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Serum magnesium levels and the risk of acute viral cardiomyopathy in children: A case-control study

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Abstract

BACKGROUND: Acute viral cardiomyopathy is a leading cause of heart failure in children. Although magnesium is essential for myocardial electrophysiology, its potential association with pediatric viral cardiomyopathy remains poorly characterized.

METHODS: This hospital-based frequency-matched case–control study (1:1 ratio, balanced for age) included 73 children with confirmed acute viral cardiomyopathy and 73 non-cardiac hospitalized controls at Afzalipour Hospital, Kerman, Iran (2023–2024). Serum total magnesium was measured on admission (reference 1.7–2.3 mg/dL); hypomagnesemia was defined as <1.7 mg/dL. Groups were compared using an independent-samples t-test and a χ^2 test. Multivariable logistic regression has been used to assess associated factors for acute viral cardiomyopathy.

RESULTS: Mean serum magnesium level was significantly lower in the cardiomyopathy group than the control group (1.80 ± 0.31 vs. 1.98 ± 0.31 mg/dL; P = 0.003). Hypomagnesemia prevalence was significantly higher in the cardiomyopathy group (39.7% vs. 9.6%; unadjusted OR (95% CI): 5.80 (2.28–14.75); P < 0.001). After adjustment for sex, BMI, fever, and underlying chronic disease, each 0.1 mg/dL decrease in serum magnesium was associated with 1.38-fold higher odds of cardiomyopathy (adjusted OR (95% CI): 1.38 (1.11–1.72); P = 0.004). Hospital stay was longer in cases (7.77 ± 2.42 vs. 5.39 ± 2.20 days; P < 0.001). In-hospital mortality was higher among the cardiomyopathy group (6.8% vs. 1.4%), though the difference was not statistically significant (P = 0.209).

CONCLUSION: Children with acute viral cardiomyopathy seem to have significantly lower serum magnesium compared with matched controls. Routine admission magnesium screening could be considered in management protocols.

Keywords: Acute Viral Cardiomyopathy; Hypomagnesemia; Magnesium; Pediatrics; Cardiology

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Introduction

Acute viral cardiomyopathy, encompassing viral myocarditis and related inflammatory cardiac syndromes, remains one of the most serious and unpredictably acquired heart diseases in childhood. Despite its relatively low incidence (estimated 1–2 cases per 100,000 children annually), it accounts for a disproportionate share of pediatric acute heart failure, sudden cardiac death, and the need for heart transplantation[1–3]. The spectrum ranges from subclinical myocardial involvement to fulminant cardiogenic shock, with in-hospital mortality historically reported between 5–10% even in contemporary cohorts[4–6].

Electrolyte disturbances are frequently observed in critically ill children and are associated with increased morbidity. Among these, hypomagnesemia has attracted particular attention because magnesium is a critical cofactor for hundreds of enzymatic reactions, plays a pivotal role in maintaining membrane stability, and acts as a physiological calcium antagonist in cardiomyocytes[7–9]. Magnesium (Mg) is a critical intracellular cation involved in myocardial contraction, electrophysiologic stability, vascular tone regulation, and modulation of inflammatory pathways. Disturbances in magnesium homeostasis, particularly hypomagnesemia, have been frequently reported in critically ill children and have been linked to arrhythmias, prolonged hospitalization, ventricular dysfunction, and increased mortality. Previous research has demonstrated higher rates of hypomagnesemia in pediatric intensive care units and in children with severe infections or cardiac disorders. However, data specifically focusing on serum magnesium status in children with acute viral cardiomyopathy remain scarce.

Low serum magnesium has been independently linked to prolonged mechanical ventilation, higher Pediatric Risk of Mortality (PRISM) scores, arrhythmia occurrence, and mortality in pediatric intensive care settings[10–12]. Moreover, magnesium modulates inflammatory pathways; reduced levels are associated with elevated C-reactive protein and pro-inflammatory cytokines, whereas supplementation can attenuate excessive inflammation[13–15].

In the context of viral myocardial injury, several mechanisms may precipitate or exacerbate hypomagnesemia: a systemic inflammatory response with cytokine-driven renal wasting, redistribution into inflamed tissue, reduced gastrointestinal absorption during critical illness, and a catecholamine surge that promotes an intracellular magnesium shift[16–18]. Despite these plausible pathophysiological links, evidence specifically addressing magnesium status in pediatric acute viral cardiomyopathy is surprisingly scarce. Most existing data derive from heterogeneous PICU populations or from the multisystem inflammatory syndrome in children (MIS-C) era, where hypomagnesemia prevalence varied widely (20–45%) but was rarely the primary focus[19–22]. No previous study, to our knowledge, has systematically compared serum magnesium levels and hypomagnesemia prevalence between children with confirmed acute viral cardiomyopathy and age-matched controls hospitalized for non-cardiac conditions.

The present study was therefore conducted to evaluate serum magnesium levels and the prevalence of hypomagnesemia in children hospitalized with acute viral cardiomyopathy compared with matched non-cardiac controls in a tertiary pediatric center in southeast Iran.

Materials and Methods

Study Design and Setting

This was a hospital-based, frequency-matched case-control study with a 1:1 matching ratio by age. The study was conducted at Afzalipour Hospital, Kerman University of Medical Sciences, Kerman, Iran, a tertiary referral center with a dedicated pediatric cardiology and pediatric intensive care unit (PICU). Data were collected retrospectively from medical records of eligible patients admitted between April 2023 and March 2024. Data extraction commenced in late September 2025 (following ethics committee approval).

Participants

- **Cases:** All children aged 0–18 years hospitalised during the study period with a confirmed diagnosis of acute viral cardiomyopathy (including fulminant myocarditis, acute myocarditis with significant left ventricular dysfunction, and viral-associated dilated cardiomyopathy)[23]. Diagnosis was established by the treating pediatric cardiologist according to contemporary guidelines and required at least two of the following: – Clinical presentation suggestive of myocarditis / acute heart failure – Elevated cardiac biomarkers (troponin I / CK-MB) – Echocardiographic evidence of new-onset left ventricular systolic dysfunction (ejection fraction <55% and/or fractional shortening <28%) – Exclusion of other causes (congenital heart disease, rheumatic, toxic, metabolic). Viral etiology was presumed based on preceding or concurrent viral illness, positive viral serology/ PCR when available, and absence of bacterial or other identifiable causes.
- **Controls:** Children hospitalized during the same period for non-cardiac reasons (respiratory infections, gastroenteritis, neurological disorders, trauma, etc.) who had no history or evidence of structural or functional heart disease. Controls were frequency-matched to cases in a 1:1 ratio based on age group (within ±12 months).

Exclusion Criteria (applied to both groups)

Patients were excluded if they had chronic kidney disease or acute kidney injury known to affect magnesium homeostasis, documented intestinal malabsorption syndromes or chronic diarrhea, receipt of oral magnesium-containing supplements or intravenous magnesium sulphate within the week before admission, maternal administration of magnesium sulphate for preterm labour prevention, or missing or incomplete documentation of serum magnesium level measured on admission. A STROBE flow diagram is presented in Figure 1.

Data Collection and Magnesium Measurement

Demographic (age, sex), anthropometric (weight, height, body mass index [BMI]), and clinical variables, including the presence of fever ≥38 °C on admission, underlying chronic disease (such as chronic respiratory disorders [e.g., asthma], neurological disorders, hematologic diseases, primary immunodeficiency, and other physician-diagnosed chronic conditions), duration of hospital stay, and in-hospital outcome were extracted from patients' medical records. Serum total magnesium was routinely measured serum total magnesium levels measured on admission or within the first 24 hours of hospital admission (as part of a standard PICU admission laboratory protocol) using the colorimetric xylidyl blue method on an automated analyzer (standard laboratory reference range: 1.7–2.3 mg/dL). Hypomagnesemia was defined as <1.7 mg/dL and hypermagnesemia as >2.3 mg/dL. All magnesium levels were extracted from the the Hospital Information System (HIS) and cross-checked with archived paper medical records and laboratory report sheets to ensure data completeness and minimize missing records.

Sample Size

Given the retrospective design of the study, all eligible cases admitted during the study period were consecutively included, and controls were selected at a 1:1 ratio. Therefore, using consecutive enrollment of all eligible cases sampling during the study period, all eligible patients who met the inclusion criteria were enrolled. As a result, 73 patients were included in the case group. An equal number of matched controls was subsequently selected.

Statistical Analysis

Continuous variables are presented as mean ± standard deviation (all passed normality testing – Kolmogorov-Smirnov). Categorical variables are presented as frequency (percentage). Because matching was successful (no significant difference in age), unconditional methods were used for primary comparisons: independent-sample t-test for continuous variables and χ^2 or Fisher's exact test for categorical variables. Odds ratios with 95% confidence intervals were calculated for hypomagnesemia.

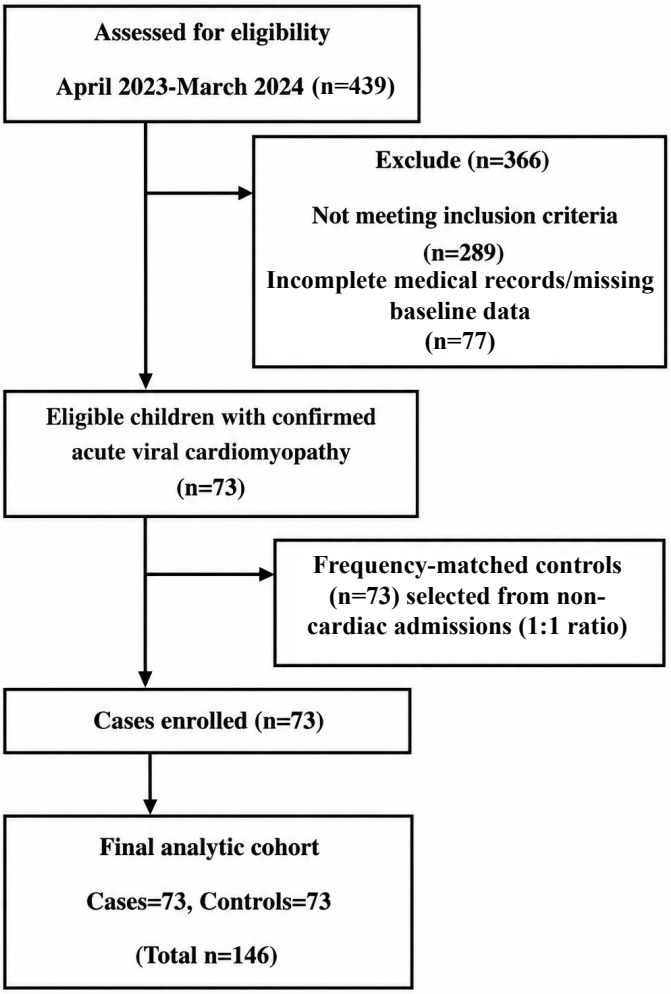


Figure 1. Flow diagram of participant selection according to the STROBE statement for case-control studies.

Multivariable binary logistic regression was performed to identify risk factors of acute viral cardiomyopathy (outcome: case = 1, control = 0). Variables with $P < 0.10$ in univariate analysis or deemed clinically relevant based on existing literature (sex, body mass index, presence of fever, and underlying chronic disease) were simultaneously entered into the model using the Enter (forced entry) method. Serum magnesium was included as a continuous variable (per 0.1 mg/dL decrease). The Enter method was selected to provide a conservative estimate of the independent effect of serum magnesium while simultaneously controlling for all pre-specified potential confounders, thereby minimizing the risk of overfitting and selection bias inherent in stepwise approaches, particularly in a moderate-sized sample. Two-sided p-values < 0.05 were considered statistically significant. Analyses were performed using IBM SPSS version 26.0 (Armonk, NY, USA).

Ethical Considerations

The study was approved by the Ethics Committee of Kerman University of Medical Sciences (IR.KMU.AH.REC.1404.119). Patient records were anonymised and de-identified before analysis. The requirement for individual informed consent was waived because of the retrospective design and use of routinely collected data.

Results

Between April 2023 and March 2024, 73 children fulfilled the diagnostic criteria for acute viral cardiomyopathy and were enrolled as cases. An equal number of age- and sex-matched controls

Table 1. Baseline characteristics and clinical characteristics of children with acute viral cardiomyopathy in cases and controls

Characteristic	Cardiomyopathy group (n=73)	control group (n=73)	P-value
Age (months)	108.52 ± 63.59	96.07 ± 59.53	0.223
Female sex	49 (67.1)	33 (45.2)	0.0076
BMI (kg/m²)	17.08 ± 2.08	17.71 ± 2.30	0.061
Fever on admission	60 (82.2)	35 (47.9)	<0.001
Underlying chronic disease	12 (16.4)	10 (13.7)	0.649
Hospital stay (days)	7.77 ± 2.42	5.39 ± 2.20	<0.001
In-hospital mortality	5 (6.8)	1 (1.4)	0.209

Note: *Data are presented as mean ± SD or n (%). P-values were calculated using an independent-samples t-test for continuous variables and χ^2 test or Fisher's exact test (when expected cell count< 5).*

Table 2. Comparison of serum magnesium levels between the two groups

Variable	Cardiomyopathy group (n=73)	Control group (n=73)	OR (95% CI)	P-value
Serum magnesium (mg/dL)	1.80 ± 0.31	1.98 ± 0.31		0.003
Normal level of magnesium (1.7–2.3 mg/dL)	35 (47.9%)	49 (67.1%)	Reference	-
Hypomagnesemia (<1.7 mg/dL)	29 (39.7%)	7 (9.6%)	5.80 (2.28 – 14.75)	<0.001
Hypermagnesemia (>2.3 mg/dL)	9 (12.3%)	17 (23.3%)	0.74 (0.30 – 1.85)	NS

Note: *Data are presented as mean ± standard deviation or number (percentage). Normal serum magnesium level (1.7–2.3 mg/dL) was considered the reference category. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated using logistic regression analysis. A p-value < 0.05 was considered statistically significant. "NS" means not significant.*

Table 3. Multivariable logistic regression analysis for associated risk factors of acute viral cardiomyopathy (outcome = acute viral cardiomyopathy)

Variable	Adjusted OR (95% CI)	p-value
Serum magnesium (per 0.1 mg/dL decrease)	1.38 (1.11–1.72)	0.004
Presence of fever	4.21 (1.88–9.43)	<0.001
Female sex	1.89 (0.89–4.02)	0.098
BMI	0.92 (0.79–1.07)	0.273
Underlying chronic disease	1.24 (0.49–3.15)	0.652

Note: *Adjusted odds ratios with 95% confidence intervals are shown. Model adjusted for sex, BMI, presence of fever, and underlying chronic disease. Serum magnesium entered as a continuous variable (per 0.1 mg/dL decrease) .All variables were entered simultaneously using the Enter method.*

were selected from children hospitalised for non-cardiac conditions during the same period (1:1 matching).

Baseline demographic and clinical characteristics are shown in Table 1. Age distribution was comparable between groups (108.52 ± 63.59 vs. 96.07 ± 59.53 months, P = 0.223); however, female sex was significantly more frequent among cases (67.1% vs. 45.2%, P = 0.0076). Therefore, sex was retained and adjusted for in the multivariable logistic regression model. BMI was similar between groups (17.08 ± 2.08 vs. 17.71 ± 2.30 kg/m², P = 0.061). Fever on admission was significantly more frequent in cases (82.2% vs. 47.9%, P < 0.001). The prevalence of underlying chronic disease did not differ significantly between the two groups (16.4% vs. 13.7%, P = 0.649). Duration of hospital stay was markedly longer in the cardiomyopathy group (7.77 ± 2.42 vs. 5.39 ± 2.20 days, P < 0.001).

Serum magnesium levels and categorical

distribution are presented in Table 2. Mean serum magnesium level was significantly lower in children with acute viral cardiomyopathy than in the control group (1.80 ± 0.31 vs. 1.98 ± 0.31 mg/dL, P = 0.003). Hypomagnesemia (<1.7 mg/dL) was present in 29/73 cases (39.7%) compared with only 7/73 controls (9.6%), yielding an unadjusted odds ratio of 5.80 (95% CI: 2.28–14.75, P < 0.001). Hypermagnesemia (>2.3 mg/dL) occurred in 12.3% of cases and 23.3% of controls (P = 0.085).

In-hospital mortality was 6.8% (5/73) in the cardiomyopathy group and 1.4% (1/73) in the non-cardiomyopathy group (P = 0.209). Notably, all five deaths in the cardiomyopathy group occurred among hypomagnesemic patients (magnesium 1.61 ± 0.18 mg/dL in non-survivors vs. 1.83 ± 0.32 mg/dL in survivors, P = 0.041). The single death in the control group had normal magnesium.

Multivariable binary logistic regression adjusting for sex, BMI, fever, and underlying chronic disease confirmed that lower serum magnesium (per 0.1 mg/dL decrease: adjusted OR 1.38, 95% CI 1.11–1.72, P = 0.004) and presence of fever (adjusted OR 4.21, 95% CI 1.88–9.43, P < 0.001) were associated risk factors for the prevalence of acute viral cardiomyopathy (Table 3).

Discussion

This study demonstrates that children hospitalized with acute viral cardiomyopathy have significantly lower serum magnesium levels and a fourfold higher prevalence of hypomagnesemia than age-matched controls admitted for non-cardiac conditions. These findings are consistent with the growing body of evidence showing that magnesium disturbances are frequent in critically ill children and may compromise cardiac performance.

Several mechanisms may account for this association. Magnesium is essential for myocardial ATP production, calcium channel regulation, and membrane stabilization. Direct viral injury to cardiomyocytes, the intense systemic inflammatory response, and catecholamine surges characteristic of acute heart failure can all drive magnesium redistribution into cells and accelerate renal losses. The resulting hypomagnesemia lowers the threshold for arrhythmias by prolonging action potential duration, depleting intracellular potassium, and facilitating excessive calcium influx, thereby worsening both systolic and diastolic function[24–25].

The 39.7% prevalence of hypomagnesemia observed in our viral cardiomyopathy cohort is among the highest reported in pediatric cardiac disease. For context, Hoseininasab et al. (2024) documented rates of 28-34% in a large Iranian MIS-C population[25], Rostami-Maskopaee et al. (2022) noted electrolyte abnormalities in 61% of MIS-C cases (magnesium disturbances prominent)[26], and Amoozgar et al. (2025) found electrolyte derangements in 61% of children with acute myocarditis in Shiraz[27]. In Iranian PICUs, Sadeghi-Bojd et al. (2021) observed hypomagnesemia in 31-45% of critically ill children[10], which closely agrees with our results. Internationally, Amar et al. (2024) reported rates of 25-40% in pediatric acute heart failure and myocarditis, with the highest figures in viral etiologies[28].

Although overall in-hospital mortality was low (6.8% in cases vs. 1.4% in controls), all five deaths in the case group occurred exclusively among hypomagnesemic patients (mean magnesium 1.61 ± 0.18 mg/dL in non-survivors vs. 1.83 ± 0.32 mg/dL in survivors; P = 0.041). While the limited number of events prevented statistical significance in multivariable models a common challenge in modern pediatric cardiomyopathy research, where mortality has fortunately declined—this striking clustering strongly parallels reports by Bogle et al. (2023) in septic children and Segev et al. (2022) in adult COVID-19 patients, both of whom identified hypomagnesemia as an independent predictor of fatal outcome[29–30]. Similarly, Dandinavar et al. (2019)[31] and Upala et al.[15] (2016) demonstrated nearly doubled mortality risk in hypomagnesemic ICU patients, reinforcing the clinical relevance of even modest magnesium deficits[15,31].

Similarly, in adult critically ill populations, Saghaleini et al. (2024)[32] demonstrated that serum magnesium levels <1.7 mg/dL were significantly associated with higher mortality and prolonged intensive care stay.

Hospital length of stay was significantly longer in cases (7.77 ± 2.42 vs. 5.39 ± 2.20 days; P < 0.001), consistent with the known association between electrolyte imbalance and prolonged critical illness. Although the difference in stay between hypomagnesemic and normomagnesemic children within the case group did not reach statistical significance, the observed trend (approximately 1-2 days longer) parallels findings from Dandinavar et al. (2019), who reported a mean 3-day increase in hypomagnesemic patients in the PICU[31].

Recent randomized trials provide supportive, but indirect, evidence for a potential therapeutic role of magnesium in pediatric cardiac populations. Furthermore, Uysal et al. (2024)[22] showed that oral magnesium supplementation significantly reduced the burden of ventricular extrasystoles in children with diverse cardiac conditions[22], whereas Szyller et al. (2023) documented a 55% reduction in postoperative junctional ectopic tachycardia and ventricular arrhythmias after intraoperative magnesium administration in congenital heart surgery[22,33]. Although according to the authors' investigation, no trials have yet specifically targeted viral cardiomyopathy, these data suggest that magnesium repletion may be beneficial in selected high-risk settings; however, extrapolation of these findings to acute viral cardiomyopathy should be made with caution, and dedicated interventional studies in this population are still required.

In summary, hypomagnesemia is highly prevalent, independently associated with acute viral cardiomyopathy in children, and consistently linked to adverse clinical trends. Routine measurement of serum magnesium on admission and consideration of correction when low could be a reasonable component of comprehensive supportive care; nevertheless, the potential impact of magnesium supplementation on hard outcomes remains uncertain and should be clarified in future prospective interventional trials.

Study Limitations and Strengths

The retrospective, single-center design inherently limits generalizability[34] to other populations or healthcare settings, particularly those with different viral epidemiology or nutritional status. Although the total sample (n=146) was adequate for the primary objective, the low number of in-hospital deaths (n = 6) and absence of systematic arrhythmia monitoring (Holter or telemetry in all patients) precluded sufficiently powered analyses of hard clinical endpoints such as fatal arrhythmia or need for mechanical circulatory support; this is a recurring challenge in pediatric cardiomyopathy research because of the fortunately low mortality in the modern era. Serial magnesium measurements were not performed, so we could not evaluate dynamic changes or the impact of in-hospital repletion. Viral confirmation relied primarily on clinical, echocardiographic, and biomarker criteria according to current American Heart Association guidelines, with PCR or serology available in only a subset of patients; thus, some misclassification of etiology cannot be entirely excluded, although this reflects real-world diagnostic practice in many centers. Finally, we did not collect data on dietary magnesium intake, concurrent medications (e.g., loop diuretics), or inflammatory markers (IL-6, TNF-α), which could have provided additional mechanistic insights.

Another key limitation of this retrospective case-control study is the potential confounding by factors affecting magnesium homeostasis, which were not assessed. These include common ICU medications such as loop diuretics (e.g., furosemide), inotropes, proton pump inhibitors (PPIs), and other drugs, as well as infectious/inflammatory conditions prevalent in critically ill patients. Such confounders may influence serum magnesium levels independently of acute viral cardiomyopathy. Future prospective

studies adjusting for these variables seem to be necessary.

Despite these limitations, the consistent clustering of adverse outcomes in hypomagnesemic children and the biological plausibility supported by recent pediatric critical care (ICU or other critical illness) trials of magnesium supplementation reinforce the clinical relevance of our findings and strongly justify larger, prospective, multicenter studies with serial sampling, continuous rhythm monitoring, and interventional arms.

Conclusion

Hypomagnesemia is highly prevalent and independently associated with acute viral cardiomyopathy in children in this retrospective case-control study. These findings highlight hypomagnesemia as a potentially important clinical marker. Early recognition of low magnesium levels and their correction are biologically plausible and widely used supportive measures in pediatric cardiac care, but their specific effect on outcomes in viral cardiomyopathy remains to be determined.

Routine monitoring of serum magnesium levels in children with cardiomyopathy may be considered as part of comprehensive management, and future well-designed interventional studies are needed to determine whether targeted magnesium correction can improve prognosis in this population.

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Conflict of interests

The authors declare no conflict of interest.

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Author's Contributions

Study Conception or Design: FDM
Data Acquisition: FDM; RD
Data Analysis or Interpretation: FA
Manuscript Drafting: FA; RD
Critical Manuscript Revision: FA; RD
All authors have approved the final manuscript and are responsible for all aspects of the work.

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