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A study on serum potassium and serum magnesium levels in acute myocardial infarction with special emphasis on rhythm disturbance

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ABSTRACT

Background and objectives. Myocardial infarction (MI) is a severe cardiac event associated with plaque formation in arteries, leading to reduced blood flow to the heart. Arrhythmias, such as bradyarrhythmia or tachyarrhythmia, commonly accompany MI, contributing to its morbidity and mortality. Electrolyte disturbances, particularly potassium and magnesium imbalances, can exacerbate arrhythmic risks in MI patients. This study aimed to assess the incidence of alterations in serum potassium and magnesium levels during acute MI and their correlation with rhythm disturbances.

Material and methods. A prospective analytical study was conducted with 150 patients diagnosed with acute MI based on ECG changes and elevated cardiac markers. Serum potassium and magnesium levels were measured, and rhythm disturbances were assessed using continuous cardiac monitoring and standard ECG. Spearman correlation analysis was performed to evaluate the association between serum potassium, serum magnesium, and rhythm changes.

Results. The study investigated the demographic and clinical characteristics of participants and the potential relationship between serum electrolyte levels and arrhythmic changes. The mean age of the participants was 54.3 years, with a male predominance (76% male, 24% female). Among the participants, 50.67% had diabetes, 52% had hypertension, and 28% had both conditions. Serum electrolyte analysis showed elevated magnesium levels in 6.66% and elevated potassium levels in 12.66% of patients. Notably, arrhythmic changes in ECG were present in 62% of patients, with sinus tachycardia being the most prevalent (17%). However, statistical analysis revealed no significant correlation between serum magnesium, serum potassium, and rhythm changes ($p > 0.05$). These findings suggest that while arrhythmic changes are common, they are not directly associated with elevated levels of these electrolytes, indicating the need for further investigation into other potential factors influencing arrhythmias in this population.

Conclusion. Despite the prevalence of electrolyte disturbances in acute MI patients, this study found no significant association between serum magnesium, serum potassium levels, and rhythm disturbances. Further research is warranted to elucidate the complex interplay between electrolyte imbalances and arrhythmias in MI.

Keywords: serum bilirubin, coronary artery disease, cardiovascular risk factors, antioxidative properties, inflammatory markers, myocardial infarction (MI), arrhythmia, potassium, magnesium, electrolyte disturbances, ECG changes, cardiac markers, correlation analysis

Abbreviations (in alphabetical order):

AWMI – Anterior Wall MI
CK-MB – Creatine Kinase-MB
ECG – Electrocardiogram
MI – myocardial infarction
NSTEMI – Non-ST Elevation MI

INTRODUCTION

Myocardial infarction (MI) is a serious cardiac event often resulting from plaque formation in the arteries, leading to reduced blood flow and oxygen supply to the heart [1]. This condition significantly increases the risk of various arrhythmias, which

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can manifest as either bradyarrhythmia or tachyarrhythmia.

Cardiomyocytes, the muscle cells of the heart, become dysfunctional during an MI primarily due to ischemia, a lack of blood supply [1]. Ischemia increases the excitability of the heart's nodal tissues, causing them to fire at an abnormally high rate, leading to arrhythmias. These arrhythmias often present with an accelerated heart rate, attempting to compensate for the failing heart. However, a heart rate exceeding 130 beats per minute (bpm) can impair diastolic filling, leading to poor organ perfusion [2].

The activation of baroreceptors and chemoreceptors due to poor perfusion can lead to reflex tachycardia, exacerbating the situation. Effective management and post-MI monitoring are crucial in clinical practice.

A significant aspect of managing MI involves monitoring electrolyte disturbances, particularly potassium and magnesium levels, as imbalances can exacerbate arrhythmic risks. Magnesium, often overlooked in clinical assessments, plays a critical role in cardiovascular health. Both hypermagnesemia [elevated magnesium levels) and hypomagnesemia (reduced magnesium levels) can lead to cardiac and non-cardiac problems. Magnesium depletion affects Na+ K+ ATPase activity, influences potassium levels, and can increase the risk of arrhythmias [3].

Hypokalemia, a low potassium level, particularly in the early phase of an MI, is often due to sympathetic nervous system activation, leading to a shift of potassium from the extracellular to the intracellular fluid compartment. Magnesium depletion may also contribute to atherosclerosis, further complicating cardiovascular health [4].

Transient declines in serum magnesium are observed in 6-46% of acute MI patients, normalizing within 14 days. Factors contributing to hypomagnesemia include neuro-hormonal activation, pharmacological treatments like diuretics, and reduced renal flow. Catecholamine over-secretion can also lead to hypokalemia and hypomagnesemia [5].

Potassium, the principal intracellular cation, must be maintained within a narrow plasma range (3-7 Meq/ml). Disturbances in potassium levels, either hyperkalemia (high potassium) or hypokalemia, can cause severe arrhythmias. Potassium is crucial for the repolarization phase of the cardiac action potential, and imbalances can affect the excitability of nodal tissues, leading to potentially fatal arrhythmias like torsade de pointes [6].

Calcium gluconate IV is used to counteract nodal excitability issues. Calcium level imbalances, either hypercalcemia or hypocalcemia, can also lead to arrhythmias. Calcium is essential for the initiation of action potentials in cardiomyocytes, and alterations in extracellular calcium can significantly affect heart function [7].

Sodium is another critical extracellular cation. Both hypernatremia (elevated sodium) and hyponatremia (reduced sodium) can impact the Renin-Angiotensin-Aldosterone axis and precipitate heart failure. The normal sodium range is 135-145 Meq/ml, and deviations can lead to various symptoms, including dizziness, seizures, and coma. This study aims to explore the impact of these intracellular cations in MI cases and estimate the incidence of alterations in their blood levels during an MI.

MATERIALS AND METHODS

Study design and setting

This analytical study was undertaken with 150 patients in the Department of Medicine, Saveetha Medical College, to identify patients with acute myocardial infarction (with ECG changes /elevated cardiac markers) and to measure their serum potassium and serum magnesium levels and to correlate their abnormalities with rhythm disturbances.

Participants

All patients confirmed to have MI with elevation of cardiac markers/ ECG, of our hospital was included in this prospective analytical study, after obtaining written informed consent. Relevant clinical history and examination were done for all the study participants.

Inclusion criteria

- All patients more than 18 years of age with acute changes in ECG/ elevated cardiac markers suggestive of acute myocardial infarction.
- Patients with arrhythmia in acute MI as diagnosed by Continuous Cardiac and standard ECG monitoring.

Exclusion criteria

- Patients not giving consent for the above study.
- Patients on thiazide/ loop diuretics.
- Conditions causing hypokalemia and hypomagnesemia like malabsorption syndromes, diabetic ketoacidosis, chronic diarrhea/ persistent vomiting.

Methodology

The study started after obtaining the approval of Institutional Ethics Committee. One hundred and fifty patients with MI as evidenced by acute changes in ECG/ cardiac markers were enrolled in the study after obtaining their informed written consent. A detailed history, examination and routine investigations like complete blood count, random blood sugar, renal function test, liver function test, urine routine were done on all the patients who were enrolled in the study. In addition, serum magnesium, serum potassium, Troponin I, CK-MB and ECG was done in all patients. All the patients were given the appropriate management as per the hospital protocol.

Statistical analysis

Demographic data are expressed as descriptive statistics. Spearman correlation was done to see the association between serum magnesium, serum potassium and rhythm changes in ECG.

RESULTS

The demographic characteristics of the study participants. Mean age of the study patients was 54.3± 10.92 (mean ± SD). In our study of the 150 patients, 76% (n=114) were male and 24 % (n=36) were female patients.

The distribution of comorbidities among study participants are shown in Figure 1. In our study, 50.67% patients had diabetes only, 52% had hypertension only, and 28% had both diabetes and hypertension.

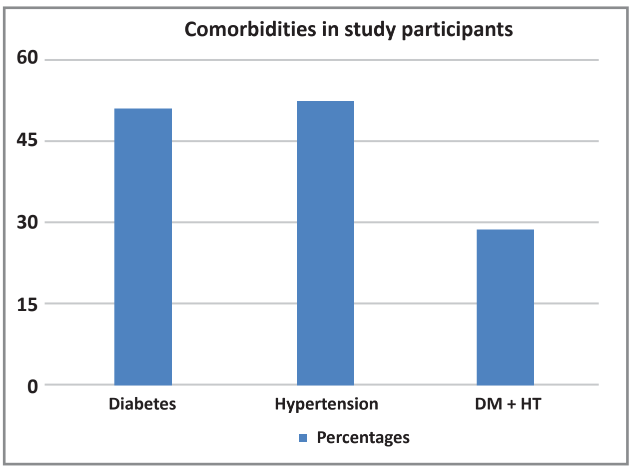

FIGURE 1. Baseline characteristics of study participants

Table 1 illustrates the serum cardiac markers and serum electrolytes in the study participants. Magnesium levels were normal in 65. 33 % patients, elevated in 6. 66% and decreased in 28% of patients. Potassium was normal in 76% patients, elevated in 12.66% and decreased in 11.33% of patients. CK -MB was elevated in 96 % and Troponin I was elevated in 100% of the patients.

The serum cardiac markers and serum electrolytes in the study participants are depicted in Figure 2. Magnesium levels were normal in 65. 33 % patients, elevated in 6. 66% and decreased in 28% of patients. Potassium was normal in 76% patients, elevated in 12.66% and decreased in 11.33% of patients. CK-MB was elevated in 96 % and Troponin I was elevated in 100% of the patients.

TABLE 1 Cardiac markers and Serum electrolytes

Parameter	Normal (%)	Elevated (%)	Decreased (%)
Magnesium	98 (65.33)	10 (6. 66)	42 (28)
Potassium	114 (76)	19 (12.66)	17 (11.33)
CK- MB	6 (4)	144 (96)	0
Troponin I	0	150 (100)	0

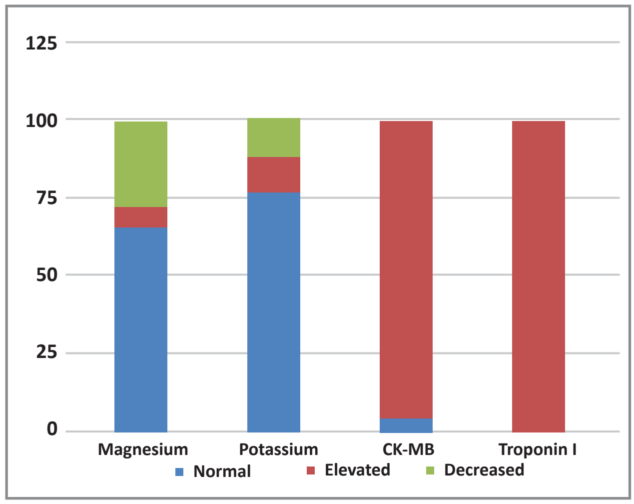

FIGURE 2. Cardiac markers and Serum electrolytes

While Figure 3 presents the ECG changes that was seen in the patients that presented with acute MI. Majority of the patients (33%) presented with AWMI, followed by 27% of patients with IWMI, 23% of patients presented with NSTEMI. PWMI was seen in 11% of patients, ASWMI was seen in 3% and ALWMI was seen in 2% of the patients.

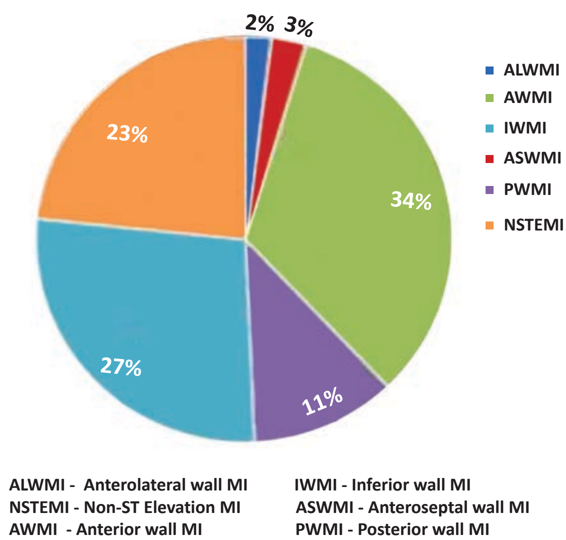

FIGURE 3. ECG changes in acute MI in study participants

Figure 4 shows the arrhythmic changes in ECG in the study participants. 38% of patients did not show

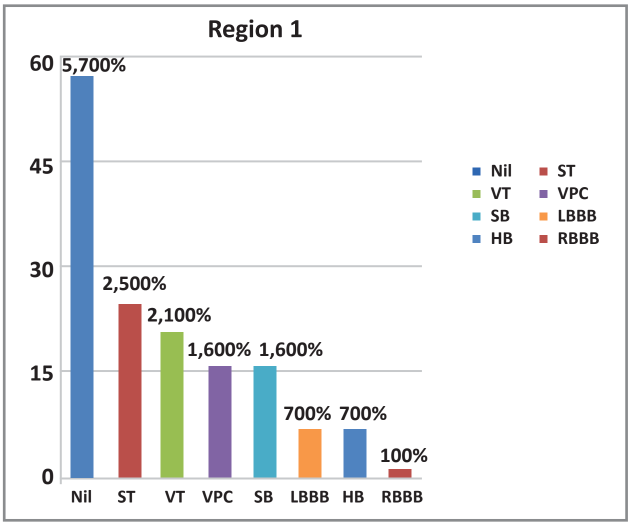

FIGURE 4. Arrhythmic changes in ECG in study participants

any arrhythmic changes. ST was seen in 17 %, VT in 14%, VPC adSBin 11% of patients each. LBBB and HB in 5% of patients. RBBB was seen in 1% of patients which was the least.

The correlation between serum magnesium and serum potassium with rhythm changes were explained in Table 2. It was seen that there was no significant association between the serum magnesium, serum potassium and the rhythm changes in the study participants.

Figure 5 reveals the correlation between serum magnesium with rhythm changes. It was seen that there was no significant association between the serum magnesium and the rhythm changes in the study participants.

Figure 6 finds the correlation between serum potassium with rhythm changes. It was seen that there was no significant association between the serum potassium and the rhythm changes in the study participants.

DISCUSSION

The acute coronary syndrome is one of the common entities in clinical practice. It often manifests

TABLE 2. Correlation between serum magnesium & potassium and rhythm changes in study participants

Parameter	Arrhythmia present	Arrhythmia absent	R2	P value
Magnesium				
Normal	61	37	0.002	0.846
Elevated	7	3		
Decreased	25	17		
Potassium				
Normal	72	42	0.002	0.832
Elevated	11	8		
Decreased	10	7		

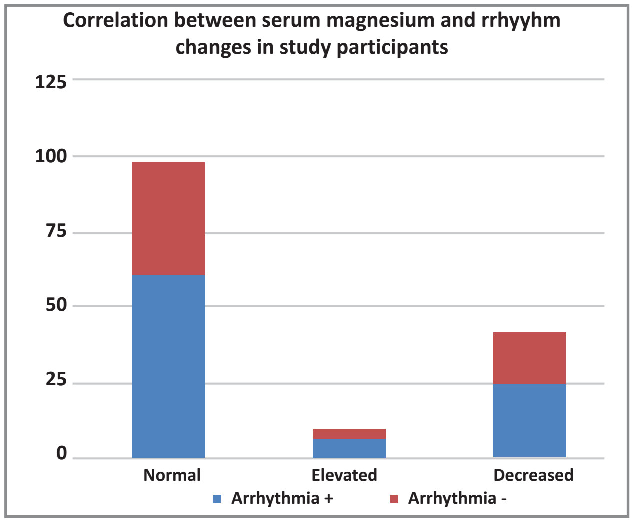

FIGURE 5. Correlation between serum magnesium and rhythm changes in study participants

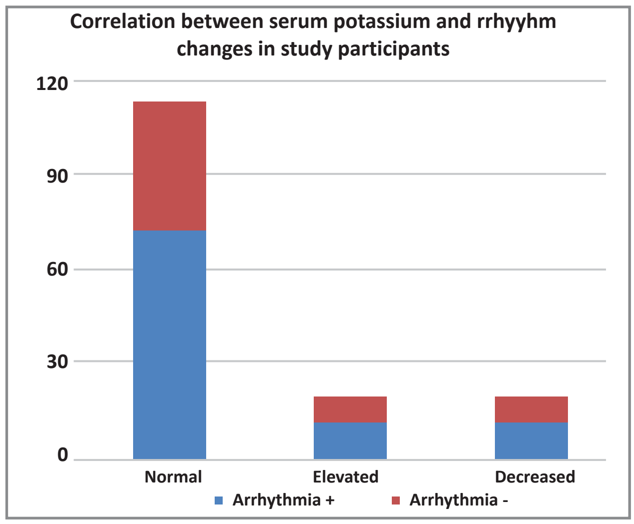

FIGURE 6. Correlation between serum potassium and rhythm changes in study participants

as either angina or myocardial infarction. Myocardial infarction (MI) is of two types based on the ECG findings, they are STEMI (ST -segment elevation MI) and NSTEMI (Non-ST-segment elevation MI). They pose a tremendous healthcare burden to the community. There are so many mortality determinants in patients with myocardial infarction. One of the significant predictors of mortality is co-existent electrolyte abnormalities such as hypomagnesemia and hypokalemia or their excess counterparts.

Hypokalemia is a dreadful condition that can cause cardiac arrest in minutes. So, monitoring potassium levels is of great concern. Hypokalemia is defined as a serum potassium level of less than 3 .5 mEq/ L. [8]. Hypokalemia is a potentially life -threatening condition. Causes range from inadequate potassium intake to increased potassium excretion.

In most cases, more than one cause may be present. It is one of the most frequently encountered electrolyte disturbances and normally, the balance between intake, distribution and excretion will determine the serum concentration. An alteration in any one of the above, results in abnormal potassium levels.Similarly, hypomagnesemia can trigger numerous arrhythmias that can be fatal [9]. Resting membrane potential is determined mainly by extracellular potassium. When serum concentration of magnesium falls below 2 Meq/ L, there is hyperpolarization of HTE membrane.

Magnesium is important for the phase 4 action potential of the ventricles. A decrease in extracellular potassium in Purkinje fibers results in increased automation. This is owed to the fact that phase 4 inflow of potassium ion is dependent on Mg ions. Often hypomagnesemia is neglected, and magnesium levels are monitored rarely in clinical settings. Compared to magnesium levels, potassium levels are observed quite frequently. In our study, most patients were above 50 years of age and had a myocardial infarction.

In the present study, 75% were males, proving that females are less prone to myocardial infarction [10]. And more than 70% of the participants had co-existing diseases like diabetes and hypertension. Regarding the potassium levels in our study participants, we found that most had normal potassium values. Only 28% of our study participants had hypokalemia. This is contrary to previous studies that say hypokalemia is highly prevalent in patients with myocardial infarction. This might be because sample collection was done immediately after admission, and that value was taken for the study. Unlike our study, some included values from different time points [11]. Some other studies assessed potassium levels at 3 - 5 days after admission, which showed hypokalemia to be highly prevalent in those patients with myocardial infarction [12]. This change in the assessment period following myocardial infarction might be responsible for the contradictory results from previous studies. In a systematic review and meta-analysis by Hoppe et al., they have found that there was a 1.6 fold increase in risk of arrhythmias with low serum potassium levels.

In the case of magnesium, as mentioned above, it is a missed cation that is most often neglected. Only in very few clinical settings is magnesium regularly monitored. In our study, none of the participants had hypomagnesemia. Some patients had hypermagnesemia, which is very few in number [13]. These results were similarly contradictory in the case of magnesium levels with most of the previous studies. In our study, most participants had normal magnesium levels; a point to be noted. Again, such a result might be due to early assessment of magnesium levels which did not reveal any change compared to the values from other studies and agreement with one study by Wang et al [14]. Most studies assessed magnesium levels a few days following admission or at different time points. Those studies have come up with different conclusions than our study. Regarding the prevalence of arrhythmias, there is a very poor correlation between potassium levels and arrhythmias. Clinically, hypokalemia is associated with torsades, polymorphic VT, Ventricular fibrillation and ectopies. This is proposed to be due to a reduction in cardiac repolarization reserve and an increase in intracellular calcium.

The majority of arrhythmias were similar in normokalemic as well as in hypokalemic individuals. This contradicts the previous studies that provide a different conclusion [15]. This might also be due to the limitation of restricting ourselves with the potassium levels on the day of admission. A similar scenario is there in the case of magnesium levels too. The prevalence is equal among people with normal magnesium levels and those with high or low magnesium levels. Again, this contradicts previous studies, which provide a different insight regarding this problem. This is again attributed to the same limitation described above for potassium. Other rules include comorbidities and independent risk factors for cardiac mortality just explained. Comorbidities were not given any emphasis in the case of analysis.

Recent studies have highlighted the significance of electrolytes in cardiovascular outcomes. Research by Casey et al. [16] indicates that hypokalemia affects 30% of MI patients and is significantly correlated with increased ventricular arrhythmia risk. This aligns with our finding that 28% of participants had hypokalemia upon admission. However, studies like Shikhooun et al. [17] showed that potassium levels can fluctuate during hospitalization, influencing arrhythmia risk at various stages.

Moreover, Cabahug et al. [18] emphasizes the under-recognized impact of hypomagnesemia on cardiac events, with 40% of MI patients with arrhythmias exhibiting low magnesium levels. This contrasts with our study, where no participants had hypomagnesemia at admission. This discrepancy may result from our focus on immediate electrolyte levels upon admission, while others measured levels at different times post-admission.

The relationship between electrolyte imbalances and arrhythmias is complex. A meta-analysis by Wu et al. [19] found that low serum potassium levels increase arrhythmia risk by 1.6 times. Despite this, our study found a weak correlation between potassium levels and arrhythmias, suggesting that early measurements may not capture the dynamic changes affecting arrhythmogenesis.

The present study limitations includes, electrolyte levels were assessed only upon admission, which may not capture fluctuations during hospitalization that could impact arrhythmia risk. Second, the predominance of male participants (75%) and the inclusion of individuals with comorbidities like diabetes and hypertension may limit the generalizability of the findings. Finally, the potential influence of confounding factors such as medication use and varying treatment protocols was not fully explored. Future research should incorporate longitudinal electrolyte monitoring and a more diverse sample to enhance the applicability of the results.

CONCLUSION

Our study suggests that monitoring potassium and magnesium might be helpful in long-term cases that require hospitalization for myocardial infarction. But in case of myocardial infarction monitoring these levels immediately after admission doesn't serve a purpose. There is little relation between the admission day electrolyte values and the development of arrhythmias. This is another new insight that is evident from our study.

Author's contributions:
Conceptualization: K.I.S.N Vaishnavi, N.Krishna Geetha, Vignesh C; Methodology: Gowri Shankar, K.I.S.N. Vaishnavi; Software: N. Krishna Geetha, K.I.S.N. Vaishnavi, Vignesh C; Validation: Gowri Shankar, N. KrishnaGeetha, Mariraj I; Formal analysis: N. Krishna Geetha, K.I.S.N. Vaishnavi; Investigation: N. Krishna Geetha, K.I.S.N. Vaishnavi, Mariraj I; Data curation: K.I.S.N. Vaishnavi, Gowri Shankar, Vignesh C; Writing-original draft preparation: N.Krishna Geetha, K.I.S.N. Vaishnavi; Writing-review and editing: N. Krishna Geetha, K.I.S.N. Vaishnavi Visualization: Gowri Shankar, K.I.S.N. Vaishnavi, Mariraj I; Project administration: K.I.S.N. Vaishnavi. All authors have read and agreed to the published version of the manuscript.

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