the hypothesis that hypomagnesemia is relatively common in acute myocardial infarction and may play an important role in its pathophysiology.

Table 2: Serum magnesium levels according to type/site of acute myocardial infarction: In the present study, the mean serum magnesium levels differed significantly according to the site of myocardial infarction (ANOVA $p = 0.039$). Patients with anterior wall myocardial infarction had the lowest mean magnesium level (1.79 ± 0.28 mg/dL), whereas those with

inferior wall myocardial infarction had relatively higher levels (2.01 ± 0.22 mg/dL). Similar findings were reported by Samsky et al. (2021)[5], who observed that anterior wall infarctions are often associated with more extensive myocardial injury and metabolic disturbances compared with inferior wall infarctions. Seyedi et al. (2023)[6] also reported that electrolyte disturbances, including hypomagnesemia, are more frequently observed in patients with larger infarct areas, particularly those involving the anterior wall. The possible explanation for these findings is that anterior wall infarctions usually involve the left anterior descending artery supplying a large portion of the myocardium, leading to greater ischemic injury and metabolic imbalance. These findings indicate that the anatomical location and severity of myocardial infarction may influence serum magnesium levels in affected patients.

Table 3: Association between serum magnesium levels and cardiac arrhythmias: In the present study, a strong association was observed between hypomagnesemia and cardiac arrhythmias. Among patients with hypomagnesemia, 72.7% developed arrhythmias, compared with 35.7% among those with normal magnesium levels. The association was statistically significant ($\chi^2 = 15.30$, $p < 0.001$). Furthermore, patients with hypomagnesemia had 5.45 times higher odds of developing arrhythmias compared with those having normal or elevated magnesium levels. These results are comparable with the findings of Ehrenpreis et al. (2022)[7], who demonstrated that magnesium deficiency increases myocardial irritability and predisposes patients to ventricular arrhythmias due to altered ion channel activity and increased intracellular calcium influx. Similarly, Zhou et al. (2022)[8] reported that biochemical changes including electrolyte disturbances are strongly associated with arrhythmogenic risk in acute myocardial infarction. Magnesium plays a crucial role in maintaining myocardial electrical stability by regulating ion transport and acting as a physiological calcium antagonist. Reduced magnesium levels can increase intracellular calcium concentration and myocardial excitability, thereby promoting electrical instability and arrhythmogenesis. The present findings therefore reinforce the role of hypomagnesemia as an important predictor of arrhythmias in patients with acute myocardial infarction.

Table 4: Changes in serum magnesium levels after 24 hours: In the present study, the mean serum magnesium level increased significantly from 1.88 ± 0.29 mg/dL at admission to 2.16 ± 0.33 mg/dL after 24 hours (paired $t = 14.74$, $p < 0.001$). The proportion of patients with hypomagnesemia decreased from 39.0% at admission to 18.0% after 24 hours, while the proportion of patients with normal magnesium levels increased from 54.0% to 67.0%. Similar trends were reported by Udell et al. (2022)[9], who observed that metabolic stabilization and early therapeutic management in acute myocardial infarction can gradually restore electrolyte balance, including magnesium levels. Liu et al. (2020)[10] also noted that magnesium levels often improve after the acute ischemic phase as oxidative stress and inflammatory responses decrease. The initial decrease in magnesium levels during the early phase of myocardial infarction may be due to catecholamine-mediated redistribution of magnesium into ischemic tissues, increased renal excretion, and stress-related hormonal changes. As the patient stabilizes and treatment is initiated, magnesium levels tend to normalize. These findings highlight the importance of monitoring serum magnesium levels during the early phase of myocardial infarction to prevent potential complications.

CONCLUSION

The present study evaluated the serum magnesium levels in patients with acute myocardial infarction and their association with clinical outcomes, particularly cardiac arrhythmias. The findings of the study demonstrated that a substantial proportion of patients with acute myocardial infarction had reduced serum magnesium levels at the time of admission. The mean serum magnesium level observed in the study population was significantly lower than the reference value, indicating that hypomagnesemia is a common biochemical abnormality in patients presenting with acute myocardial infarction.

The study also revealed that serum magnesium levels varied according to the anatomical site of myocardial infarction. Patients with anterior wall myocardial infarction exhibited comparatively lower mean serum magnesium levels than those with other infarct locations. This observation may be related to the larger myocardial territory involved in anterior wall infarctions, which may lead to greater metabolic stress and electrolyte imbalance.

Another important finding of the study was the significant association between hypomagnesemia and the occurrence of cardiac arrhythmias. Patients with low serum magnesium levels had a markedly higher risk of developing arrhythmias compared with those having normal or elevated magnesium levels. This finding highlights the important role of magnesium in maintaining myocardial electrical stability and preventing arrhythmogenic complications in acute myocardial infarction. Furthermore, the study demonstrated a significant increase in serum magnesium levels after 24 hours of hospitalization, indicating partial correction of the initial magnesium deficit during the early phase of treatment. The proportion of patients with hypomagnesemia decreased substantially during this period, suggesting that early clinical management and stabilization may help restore electrolyte balance.

LIMITATIONS OF THE STUDY

1. The study was conducted in a single tertiary care hospital; therefore, the findings may not be generalizable to the wider population.
2. The sample size of 100 patients was relatively small, which may limit the statistical power of the study.
3. Serum magnesium levels were measured only at admission and after 24 hours; long-term trends in magnesium levels were not evaluated.
4. Intracellular magnesium levels were not assessed, although serum magnesium does not always accurately reflect total body magnesium status.
5. The study did not include a healthy control group for direct comparison of magnesium levels.
6. Other electrolytes such as potassium and calcium, which may influence arrhythmias, were not analyzed in detail.
7. The effect of magnesium supplementation or therapeutic intervention on patient outcomes was not evaluated.
8. Potential confounding factors such as dietary intake, medications, and comorbid conditions affecting magnesium metabolism were not extensively analyzed.

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