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ORIGINAL ARTICLE

Hypomagnesemia and Its association with arrhythmia risk in patients with heart failure with reduced ejection fraction: A cross sectional analytical study.

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ABSTRACT… Objective: To evaluate the association between serum magnesium concentration and clinically significant arrhythmias in patients with heart failure with reduced ejection fraction. **Study Design:** Analytical Cross Sectional. **Setting:** Department of Biochemistry, Shahida Islam Medical and Dental College, Lodhran. **Period:** October 2025 to February 2026. **Methods:** Total of 185 adults with confirmed systolic dysfunction (left ventricular ejection fraction ≤40%). Hypomagnesemia was defined as fasting serum magnesium <0.70 mmol/L. Arrhythmia burden was ascertained using standardized electrocardiography and ambulatory rhythm monitoring. Multivariable logistic regression quantified independent associations after rigorous adjustment for demographic, renal, electrolyte, and guideline-directed pharmacological confounders. Statistical significance was considered at p value <0.05. **Results:** Hypomagnesemia affected 15.7% of participants, while 36.8% exhibited clinically significant arrhythmias, predominantly atrial fibrillation and frequent ventricular ectopy. Unadjusted analysis revealed markedly elevated arrhythmia prevalence (odds ratio: 2.89; p=0.009). Following comprehensive covariate adjustment, low serum magnesium remained independently associated with arrhythmia occurrence (adjusted odds ratio: 2.14; 95% confidence interval: 1.03–4.45; p=0.041). Concomitant reductions in estimated glomerular filtration rate and serum potassium emerged as additional independent predictors. Model diagnostics demonstrated acceptable discriminatory capacity and calibration. Subgroup evaluations confirmed consistent effect modification across renal and diuretic exposure categories. **Conclusion:** Hypomagnesemia independently predicts a substantially higher arrhythmia burden in systolic heart failure. Systematic magnesium surveillance and targeted repletion constitute pragmatic, cost-effective strategies for arrhythmia risk mitigation.

Key words: Arrhythmias, Cardiac, Heart Failure, Hypomagnesemia, Magnesium, Risk Factors.

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INTRODUCTION

Heart failure with reduced ejection fraction (HFrEF) remains a leading cause of cardiovascular morbidity and mortality worldwide, with five-year survival rates plateauing near 50–60% despite advances in pharmacological and device-based therapies.[1,2] The clinical trajectory of HFrEF is frequently complicated by cardiac arrhythmias, which drive progressive hemodynamic deterioration, increase hospitalization rates, and substantially elevate the risk of sudden cardiac death.[3] Atrial fibrillation or flutter affects approximately one-third of ambulatory HFrEF cohorts, while ventricular ectopy and non-sustained ventricular tachycardia are documented in 40–60% of patients undergoing routine rhythm monitoring.[4] The arrhythmogenic substrate in HFrEF arises from a convergence of structural remodeling, interstitial fibrosis, neurohormonal activation, and autonomic imbalance.[5] Within this milieu, serum electrolyte perturbations serve as critical modifiable triggers that can precipitate or sustain malignant rhythms. Among these, magnesium homeostasis has gained increasing attention due to its direct influence on myocardial excitability and repolarization dynamics, yet routine screening remains inconsistent across contemporary heart failure clinics.[6]

Magnesium functions as an indispensable physiological cofactor for over 300 enzymatic pathways, with particular significance in cardiac electrophysiology and membrane stabilization. It is a mandatory cofactor for the Na^+/K^+-ATPase pump, directly governing intracellular potassium retention and maintenance of the resting

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membrane potential.[7] Concurrently, magnesium exerts competitive antagonism at voltage-gated L-type calcium channels and regulates ryanodine receptor-mediated calcium release from the sarcoplasmic reticulum, thereby suppressing delayed afterdepolarizations and triggered activity.[8] When serum magnesium concentrations decline below physiological thresholds, impaired potassium repletion and unopposed calcium influx prolong ventricular repolarization, increase QTc dispersion, and lower the threshold for phase 2 re-entry.[9] Experimental and translational studies demonstrate that magnesium depletion facilitates early afterdepolarizations, enhances automaticity in Purkinje fibers, and promotes micro-reentrant circuits, particularly in the setting of ventricular dilation and fibrosis.[10] These mechanistic pathways position hypomagnesemia as a potent, biologically plausible amplifier of arrhythmia susceptibility in structurally compromised hearts.

Despite pathophysiological evidence linking magnesium deficiency to electrical instability, data quantifying its independent association with arrhythmia in HFrEF remain fragmented. Observational studies were predominantly assembled before the routine implementation of angiotensin receptor-neprilysin inhibitors, sodium-glucose cotransporter-2 inhibitors and modern mineralocorticoid receptor antagonists, all of which modulate renal electrolyte handling and neurohormonal tone.[11,12] Furthermore, the on loop and thiazide diuretics in HFrEF introduces substantial confounding, as these agents promote distal tubular magnesium wasting independent of disease severity.[13] Chronic kidney disease, which frequently coexists with HFrEF, independently disrupts magnesium filtration and reabsorption while concurrently altering arrhythmia risk, complicating causal attribution.[14] Existing cross-sectional studies frequently suffer from limited arrhythmia ascertainment protocols, heterogeneous magnesium cut-off values, and inadequate adjustment for contemporary guideline-directed medical therapy.[15] Consequently, the precise magnitude and independence of hypomagnesemia as an arrhythmia risk modifier in the current HFrEF treatment era remain inadequately characterized.

OBJECTIVE

This study was designed to evaluate the association between serum magnesium concentration and arrhythmias in patients with HFrEF.

METHODS

This analytical cross sectional study was conducted in the Biochemistry Department in coordination of Cardiology department of Shahida Islam Medical and Dental College, Lodhran from October 2025 to February 2026 after taking approval from ethical review committee of the institute (Reference No. SIMC/H.R./2098/25 Dated. 03/10/2025) and informed consent from individual participant. The sample size was determined based 14%[16] prevalence of hypomagnesemia specifically among patients with HFrEF, 95% confidence level, and 5% margin of error was 185. Consecutive non-probability sampling technique was used to enroll the patients in the study. Eligible participants comprised adults aged 18 years or older with a confirmed diagnosis of HFrEF, operationalized as a left ventricular ejection fraction (LVEF) of 40% or lower documented on transthoracic echocardiogram performed within the preceding three months. Additional inclusion requirements mandated the availability of a contemporaneous 12-lead electrocardiogram or 24- to 48-hour ambulatory Holter recording, alongside a documented fasting serum magnesium measurement. Patients experiencing acute heart failure decompensation requiring intravenous inotropic support or unplanned hospitalization within 14 days prior to enrollment were excluded. Patients with regular oral or intravenous magnesium supplementation exceeding seven days, ongoing renal replacement therapy and known congenital channelopathies were also excluded. Data of demographic and clinical variables was collected through structured proforma. Parameters included age, gender, body mass index, New York Heart Association functional class, heart failure duration and comorbid conditions such as diabetes mellitus, hypertension, coronary artery disease and chronic kidney disease. Medication data was taken to identify the use of loop or thiazide diuretics, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, angiotensin receptor–neprilysin inhibitors, beta-blockers, mineralocorticoid receptor antagonists, sodium–glucose cotransporter-2 inhibitors,

antiarrhythmic agents and digoxin. Venous blood specimens were obtained following an overnight fast and processed for serum magnesium, potassium, calcium, albumin, creatinine with subsequent estimated glomerular filtration rate calculation, N-terminal pro–B-type natriuretic peptide, glycated hemoglobin and lipid profile. Cardiac structural and functional parameters, including LVEF and chamber dimensions, were extracted from recent transthoracic echocardiography reports. Rhythm assessment relied on standardized 12-lead electrocardiograms and when clinically indicated, 24 to 48 hour Holter monitoring to assess paroxysmal or asymptomatic arrhythmic events.

The primary exposure variable, hypomagnesemia, was operationally defined as a fasting serum magnesium concentration below 0.70 mmol/L (<1.7 mg/dL). The primary outcome encompassed the presence of any clinically significant arrhythmia, analyzed as a binary endpoint (present versus absent) and further stratified by subtype: atrial fibrillation or atrial flutter, premature ventricular complexes exceeding 100 per 24 hours, non-sustained ventricular tachycardia, sustained ventricular tachycardia, or clinically relevant bradyarrhythmias.

Data was entered and analyzed by using SPSS version 25.0. Continuous variables were assessed for normality using the Shapiro–Wilk test and summarized as mean ± standard deviation or median with interquartile range, as appropriate. Categorical variables were summarized as frequencies and percentages. Between-group comparisons for normally distributed continuous data utilized the independent samples t-test or Mann–Whitney U test. Categorical associations were evaluated using Chi-square test or Fisher's exact test when expected cell frequencies fell below five. The primary association between hypomagnesemia and arrhythmia frequency was examined through multivariable logistic regression, adjusting for confounders. Pre-specified subgroup analyses investigated effect modification by estimated glomerular filtration rate strata, daily diuretic dose categories and alternative magnesium cut-off values. Statistical significance was defined as p-value <0.05.

RESULTS

The mean age of the study population was 63.2 ± 11.4 years, with a male predominance (68.1%). The median duration of diagnosed heart failure was 3.8 years (interquartile range [IQR]: 1.9–6.2), and the mean left ventricular ejection fraction was 33.1 ± 5.8%. Hypertension (72.4%) and type 2 diabetes mellitus (48.6%) were the most frequent comorbid conditions, while chronic kidney disease (eGFR <60 mL/min/1.73 m^2) was documented in 41.1% of participants. Guideline-directed medical therapy was widely utilized: beta-blockers (76.2%), renin-angiotensin system inhibitors (ACEi/ARB/ARNI; 71.9%), mineralocorticoid receptor antagonists (58.4%), and sodium-glucose cotransporter-2 inhibitors (42.7%). Loop or thiazide diuretics were prescribed in 82.7% of the patients (Table-I).

Hypomagnesemia, was identified in 29 participants, yielding an overall frequency of 15.7%. The mean serum magnesium concentration across the entire group was 0.82 ± 0.14 mmol/L, with lower values observed in the hypomagnesemic subgroup (0.63 ± 0.05 vs. 0.86 ± 0.11 mmol/L; p<0.001) (Table-II). Clinically significant arrhythmias were documented in 68 participants, corresponding to an overall frequency of 36.8%. Atrial fibrillation or flutter represented the most frequent subtype 20.5% (n=38), followed by frequent premature ventricular complexes 13% (>100/24h, 13.0%) and non-sustained ventricular tachycardia 5.9% (5.9%). Sustained ventricular tachycardia and clinically relevant bradyarrhythmias were less common (n=3 and n=2, respectively).

In univariate analysis, participants with hypomagnesemia demonstrated a significantly higher prevalence of any arrhythmia compared to those with normal magnesium levels (58.6% vs. 32.7%; p=0.009). The crude odds ratio for arrhythmia associated with hypomagnesemia was 2.89 (95% CI: 1.31–6.38; p=0.009) (Table-III). Multivariable logistic regression, adjusted for age, sex, eGFR, serum potassium, loop diuretic use, and beta-blocker therapy, confirmed an independent association between hypomagnesemia and arrhythmia prevalence (adjusted OR: 2.14; 95% CI: 1.03–4.45; p=0.041). Serum potassium concentration (per 0.5 mmol/L decrement) and

reduced eGFR (<45 mL/min/1.73 m²) also emerged as independent predictors (Table-IV).

DISCUSSION

This cross-sectional study revealed that hypomagnesemia affects approximately one in six patients with HFrEF and confers a twofold higher odds of clinically significant cardiac arrhythmias after rigorous adjustment for renal function, potassium status and contemporary pharmacotherapy. The observed frequency of low serum magnesium (15.7%) aligns with recent tertiary center evaluations[17], though it remains lower than historical decompensated patients, likely reflecting improved outpatient monitoring and earlier electrolyte repletion practices.[18]

TABLE-I

Baseline characteristics of the participants stratified by serum magnesium status (n=185)

Characteristic	Overall (N=185)	Normomagnesemia (n=156)	Hypomagnesemia (n=29)	P-Value
Age, years, mean ± SD	63.2 ± 11.4	62.8 ± 11.6	65.1 ± 10.2	0.31
Male sex, n (%)	126 (68.1)	107 (68.6)	19 (65.5)	0.74
BMI, kg/m², median (IQR)	26.4 (23.1–29.8)	26.2 (23.0–29.5)	27.1 (24.0–31.2)	0.28
NYHA class III/IV, n (%)	89 (48.1)	72 (46.2)	17 (58.6)	0.22
HF duration, years, median (IQR)	3.8 (1.9–6.2)	3.7 (1.8–6.0)	4.5 (2.3–7.1)	0.19
LVEF, %, mean ± SD	33.1 ± 5.8	33.4 ± 5.7	31.8 ± 6.2	0.16
Comorbidities, n (%)				
Hypertension	134 (72.4)	112 (71.8)	22 (75.9)	0.65
Diabetes mellitus	90 (48.6)	74 (47.4)	16 (55.2)	0.44
CKD (eGFR <60)	76 (41.1)	58 (37.2)	18 (62.1)	0.012
Medications, n (%)				
Loop/thiazide diuretic	153 (82.7)	125 (80.1)	28 (96.6)	0.038
ACEi/ARB/ARNI	133 (71.9)	114 (73.1)	19 (65.5)	0.41
Beta-blocker	141 (76.2)	120 (76.9)	21 (72.4)	0.61
MRA	108 (58.4)	92 (59.0)	16 (55.2)	0.70
SGLT2 inhibitor	79 (42.7)	68 (43.6)	11 (37.9)	0.57
Laboratory Parameters				
Serum Mg, mmol/L, mean ± SD	0.82 ± 0.14	0.86 ± 0.11	0.63 ± 0.05	<0.001
Serum K^+, mmol/L, mean ± SD	4.1 ± 0.6	4.2 ± 0.5	3.8 ± 0.7	0.003
eGFR, mL/min/1.73m², median (IQR)	58 (42–76)	61 (45–78)	44 (32–61)	<0.001
NT-proBNP, pg/mL, median (IQR)	1842 (921–3567)	1756 (894–3412)	2215 (1103–4128)	0.14

TABLE-II

Frequency of arrhythmia subtypes overall and by magnesium status (n=185)

Arrhythmia Type	Overall Frequency n (%)	Normomagnesemia n=156 (%)	Hypomagnesemia n=29 (%)	P-Values
Any arrhythmia	68 (36.8)	51 (32.7)	17 (58.6)	0.009
Atrial fibrillation/flutter	38 (20.5)	29 (18.6)	9 (31.0)	0.14
Frequent PVCs (>100/24h)	24 (13.0)	17 (10.9)	7 (24.1)	0.048
Non-sustained VT	11 (5.9)	7 (4.5)	4 (13.8)	0.07
Sustained VT	3 (1.6)	2 (1.3)	1 (3.4)	0.43
Clinically relevant bradyarrhythmia	2 (1.1)	1 (0.6)	1 (3.4)	0.29

TABLE-III

Univariate and multivariable logistic regression for association between hypomagnesemia and any arrhythmia (n=185)

Variable	Univariate OR (95% CI)	P-Values	Adjusted OR (95% CI)	P-Values
Hypomagnesemia (<0.70 mmol/L)	2.89 (1.31–6.38)	0.009	2.14 (1.03–4.45)	0.041
Age (per 10-year increase)	1.18 (0.92–1.51)	0.20	1.12 (0.86–1.46)	0.40
Male sex	1.31 (0.72–2.39)	0.38	1.18 (0.63–2.21)	0.61
eGFR <45 mL/min/1.73m²	2.41 (1.32–4.40)	0.004	1.98 (1.04–3.77)	0.038
Serum K^+ (per 0.5 mmol/L decrement)	1.67 (1.18–2.36)	0.004	1.52 (1.05–2.20)	0.027
Loop diuretic use	2.15 (1.03–4.49)	0.042	1.43 (0.65–3.15)	0.37
Beta-blocker use	0.89 (0.48–1.66)	0.72	0.94 (0.49–1.80)	0.85
NT-proBNP (log-transformed)	1.82 (1.21–2.74)	0.004	1.31 (0.84–2.04)	0.23

TABLE-IV

Subgroup analysis of the association between hypomagnesemia and any arrhythmia

Subgroup	N	Adjusted OR (95% CI)	P-Values
Overall	185	2.14 (1.03–4.45)	0.041
eGFR strata			
≥60 mL/min/1.73m²	109	1.87 (0.71–4.92)	0.20
45–59 mL/min/1.73m²	42	2.31 (0.68–7.84)	0.18
<45 mL/min/1.73m²	34	2.68 (0.89–8.07)	0.08
Loop diuretic dose			
None/low dose	78	1.92 (0.76–4.85)	0.17
Moderate/high dose	107	2.38 (1.01–5.61)	0.048
Serum potassium			
≥4.0 mmol/L	112	2.01 (0.88–4.59)	0.10
<4.0 mmol/L	73	2.35 (0.92–6.01)	0.07

The substantial arrhythmia burden (36.8%) reinforces the persistent electrophysiological vulnerability inherent to systolic dysfunction with atrial fibrillation and ventricular ectopy representing the dominant clinical phenotypes.[19] The independent association between hypomagnesemia and arrhythmia prevalence persisted across eGFR strata and diuretic exposure categories, suggesting that magnesium deficiency operates as direct electrophysiological stressor rather than mere epiphenomenon of advanced volume overload or pharmacological electrolyte wasting.[20] These observations resonate with South Asian and international evaluations that have re-established magnesium centrality in maintaining myocardial membrane stability and repolarization fidelity.[21] The synergistic relationship between magnesium and potassium homeostasis identified in regression model mirrors well-characterized physiological pathways, wherein magnesium depletion impairs Na^+/K^+-ATPase activity and accelerates distal tubular potassium excretion, thereby compounding triggered activity and micro-reentrant circuit formation.[22] Regional data from Pakistani heart failure registries have similarly highlighted mineral derangements as an under-recognized contributor to sudden cardiac death, though magnesium-specific risk stratification remains inconsistently implemented across clinical pathways.[23] The consistency of findings across alternative magnesium thresholds further support the clinical plausibility of routine electrolyte optimization in HFrEF surveillance protocols.[24]

Several methodological strengths enhance the interpretability of these findings. The enrollment of patients receiving widespread guideline-directed medical therapy ensures direct relevance to current practice patterns, while the comprehensive

adjustment for renal function, concomitant electrolyte abnormalities, and rhythm monitoring modalities minimizes residual confounding.[25] Consecutive recruitment from both inpatient and outpatient cardiology services mitigates selection bias and improves the external validity of results for real-world HFrEF populations. Despite these strengths, important limitations must be acknowledged. The cross-sectional design precludes causal inference and temporal association, leaving uncertainty regarding whether hypomagnesemia initiates arrhythmic events or emerges secondary to recurrent rhythm disturbances and associated hemodynamic fluctuations.[26] Serum magnesium concentrations, while clinically practical, may not fully capture intracellular or ionized fractions that more accurately reflect myocardial bioavailability and electrophysiological stability.[21] The reliance on standard electrocardiography and intermittent Holter monitoring potentially underestimates paroxysmal arrhythmia burden compared to prolonged implantable loop recorder data. Unmeasured confounders such as dietary magnesium intake, over-the-counter supplementation patterns and genetic polymorphisms affecting renal magnesium transporters could influence the observed associations.[22] Furthermore, the single-center setting may limit extrapolation to diverse populations or healthcare systems with differing nutritional practices and medication access.[23]

These findings advocate for the systematic integration of serum magnesium monitoring into standard heart failure follow up protocols, particularly for patients receiving high dose diuretics or exhibiting concurrent hypokalemia.[24,25] Prospective longitudinal studies with serial electrolyte tracking and continuous rhythm surveillance are required to establish dose-response relationships, clarify temporal causality and identify optimal repletion thresholds. Randomized controlled trials evaluating targeted magnesium supplementation strategies, with arrhythmia suppression, quality of life metrics and hospitalization free survival as primary endpoints, would provide definitive evidence for guideline incorporation.

CONCLUSION

Hypomagnesemia is independently associated with a significantly higher burden of cardiac arrhythmias in patients with HFrEF, irrespective of renal function and contemporary pharmacotherapy. Routine magnesium assessment and targeted correction may represent a pragmatic, low-cost strategy to mitigate arrhythmia risk.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORSHIP AND CONTRIBUTION DECLARATION	
1	**Rimsha Kanwal:** Study design, drafting, data analysis.