

Serum Magnesium and Ventricular Repolarization in Adolescents Without Documented Cardiovascular Disease: A Retrospective Study

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Abstract

Objective: To evaluate the association between serum magnesium status and electrocardiographic parameters, particularly ventricular repolarization, among adolescents without documented cardiovascular disease.

Methods: This retrospective analytical study included 150 records of adolescents aged 10–19 years with available serum magnesium measurements and 12-lead electrocardiograms. Serum magnesium was evaluated continuously and categorized as low (<1.7 mg/dL) or normal (1.7–2.4 mg/dL). Heart rate, PR interval, QRS duration, QT interval, and corrected QT (QTc) interval were analyzed. Correlation and multivariable linear regression analyses were performed to assess the relationship between serum magnesium and QTc.

Results: Of 150 eligible records, 29 (19.3%) had low serum magnesium. Adolescents with low magnesium demonstrated higher resting heart rate (82.5 ± 11.8 vs. 77.7 ± 11.1 beats/min; $P=0.039$) and longer QT (385.8 ± 27.1 vs. 372.3 ± 25.8 ms; $P=0.014$) and QTc intervals (431.0 ± 22.7 vs. 414.6 ± 23.3 ms; $P=0.001$). PR interval and QRS duration did not differ significantly between groups. Serum magnesium was inversely correlated with QTc ($r=-0.32$; $P<0.001$) and remained independently associated with QTc after adjustment for age, sex, and heart rate ($\beta=-0.27$; $P=0.001$).

Conclusion: Lower serum magnesium was associated with longer ventricular repolarization intervals among adolescents without documented cardiovascular disease. Prospective studies are required to determine the clinical and preventive significance of this association.

Keywords: Adolescent; Magnesium; Electrocardiography; QT Interval; Cardiovascular Health.

Introduction

Adolescence is a period of substantial physiological, metabolic, and developmental transition during which nutritional requirements increase to support rapid somatic growth, changes in body composition, skeletal mineralization, and maturation of multiple organ systems. Nutritional inadequacies arising during this period may have consequences extending beyond immediate growth and development and may contribute to cardiovascular and metabolic risk profiles that persist into adulthood [1,2]. Increasing attention has therefore been directed toward potentially modifiable nutritional factors that may influence early cardiovascular physiology before clinically apparent disease develops.

Magnesium is an essential intracellular divalent cation involved in energy metabolism, enzymatic activity, membrane stabilization, neuromuscular function, and electrolyte homeostasis [3,4]. It acts as a cofactor in numerous adenosine triphosphate-dependent reactions and contributes to normal cellular function through regulation of transmembrane ion transport. During adolescence, adequate magnesium availability is particularly relevant because of increased requirements associated with growth and tissue development. Dietary patterns characterized by greater consumption of processed foods and lower intake of magnesium-rich foods may potentially contribute to suboptimal magnesium status in young populations [1,3].

Within the cardiovascular system, magnesium plays an important role in myocardial excitability, vascular tone, cardiac conduction, and maintenance of normal rhythm. Its electrophysiological actions involve interactions with potassium and calcium channels, regulation of sodium–potassium ATPase activity, and stabilization of cellular

membrane potential [4,5]. Reduced magnesium availability may impair intracellular potassium retention, alter calcium influx, and increase myocardial excitability. These effects provide a biologically plausible mechanism through which variations in magnesium status could influence cardiac electrical activity even in individuals without recognized cardiovascular disease [5,6].

The electrocardiogram (ECG) offers a simple and non-invasive means of assessing cardiac electrical function. Heart rate, PR interval, QRS duration, QT interval, and corrected QT (QTc) interval provide information regarding impulse generation, atrioventricular conduction, ventricular depolarization, and ventricular repolarization. Among these parameters, QTc is of particular interest because it reflects the duration of ventricular electrical activity after correction for heart rate. Prolongation or increased heterogeneity of ventricular repolarization may indicate reduced electrical stability and, when sufficiently pronounced or accompanied by other risk factors, increased susceptibility to ventricular arrhythmias [6,7].

Magnesium status has previously been associated with cardiac repolarization and rhythm abnormalities. Hypomagnesemia can alter myocardial ion-channel activity and has been associated with ventricular ectopy, QT-related abnormalities, and increased susceptibility to arrhythmias [5,8]. Electrocardiographic investigations in individuals with magnesium deficiency have demonstrated alterations predominantly involving ventricular repolarization, while conventional measures of atrioventricular and intraventricular conduction may remain comparatively unaffected [8]. These observations raise the possibility that QT and QTc may represent sensitive ECG parameters through which subtle variations in magnesium status can be explored.

Although cardiovascular disease generally becomes clinically evident later in life, its physiological and behavioral determinants may begin much earlier. Adolescence therefore provides an important window for investigating early cardiovascular risk indicators and potentially modifiable nutritional exposures [2,9]. Assessment during this stage may identify subtle physiological differences before the emergence of hypertension, diabetes, structural cardiovascular disease, or other conditions that complicate interpretation of cardiac electrophysiology in adult populations.

Despite the established physiological importance of magnesium, comparatively limited information is available regarding the relationship between serum magnesium and routine ECG parameters in apparently healthy adolescents. Much of the existing evidence regarding magnesium and cardiac electrophysiology has been derived from adults, hospitalized populations, individuals with clinically significant electrolyte abnormalities, or patients with established cardiovascular disease [5,8,10]. Findings from these populations cannot necessarily be extrapolated directly to adolescents because cardiac rate, autonomic regulation, body composition, nutritional requirements, and developmental physiology differ across age groups.

Investigating serum magnesium alongside ECG parameters in apparently healthy adolescents may therefore provide useful insight into the relationship between micronutrient status and early cardiac electrical physiology. Identification of an association between lower magnesium concentrations and altered ventricular repolarization would not establish future cardiovascular disease or justify routine supplementation; however, it could provide a rationale for prospective nutritional and electrophysiological studies in adolescent populations. Such evidence may ultimately contribute to a better understanding of potentially modifiable factors influencing cardiovascular health during early life.

Therefore, the present study aimed to evaluate the association between serum magnesium status and electrocardiographic parameters in apparently healthy adolescents. Particular emphasis was placed on the relationship between serum magnesium concentration and ventricular repolarization, represented by QT and QTc intervals, while heart rate, PR interval, and QRS duration were evaluated as additional electrophysiological parameters. The study further assessed whether serum magnesium was independently associated with QTc after accounting for relevant demographic and anthropometric characteristics.

Materials and Methods

This retrospective analytical study was conducted using existing clinical and laboratory records of adolescents aged 10–19 years who had undergone both serum magnesium testing and standard 12-lead electrocardiography during the predefined study period. Records were retrieved from the electronic or paper-based database of [institution/diagnostic center] between [start date] and [end date].

Eligible records included adolescents with documented serum magnesium concentration and an interpretable resting 12-lead ECG performed within [same day/24–48 hours] of biochemical testing. Records were excluded if there was

documented congenital or acquired cardiovascular disease, clinically significant arrhythmia, chronic kidney disease, major endocrine disorder, acute severe illness, or use of medications known to markedly alter cardiac conduction or magnesium status. Records with incomplete demographic information, missing magnesium values, technically inadequate ECGs, or major conduction abnormalities were also excluded.

For each eligible record, age, sex, available anthropometric information, serum magnesium concentration, heart rate, PR interval, QRS duration, QT interval, and QTc interval were extracted using a standardized data collection sheet. Where height and weight were available, body mass index was calculated and interpreted according to age- and sex-appropriate standards. No additional clinical examination, blood sampling, ECG recording, or direct participant contact was undertaken for the purpose of the study.

Serum magnesium values were obtained from routine laboratory investigations performed using the institution's standard biochemical method. Magnesium concentration was expressed in mg/dL and evaluated both as a continuous variable and categorically. For analytical purposes, values below 1.7 mg/dL were considered low, while values between 1.7 and 2.4 mg/dL were considered within the reference range, subject to confirmation against the reporting laboratory's reference interval.

Electrocardiographic measurements were obtained from existing standard 12-lead ECG records. ECGs had been recorded using routine calibration of 25 mm/s paper speed and 10 mm/mV amplitude. The principal variables extracted were resting heart rate, PR interval, QRS duration, QT interval, and corrected QT interval. Where QTc was not directly reported, it was calculated from QT and RR intervals using Bazett's formula.

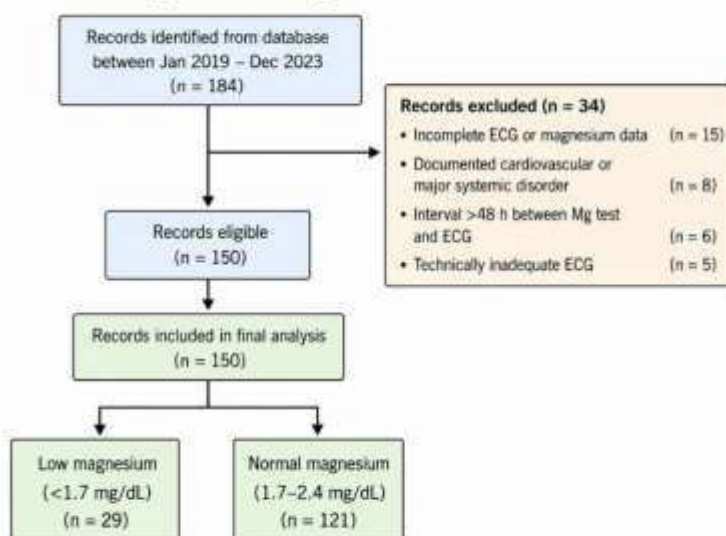
Data were analyzed using IBM SPSS Statistics version 27. Continuous variables were summarized as mean $\pm$ standard deviation or median and interquartile range, depending on distribution, while categorical variables were presented as frequencies and percentages. ECG parameters were compared between adolescents with low and normal serum magnesium using the independent-samples t-test or Mann-Whitney U test as appropriate. Pearson or Spearman correlation analysis was performed to evaluate relationships between serum magnesium concentration and ECG parameters. Multivariable linear regression was used to assess the independent association between serum magnesium and QTc after adjustment for available covariates such as age, sex, BMI, and heart rate. A P-value <0.05 was considered statistically significant.

Ethical approval was obtained from the relevant institutional ethics review committee before record extraction. As the study involved retrospective analysis of existing de-identified records with no direct participant contact or intervention, a waiver of individual informed consent was requested or obtained in accordance with institutional policy.

Results

A total of 184 adolescent records containing serum magnesium measurements and electrocardiographic data were initially identified during the study period. After eligibility assessment, 34 records were excluded because of incomplete ECG or biochemical data ($n=15$), a documented cardiovascular or major systemic disorder ($n=8$), an interval exceeding the predefined period between serum magnesium measurement and ECG recording ($n=6$), or technically inadequate ECG recordings ($n=5$). The final analysis therefore included 150 records of adolescents aged 10–19 years.

Figure 1. Flow diagram of record selection.



The mean age of the adolescents included in the analysis was 15.1 ± 2.4 years, and 79 (52.7%) records were from males and 71 (47.3%) from females. The mean serum magnesium concentration was 1.93 ± 0.27 mg/dL. Based on the predefined laboratory threshold, 29 (19.3%) records were classified as having low serum magnesium (<1.7 mg/dL), while 121 (80.7%) had magnesium concentrations within the reference range. Age and sex distribution did not differ significantly between magnesium-status groups.

Table 1. Characteristics of adolescent records according to serum magnesium status

Variable	Overall (n=150)	Low Mg (n=29)	Normal Mg (n=121)	P-value
Age, years	15.1 ± 2.4	15.4 ± 2.5	15.0 ± 2.4	0.421
Male, n (%)	79 (52.7)	16 (55.2)	63 (52.1)	0.765
Female, n (%)	71 (47.3)	13 (44.8)	58 (47.9)	—
Serum magnesium, mg/dL	1.93 ± 0.27	1.56 ± 0.09	2.02 ± 0.20	<0.001

Electrocardiographic analysis demonstrated a mean resting heart rate of 78.6 ± 11.4 beats/min, mean PR interval of 151.8 ± 18.1 ms, QRS duration of 87.3 ± 9.1 ms, QT interval of 374.9 ± 26.5 ms, and QTc interval of 417.8 ± 23.9 ms across the analyzed records.

Adolescents with low serum magnesium had a significantly higher mean resting heart rate than those with magnesium concentrations within the reference range (82.5 ± 11.8 vs. 77.7 ± 11.1 beats/min; $P=0.039$). The low-magnesium group also demonstrated significantly longer QT (385.8 ± 27.1 vs. 372.3 ± 25.8 ms; $P=0.014$) and QTc intervals (431.0 ± 22.7 vs. 414.6 ± 23.3 ms; $P=0.001$). In contrast, differences in PR interval and QRS duration were not statistically significant.

Table 2. Electrocardiographic parameters according to serum magnesium status

ECG parameter	Overall (n=150)	Low Mg (n=29)	Normal Mg (n=121)	P-value
Heart rate, beats/min	78.6 ± 11.4	82.5 ± 11.8	77.7 ± 11.1	0.039
PR interval, ms	151.8 ± 18.1	154.4 ± 18.7	151.2 ± 18.0	0.391
QRS duration, ms	87.3 ± 9.1	88.4 ± 9.5	87.0 ± 9.0	0.457
QT interval, ms	374.9 ± 26.5	385.8 ± 27.1	372.3 ± 25.8	0.014
QTc interval, ms	417.8 ± 23.9	431.0 ± 22.7	414.6 ± 23.3	0.001

When serum magnesium was evaluated as a continuous variable, significant inverse relationships were observed with QT and QTc intervals. Serum magnesium showed its strongest association with QTc ($r=-0.32$, $P<0.001$), followed by QT interval ($r=-0.25$, $P=0.002$). A weaker inverse correlation was observed with resting heart rate ($r=-0.17$, $P=0.038$). Serum magnesium was not significantly correlated with PR interval or QRS duration.

Table 3. Correlation between serum magnesium concentration and electrocardiographic parameters

ECG parameter Pearson's r P-value

Heart rate	-0.17	0.038
PR interval	-0.08	0.330
QRS duration	-0.05	0.543
QT interval	-0.25	0.002
QTc interval	-0.32	<0.001

Multivariable linear regression was performed to determine whether serum magnesium was independently associated with QTc. Because the study was retrospective, adjustment was restricted to variables consistently available in the records. After adjustment for age, sex, and resting heart rate, serum magnesium remained independently and inversely associated with QTc ($B=-23.4$ ms per 1 mg/dL increase; standardized $\beta=-0.27$; 95% CI: -36.7 to -10.1; $P=0.001$). Resting heart rate was also independently associated with QTc, while age and sex were not significant predictors.

Table 4. Multivariable linear regression analysis for QTc interval

Predictor	B	Standardized β	95% CI for B	P-value
Serum magnesium, mg/dL	-23.4	-0.27	-36.7 to -10.1	0.001
Age, years	0.52	0.05	-0.58 to 1.62	0.351
Male sex	-3.1	-0.06	-10.5 to 4.3	0.409
Heart rate, beats/min	0.38	0.18	0.06 to 0.70	0.020

Overall, the retrospective analysis demonstrated that lower serum magnesium was associated predominantly with longer ventricular repolarization intervals, particularly QTc, rather than with alterations in atrioventricular or intraventricular conduction. The association between serum magnesium and QTc persisted after adjustment for age, sex, and resting heart rate.

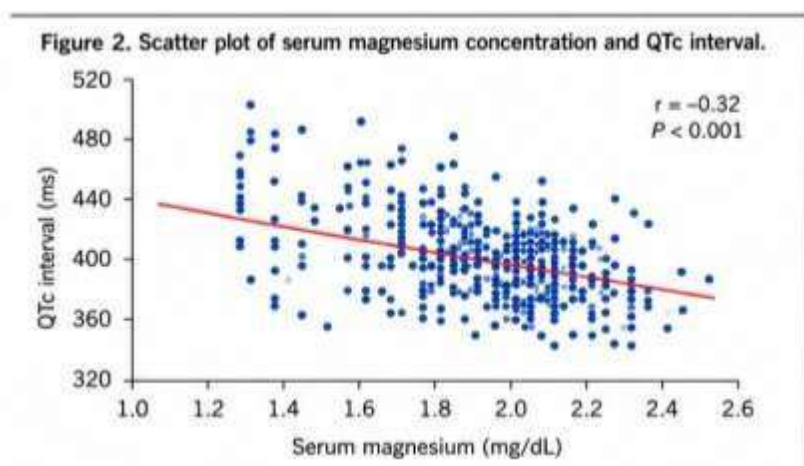
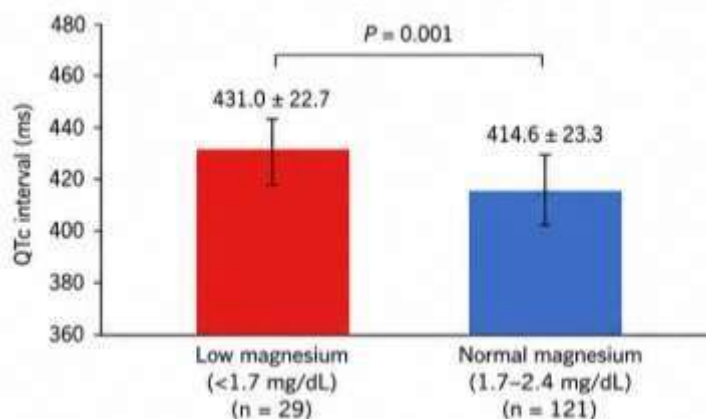


Figure 3. QTc interval according to serum magnesium status.



Discussion

The present retrospective study identified a significant relationship between serum magnesium status and cardiac electrical parameters among adolescents without documented cardiovascular disease. The principal finding was that lower serum magnesium concentrations were associated with longer QT and QTc intervals, whereas PR interval and QRS duration showed no significant relationship with magnesium status. Adolescents with serum magnesium concentrations below 1.7 mg/dL also demonstrated a modestly higher resting heart rate. Importantly, the inverse association between serum magnesium and QTc persisted after adjustment for age, sex, and heart rate. Collectively, these findings suggest that variations in magnesium status may be more closely related to ventricular repolarization than to atrioventricular or intraventricular conduction.

The observed relationship is physiologically plausible given the central role of magnesium in myocardial electrophysiology. Magnesium influences membrane stability, sodium-potassium ATPase activity, intracellular potassium homeostasis, and calcium-dependent cellular processes [2-4]. Alterations in magnesium availability can consequently affect transmembrane ionic gradients and currents involved in the cardiac action potential. Experimental evidence further indicates that intracellular magnesium contributes to regulation of inward rectifier potassium currents and cardiac repolarization reserve [12]. A reduction in magnesium availability could therefore influence ventricular action-potential duration and manifest electrocardiographically as changes in QT-related parameters.

In the present analysis, adolescents with low serum magnesium had a mean QTc of 431.0 ± 22.7 ms compared with 414.6 ± 23.3 ms among those with magnesium concentrations within the reference range. Serum magnesium also demonstrated an inverse correlation with QTc ($r = -0.32$, $P < 0.001$), and the association remained significant in multivariable analysis. These findings are broadly consistent with previous electrophysiological studies linking hypomagnesemia with altered ventricular repolarization. Yang et al. reported significant ECG changes among patients with isolated hypomagnesemia, supporting an association between magnesium deficiency and repolarization abnormalities [5]. More recently, Aslan et al. demonstrated longer QT, QTc, and other ventricular repolarization indices in individuals with lower magnesium concentrations and identified serum magnesium as an independent predictor of QTc [11]. Although these investigations were primarily conducted in adult populations, their findings provide mechanistic and clinical support for the direction of association observed in the present adolescent sample. An important feature of the present findings is the apparent specificity of the association. Neither PR interval nor QRS duration differed significantly according to magnesium status, and their correlations with serum magnesium were weak and nonsignificant. The PR interval predominantly reflects atrial and atrioventricular conduction, whereas QRS duration represents ventricular depolarization. In contrast, QT and QTc incorporate ventricular repolarization, a phase of cardiac electrical activity strongly dependent on coordinated potassium and calcium currents. The selective association of magnesium with QT-related measures therefore supports the possibility that modest variations in magnesium status may influence repolarization before producing detectable changes in conventional conduction intervals. This pattern is also compatible with previous observations in magnesium-deficient populations [5,11].

A weaker inverse association was observed between serum magnesium and resting heart rate. Adolescents with low magnesium had a higher mean heart rate than those with magnesium concentrations within the reference range, although the magnitude of this difference was considerably smaller than that observed for QTc. Heart rate during adolescence is influenced by several factors, including age, autonomic tone, physical activity, emotional state, hydration, and metabolic conditions. Magnesium may contribute indirectly through effects on cellular excitability and autonomic cardiovascular regulation; however, because these determinants could not be comprehensively assessed from retrospective records, the observed heart-rate association should be interpreted cautiously. The persistence of the magnesium–QTc association after adjustment for heart rate is therefore particularly relevant.

The adolescent context of these findings deserves consideration. Adolescence is characterized by rapid somatic growth, changes in body composition, hormonal maturation, evolving autonomic regulation, and increased nutritional requirements. At the same time, dietary patterns during adolescence may include insufficient consumption of vegetables, whole grains, legumes, nuts, and other magnesium-rich foods. Magnesium inadequacy has been recognized as a potentially modifiable nutritional concern, while broader evidence links magnesium intake and circulating magnesium concentrations with cardiovascular and metabolic health [1,8,15]. The present findings extend this concept by suggesting that magnesium status may also be associated with measurable differences in cardiac electrical physiology during adolescence.

Nevertheless, the observed differences should not be interpreted as evidence that adolescents with lower magnesium concentrations have established cardiovascular disease or are necessarily at imminent risk of arrhythmia. QTc is influenced by multiple physiological, genetic, metabolic, and pharmacological factors, and the mean QTc values observed in both groups must be considered within the broader clinical context. Rather than functioning as a diagnostic marker of disease, the present findings identify an electrophysiological association that may warrant further investigation. This distinction is particularly important when studying otherwise young populations in whom unnecessary labeling of cardiovascular risk should be avoided.

The findings also have potential preventive relevance. Magnesium status is influenced by dietary intake, gastrointestinal absorption, renal handling, and medication exposure [14–16]. Unlike fixed cardiovascular risk determinants, nutritional exposures are potentially modifiable. However, the present study does not demonstrate that magnesium supplementation shortens QTc or improves cardiovascular outcomes in adolescents. Evidence supporting magnesium administration in specific clinical circumstances, including certain ventricular arrhythmias and torsades de pointes, cannot be directly extrapolated to routine supplementation of healthy adolescents [17–19]. Prospective studies are required to determine whether correction of low magnesium status produces measurable changes in ventricular repolarization in adolescents.

Several strengths of this study should be acknowledged. Focusing on adolescents without documented cardiovascular disease reduced the influence of established cardiac disorders that commonly complicate interpretation of magnesium–ECG relationships in adult clinical populations. Evaluation of magnesium both categorically and continuously provided complementary evidence, while the consistency between group comparisons, correlation analysis, and multivariable regression strengthened the internal pattern of findings. The absence of significant relationships with PR and QRS parameters also argues against a nonspecific association across all ECG measurements.

The study nevertheless has important limitations. Its retrospective design restricts inference to association and prevents determination of temporality or causality. The analysis depended on information available in existing records, and unmeasured factors such as dietary magnesium intake, serum potassium and calcium, caffeine or energy-drink consumption, physical activity, hydration, medication exposure, pubertal stage, and autonomic influences could have contributed to the observed relationships. Serum magnesium represents only a small proportion of total-body magnesium and may not fully reflect intracellular magnesium status. A single magnesium measurement may also be affected by short-term biological variability. Furthermore, ECG measurements obtained during routine clinical care may be subject to measurement variability, and QT correction using Bazett's formula can introduce heart-rate-related error, particularly at higher heart rates. Finally, the findings are based on records from a defined clinical setting and may not be generalizable to the wider adolescent population.

Future prospective studies should therefore include standardized ECG acquisition, repeated magnesium measurements, concurrent assessment of potassium and calcium, detailed dietary evaluation, medication and stimulant exposure, physical activity, and age- and sex-specific developmental characteristics. Alternative QT correction methods such as Fridericia's formula may also be evaluated alongside Bazett-corrected QT. Longitudinal

studies could determine whether magnesium status predicts persistent changes in ventricular repolarization and whether improvement in magnesium status is accompanied by corresponding electrophysiological changes.

In conclusion, this retrospective analysis demonstrated that lower serum magnesium concentrations were associated with longer QT and QTc intervals among adolescents without documented cardiovascular disease, while PR interval and QRS duration were not significantly related to magnesium status. Serum magnesium remained independently and inversely associated with QTc after adjustment for age, sex, and heart rate. These findings support a potential relationship between magnesium status and ventricular repolarization during adolescence but do not establish causality or clinical arrhythmic risk. Prospective studies are warranted to clarify the physiological and potential preventive significance of this association in adolescent populations.

Conclusion

Lower serum magnesium status was associated with higher resting heart rate and longer QT and QTc intervals among adolescents without documented cardiovascular disease, while PR interval and QRS duration showed no significant relationship with magnesium status. Serum magnesium demonstrated an independent inverse association with QTc after adjustment for age, sex, and heart rate, suggesting that magnesium status may be more closely related to ventricular repolarization than to atrioventricular or intraventricular conduction. These findings indicate a potential relationship between magnesium homeostasis and cardiac electrical physiology during adolescence. However, given the retrospective observational design, causality and future cardiovascular or arrhythmic risk cannot be inferred. Prospective studies are required to determine the clinical significance of this association and whether changes in magnesium status are accompanied by corresponding changes in ventricular repolarization.

Limitations

The retrospective design limits causal inference and restricted the analysis to variables available in existing records. A single serum magnesium measurement may not accurately represent intracellular magnesium stores or long-term magnesium status. Potentially relevant factors, including dietary magnesium intake, serum potassium and calcium, caffeine or energy-drink consumption, physical activity, hydration status, pubertal stage, and medication exposure, could not be comprehensively evaluated. In addition, routine ECG measurements may be subject to measurement variability, and QTc calculation using Bazett's formula may introduce heart-rate-related error. The findings were derived from a defined clinical setting and may therefore not be fully generalizable to the broader adolescent population. Prospective studies incorporating standardized ECG acquisition, repeated magnesium measurements, additional electrolyte assessment, and alternative QT correction methods are warranted.

Clinical and Preventive Implications

The association between lower serum magnesium and longer ventricular repolarization intervals suggests that magnesium status may represent a potentially modifiable physiological factor relevant to cardiac electrical health during adolescence. Serum magnesium assessment may warrant consideration when otherwise unexplained QTc prolongation is identified, particularly when biochemical testing is clinically indicated. However, the present findings do not demonstrate that lower magnesium causes QTc prolongation, nor do they establish a benefit from routine magnesium screening or supplementation in adolescents. Prospective and interventional studies are required before specific preventive or therapeutic recommendations can be made.

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Authors' Contributions

All authors contributed to the conception and design of the study, data acquisition and/or interpretation, critical revision of the manuscript, and approval of the final version. Specific author contributions should be finalized according to the actual roles of individual authors before submission.

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Conflict of Interest

The authors declare no conflict of interest.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to institutional and ethical requirements.

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