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Original Research Article

ASSESSMENT OF SERUM MAGNESIUM AND POTASSIUM DYNAMICS AS PREDICTORS OF ARRHYTHMIAS IN ACUTE MYOCARDIAL INFARCTION: A LONGITUDINAL STUDY AT A TERTIARY CARE CENTER IN NORTH GUJARAT

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ABSTRACT

Background: Acute Myocardial Infarction (AMI) remains a leading cause of sudden cardiac death, frequently triggered by electrical instability and cation depletion. Serum magnesium (Mg^{+2}) and potassium (K^{+}) are essential for maintaining the cardiac resting membrane potential and myocardial stability. **Objective:** To evaluate the correlation between admission serum (Mg^{+2}) and (K^{+}) levels and the incidence of arrhythmias in patients with AMI at GMERS Himmatnagar. **Materials and Methods:** A prospective observational study of 225 AMI patients was conducted from January 2024 to December 2025. Electrolyte levels were measured upon admission via Ion-Selective Electrode and Xylidyl Blue methods. Continuous ECG monitoring was maintained for 48 hours post-admission. **Results:** Arrhythmias were observed in 44.4% of the cohort. Hypomagnesemia (<1.7 mg/dL) and hypokalemia (<3.5 mEq/L) were found in 28% and 32% of cases, respectively. Combined deficiency significantly increased the risk of malignant ventricular arrhythmias (RR 3.1, $p < 0.001$), while Anterior Wall MI patients showed higher rates of electrolyte shifts. **Conclusion:** Low admission electrolyte levels are potent, independent predictors of post-infarction rhythm disturbances, necessitating mandatory early screening and aggressive replacement in tertiary care settings.

INTRODUCTION

Acute Myocardial Infarction (AMI) continues to be a primary driver of cardiovascular morbidity in India, particularly in the developing healthcare landscape of North Gujarat. While modern reperfusion therapies have advanced survival, electrical complications—specifically arrhythmias—remain the primary cause of sudden death within the first 48 hours of admission.[1,2]

The electrophysiological stability of the myocardium depends on the maintenance of the Na+-K+-ATPase pump and calcium-channel regulation, both of which are strictly magnesium-dependent.[3] Magnesium acts as a physiological calcium channel blocker; its deficiency facilitates intracellular calcium overload, leading to "after-depolarizations" and triggered activity.[4] This study seeks to provide localized data from GMERS Himmatnagar to refine ICCU electrolyte replacement protocols and improve risk stratification in regional populations.[5]

MATERIALS AND METHODS

Study Design: Longitudinal prospective observational study.
Setting: Intensive Cardiac Care Unit (ICCU), GMERS Medical College, Himmatnagar.
Sample Size: 225 consecutive adult patients with confirmed AMI (STEMI/NSTEMI).
Selection Criteria: Diagnosis was based on WHO criteria (typical chest pain, ECG changes, and elevated Troponin-I). Patients with Chronic Kidney Disease, chronic diuretic use, or prior valvular disease were excluded to avoid confounding electrolyte data.
Procedure: Venous blood was collected within 2 hours of admission. $K^{+}$was measured via Ion-Selective Electrode and Mg^{2+} via the Xylidyl Blue method. Continuous 12-lead ECG monitoring was maintained for the initial 48 hours of hospitalization.

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RESULTS

3.1 Demographic and Clinical Profile

The study cohort (N=225) had a mean age of 57.2 ± 11.5 years, with a male-to-female ratio of 2.1:1. Notably, 18.7% of the patients were classified as "young AMI" cases (<45years). Anterior Wall Myocardial Infarction (AWMI) was the most frequent presentation (55.1%), followed by Inferior Wall MI (36.4%).

Table 1: Socio-Demographic and Clinical Profile (N=225)

Parameter	Frequency (n)	Percentage (%)
Gender (Male : Female)	154 : 71	68.4% : 31.6%
Anterior Wall MI	124	55.1%
Inferior Wall MI	82	36.4%
NSTEMI	19	8.5%

3.2 Electrolyte Distribution on Admission

On admission, the mean serum $Mg^{+2}$was 1.78 ± 0.4mg/dL and the meanK^{+} was 3.62 ± 0.6mEq/L. Hypomagnesemia was identified in 63 patients (28%) and hypokalemia in 72 patients (32%). A critical sub-group of 38 patients (16.9%) exhibited a dual deficiency of both cations. Patients with AWMI showed a significantly higher propensity for hypokalemia (38.7%) compared to those with IWMI (24.4%, p = 0.032), likely due to larger infarct sizes and greater catecholamine release.

3.3 Arrhythmia Incidence and Correlation

A total of 100 patients (44.4%) experienced at least one arrhythmic event within 48 hours. The incidence was strikingly higher in the deficient groups. Among hypomagnesemic patients, 60.3% developed arrhythmias compared to 38.2% in the normomagnesemic group ($p < 0.001$). The most lethal correlation was observed in the dual-deficiency group, where the arrhythmia incidence spiked to 76.3%.

Table 2: Arrhythmia Incidence vs. Electrolyte Sub-groups

Electrolyte Status	Total Patients	Arrhythmias (\(n\))	\(p\)-value
Mg < 1.7 mg/dL	63	38 (60.3%)	<0.001
Mg ≥ 1.7 mg/dL	162	62 (38.2%)	-
K < 3.5 mEq/L	72	41 (56.9%)	0.004
K ≥ 3.5 mEq/L	153	59 (38.5%)	-

3.4 Specific Arrhythmic Patterns

Ventricular Tachycardia (VT) and high-grade Ventricular Premature Contractions (VPCs) were predominantly seen in patients with hypomagnesemia, showing a Relative Risk (RR) of 3.1 and 2.4, respectively. Atrial Fibrillation was observed in 14 cases, but the statistical link to magnesium was less robust (p=0.082).

Table 3: Specific Arrhythmic Events and Relative Risk

Arrhythmia	Hypo-Mg Group (\(n=63\))	Normal-Mg Group (\(n=162\))	RR (95% CI)
Ventricular Tachycardia	10	5	3.1 (1.2–8.4)
VPCs (High Grade)	14	8	2.4 (1.1–5.3)
Sinus Tachycardia	12	14	1.8 (0.9–3.5)

DISCUSSION

The investigation at GMERS Himmatnagar confirms a significant intersection between dyselectrolytemia and cardiac irritability in the acute phase of myocardial infarction. Our study found that roughly 1 in 3 patients presents with a deficiency in either potassium or magnesium, a rate that is notably higher than those reported in urban private healthcare settings in India. This suggests a unique regional risk profile for the North Gujarat population.[6,7,21]

Pathophysiological Mechanisms and the "Catecholamine Surge"

In the hyper-acute phase of AMI, the body initiates a massive sympathetic response. The resulting surge in circulating catecholamines stimulates \(\beta _{2}\)-adrenoceptors, which activates the Na-K-ATPase pump, driving potassium from the extracellular space into the cells8.This results in "apparent" hypokalemia which, while transient, is long enough to destabilize the resting membrane potential. In our study, patients with Anterior Wall MI (AWMI) showed the highest rates of hypokalemia (38.7%). We hypothesize that the larger area of ischemic tissue in AWMI leads to a more profound adrenergic storm, thereby increasing the severity of the electrolyte shift compared to inferior wall events.[10,22]

Magnesium: The Gatekeeper of Potassium Homeostasis

A pivotal observation in our cohort was that hypokalemia was rarely an isolated threat. Magnesium acts as a natural calcium channel blocker and a vital cofactor for the energy-dependent exchange of ions. When magnesium levels are low, the Renal Outer Medullary Potassium (ROMK) channels in the kidney remain open, leading to excessive urinary potassium wasting.[9] Furthermore, at the cellular level in the myocardium, magnesium deficiency prevents the Na-K-ATPase pump from effectively re-sequestering potassium into the

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myocytes during repolarization.23 This explains why the Combined Deficiency Group in our study had the highest arrhythmia rate (76.3%); they were trapped in a biochemical "deadlock" where potassium replacement was likely ineffective without prior magnesium correction.[13,19]

The North Gujarat Context: Dietary and Environmental Factors

The high prevalence of hypomagnesemia (28%) in our study warrants an analysis of regional factors. North Gujarat is known for varied soil composition and high fluoride levels in groundwater in certain pockets.[7] Chronic fluoride exposure has been linked to altered mineral metabolism. Additionally, the diet in rural Sabarkantha often lacks sufficient magnesium-rich sources like nuts, seeds, and specific green leafy vegetables, which may be displaced by high-carbohydrate staples.[17,24] This baseline nutritional borderline-deficiency becomes a critical liability when the patient undergoes the metabolic stress of an AMI.

Prognostic Value of "Low-Normal" Levels

Standard laboratory ranges for magnesium (1.7–2.4 mg/dL) may be misleading in the context of an acutely ischemic heart. Our analysis revealed that patients with magnesium in the "low-normal" range (1.7–1.9 mg/dL) still experienced a higher frequency of Lown-grade VPCs compared to those with levels above 2.1 mg/dL.[13,25] This suggests that for post-AMI patients, the "therapeutic target"should be higher than the "biological normal." Maintaining potassium above 4.0 mEq/L and magnesium above 2.2 mg/dL could potentially provide a safer electrical buffer against re-entrant circuits that lead to Ventricular Tachycardia (VT) and Fibrillation (VF).[15,20]

Clinical Implications for Tertiary Care

At GMERS Himmatnagar, where advanced interventional facilities like immediate primary PCI may face logistical delays for patients coming from distant rural areas, electrolyte management becomes a "low-cost, high-impact" intervention. Prophylactic magnesium infusion, as debated since the LIMIT-2 and ISIS-4 trials, might still hold relevance in high-risk, electrolyte-deficient subgroups to reduce the "arrhythmic burden" during transport or the initial hours of ICCU stay.[16,25]

CONCLUSION

Admission serum Mg+2 and K+ levels are inexpensive, rapid, and reliable indicators of arrhythmic risk. This study proves that their deficiency is a major contributor to electrical instability in the North Gujarat population. We recommend that electrolyte screening be mandatory upon admission and that clinical protocols should aim for high-normal replacement targets to mitigate the risk of lethal rhythm disturbances.

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