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SERUM ELECTROLYTE ABNORMALITIES AND THEIR ASSOCIATION WITH CARDIAC ARRHYTHMIAS IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION: A PROSPECTIVE OBSERVATIONAL STUDY

Dr Puneet Patil	Professor , Department of General Medicine,ACPM Medical College and Hospital, Dhule
Dr Manjiri Kulkarni	Professor , Department of General Medicine, B.S.P Medical College and Hospital, Chh. Sambhajinagar
Dr Ninad Patil	Assistant Professor Department of General Medicine, ACPM Medical College and Hospital, Dhule
Dr Utkarsha Sandip Deshmukh*	Junior Resident,Department of General Medicine,ACPM Medical College and Hospital,Dhule*Corresponding Author

ABSTRACT **Background:** Acute myocardial infarction (AMI) is frequently complicated by cardiac arrhythmias. Electrolyte abnormalities alter myocardial membrane potential, impulse generation and conduction, thereby increasing arrhythmogenic risk. **Objective:** To determine the frequency of serum electrolyte abnormalities and evaluate their association with cardiac arrhythmias in patients with AMI. **Methods:** This prospective observational study included 163 patients with AMI. Clinical profile, electrocardiography, echocardiography and serum sodium, potassium, calcium and magnesium were assessed. Patients were monitored throughout hospitalization for arrhythmias. **Results:** Mean age was 46.75±11.46 years and 68.71% were male. Arrhythmias occurred in 52.76%. Hyponatremia, hypokalemia, hypomagnesemia and hypocalcemia were significantly more frequent among patients with arrhythmias. Hypomagnesemia had the strongest association (OR 3.97), followed by hypocalcemia (OR 3.46), hypokalemia (OR 3.05) and hyponatremia (OR 2.89). **Conclusion**: Electrolyte abnormalities are significantly associated with arrhythmias in AMI. Routine monitoring and prompt correction may help identify high-risk patients and improve in-hospital care.

KEYWORDS : Acute myocardial infarction; cardiac arrhythmias; electrolytes; hyponatremia; hypokalemia; hypomagnesemia; hypocalcemia.

INTRODUCTION

Acute myocardial infarction remains a major cause of cardiovascular morbidity and mortality worldwide. Cardiac arrhythmias are among its most important early complications and may range from isolated premature complexes to atrial fibrillation, atrioventricular block, ventricular tachycardia and ventricular fibrillation. Their occurrence is influenced by the extent and location of ischemia, autonomic activation, reperfusion injury, left ventricular dysfunction and metabolic disturbances.

Serum electrolytes are essential determinants of myocardial excitability and conduction. Potassium influences the resting membrane potential and repolarization; both deficiency and excess may promote conduction abnormalities and malignant ventricular arrhythmias. Magnesium modulates potassium transport, calcium influx and membrane stability, and deficiency may facilitate early afterdepolarizations and ventricular ectopy. Calcium participates in excitation–contraction coupling and action-potential plateau formation, while sodium is important for rapid depolarization and reflects neurohormonal activation and disease severity in acute cardiac illness.

AMI is accompanied by catecholamine excess, activation of the renin–angiotensin–aldosterone system, intracellular electrolyte shifts, vomiting, renal dysfunction and exposure to diuretics or other therapies. These factors may produce or aggravate electrolyte disturbances. Early recognition is clinically relevant because electrolyte abnormalities are potentially modifiable contributors to arrhythmogenesis. The present study evaluated serum sodium, potassium, calcium and magnesium abnormalities and their association with cardiac arrhythmias among patients with AMI.

AIM AND OBJECTIVES

Aim: To study serum electrolyte abnormalities and their association with cardiac arrhythmias in patients with acute myocardial infarction. Objectives: (1) To describe the demographic and clinical profile of patients with AMI; (2) to determine the occurrence of cardiac arrhythmias; (3) to assess serum sodium, potassium, calcium and magnesium abnormalities; and (4) to determine the strength of association between individual electrolyte abnormalities and arrhythmias.

MATERIALS AND METHODS

Study design and setting: A prospective observational study conducted in the Department of General Medicine from 2023 to 2025. Study population: A total of 163 patients diagnosed with acute myocardial infarction were enrolled. Diagnosis was based on the clinical presentation, electrocardiographic findings and relevant cardiac biomarkers according to accepted criteria.

After informed evaluation, demographic details, presenting symptoms, cardiovascular risk factors and examination findings were recorded. All patients underwent serial electrocardiography, echocardiography and laboratory investigations. Serum sodium, potassium, total calcium and magnesium were measured. Patients were monitored throughout hospitalization for the development of cardiac arrhythmias. Arrhythmias detected clinically or on ECG/continuous monitoring were documented.

Statistical analysis: Data were analysed using SPSS version 21.0. Continuous variables were summarized as mean±standard deviation and categorical variables as number and percentage. The frequency of electrolyte abnormalities was compared between patients with and without arrhythmias. Odds ratios (OR) with 95% confidence intervals (CI) were calculated to estimate the strength of association. A p value <0.05 was considered statistically significant.

RESULTS

A total of 163 patients with AMI were included. The mean age was 46.75±11.46 years. Males constituted 68.71% of the study population. Cardiac arrhythmias occurred in 86 patients (52.76%).

Table 1. Demographic characteristics

Variable	Number	Percentage (%)
Male	112	68.71
Female	51	31.29
Mean age	46.75±11.46 years	—

The study population showed a clear male predominance, with males accounting for more than two-thirds of patients. The relatively young mean age indicates a substantial burden of AMI in middle-aged adults.

Table 2. Occurrence of cardiac arrhythmias

Arrhythmia status	Number	Percentage (%)
Present	86	52.76
Absent	77	47.24
Total	163	100.00

More than half of the patients developed an arrhythmia during hospitalization, demonstrating that rhythm disturbances were a frequent complication of AMI in this cohort.

Table 3. Electrolyte abnormalities according to arrhythmia status

Electrolyte abnormality	Arrhythmia present n (%)	Arrhythmia absent n (%)	p value
Hyponatremia	30 (34.88)	12 (15.58)	0.005
Hypokalemia	31 (36.05)	12 (15.58)	0.003
Hypomagnesemia	32 (37.21)	10 (12.99)	0.004
Hypocalcemia	27 (31.40)	9 (11.69)	0.003

All four electrolyte abnormalities were significantly more frequent in patients who developed arrhythmias. Hypomagnesemia was the most frequent abnormality in the arrhythmia group, followed by hypokalemia, hyponatremia and hypocalcemia.

Table 4. Odds-ratio analysis for association with cardiac arrhythmias

Electrolyte abnormality	Odds ratio	95% confidence interval	p value
Hyponatremia	2.89	1.34–6.23	0.005
Hypokalemia	3.05	1.42–6.55	0.003
Hypomagnesemia	3.97	1.79–8.80	0.004
Hypocalcemia	3.46	1.47–8.16	0.003

Each electrolyte abnormality significantly increased the odds of arrhythmia. Hypomagnesemia demonstrated the strongest association, with nearly fourfold higher odds, followed by hypocalcemia, hypokalemia and hyponatremia.

Table 5. Ranking of electrolyte abnormalities by strength of association

Rank	Electrolyte abnormality	Odds ratio
1	Hypomagnesemia	3.97
2	Hypocalcemia	3.46
3	Hypokalemia	3.05
4	Hyponatremia	2.89

Hypomagnesemia had the strongest association with arrhythmias, followed by hypocalcemia, hypokalemia and hyponatremia.

Table 6. Absolute difference in prevalence of electrolyte abnormalities

Electrolyte abnormality	Arrhythmia present (%)	Arrhythmia absent (%)	Absolute difference
Hyponatremia	34.88	15.58	19.30
Hypokalemia	36.05	15.58	20.47
Hypomagnesemia	37.21	12.99	24.22
Hypocalcemia	31.40	11.69	19.71

The largest absolute difference between the arrhythmia and non-arrhythmia groups was observed for hypomagnesemia, supporting its prominent association with electrical instability.

DISCUSSION

In this prospective observational study, arrhythmias occurred in 52.76% of patients with AMI. This high frequency is clinically plausible because acute ischemia alters transmembrane ionic currents, creates electrical heterogeneity and promotes re-entry, abnormal automaticity and triggered activity. Reperfusion, autonomic activation and ventricular dysfunction may further increase susceptibility.

Hyponatremia was significantly more frequent among patients with arrhythmias and was associated with 2.89-fold higher odds. Although sodium disturbance may not directly cause all rhythm abnormalities, it can reflect neurohormonal activation, hemodynamic compromise and greater illness severity. It may also coexist with other electrolyte abnormalities that influence myocardial conduction.

Hypokalemia was associated with approximately threefold higher odds of arrhythmia. Reduced extracellular potassium hyperpolarizes the cell membrane, prolongs repolarization and increases dispersion of refractoriness. These changes facilitate ectopy, early afterdepolarizations and re-entrant ventricular arrhythmias, particularly in ischemic myocardium. Careful potassium monitoring is therefore important during the acute phase of

Hypomagnesemia demonstrated the strongest association, with an OR of 3.97. Magnesium stabilizes myocardial cell membranes, modulates calcium influx and supports intracellular potassium homeostasis. Deficiency may increase ventricular excitability, prolong repolarization and promote polymorphic ventricular tachyarrhythmias. Coexisting hypokalemia may be difficult to correct until magnesium deficiency is addressed.

Hypocalcemia was also significantly associated with arrhythmias, with an OR of 3.46. Calcium has a central role in the plateau phase of the cardiac action potential and excitation–contraction coupling. Low calcium may prolong the QT interval and increase susceptibility to ventricular electrical instability. The significant associations observed across all four electrolytes support routine biochemical assessment in AMI.

Clinical implications: Electrolyte measurement is inexpensive, widely available and potentially actionable. Early detection and appropriate correction should accompany standard AMI management, particularly in patients with ventricular dysfunction, recurrent ectopy, QT prolongation or exposure to diuretics. Correction should be individualized and performed with ECG and renal-function monitoring, avoiding overcorrection.

Limitations: This was a single-centre study with a moderate sample size and no long-term follow-up. The observational design establishes association but not causation. Potential confounders such as infarct location, reperfusion status, left ventricular function, renal function and medication exposure may influence both electrolyte levels and arrhythmia risk. Larger multicentric studies with serial electrolyte measurements and detailed arrhythmia classification are warranted.

CONCLUSION

Serum electrolyte abnormalities were common among patients with acute myocardial infarction and were significantly associated with cardiac arrhythmias. Hypomagnesemia showed the strongest association, followed by hypocalcemia, hypokalemia and hyponatremia. Routine assessment, close monitoring and prompt, appropriate correction of electrolyte disturbances should form an integral component of AMI care and may assist in early identification of patients at increased arrhythmic risk.

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