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Original Research

The effect of vitamin D levels on ventricular repolarization parameters in children

Vitamin D levels ventricular repolarization

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Abstract

Aim: In our study, we evaluated the relationship between serum 25(OH)D levels and ventricular repolarization parameters that can be used to predict ventricular arrhythmias in electrocardiography.

Methods: This prospective study included 153 children aged 5–17 years. Participants were classified as having serum 25-hydroxyvitamin D (25[OH]D) levels <20 ng/mL or ≥20 ng/mL and were further classified as deficient (<12 ng/mL), insufficient (12-20 ng/mL), or sufficient (>20 ng/mL). Laboratory tests included 25(OH)D, parathyroid hormone, and minerals. Standard 12-lead electrocardiograms were analyzed for QTmaximum, QTminimum, QT interval, QT dispersion, corrected QTmaximum, corrected QTminimum, corrected QT interval, corrected QT dispersion, Tpeak-Tend maximum, and Tpeak-Tend minimum parameters. Correlation and regression analyses were performed.

Results: Children with vitamin D < 20 ng/mL exhibited significantly higher QT maximum, corrected QT interval, QT dispersion, Tpeak-Tend, and Tpeak-Tend/inetrval QT and Tpeak-Tend/corrected QT ratios ($P < .001$). Both the deficiency and insufficiency groups showed elevated repolarization markers compared with those in the sufficient group. Serum 25(OH)D correlated negatively with QT dispersion and corrected QT maximum, while parathyroid hormone correlated positively with interval QT and QT maximum. A statistically significant effect of Vitamin D value on corrected QT maximum was determined. Accordingly, a 1-unit increase in Vitamin D value causes a 0.673 unit decrease in corrected QT maximum value.

Conclusion: Vitamin D deficiency and insufficiency may be associated with altered ventricular repolarization, indicating increased electrical heterogeneity and potential arrhythmogenic susceptibility.

Keywords

vitamin D, ventricular repolarization, arrhythmia, child

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Introduction

The primary physiological role of vitamin D is the regulation of calcium and phosphorus metabolism, which is essential for bone mineralization. However, vitamin D receptors are also expressed in various cell types, including cardiomyocytes. Through these receptors, vitamin D contributes to cardiovascular protection by modulating endothelial function, suppressing atherogenic T lymphocytes, and inhibiting vascular smooth muscle proliferation.[1] Vitamin D deficiency has been associated with autonomic dysfunction and ionic channel abnormalities, which may lead to malignant arrhythmias and sudden cardiac death.[2]

Arrhythmias may be transient or persistent, congenital or acquired, or occur secondary to medications or toxins. They can be associated with specific forms of congenital heart disease (CHD), complications following surgical repair, genetic disorders, or inflammatory mechanisms such as maternal connective tissue disease.[3] Both bradyarrhythmias and tachyarrhythmias may reduce cardiac output, transition into life-threatening ventricular fibrillation, or lead to cardiomyopathy when persistent. Arrhythmias may present with various clinical manifestations or may result in sudden death.[4]

The cardiac conduction system is routinely evaluated using electrocardiography (ECG). Ventricular repolarization parameters are of particular interest because they may predict susceptibility to malignant ventricular arrhythmias. Commonly used markers include the QT interval (QT), QT dispersion (QTd), corrected QT (QTc), and QTc dispersion (QTcd). More recently, parameters representing the transmural dispersion of repolarization, such as Tpeak–Tend (Tp-e), Tp-e dispersion (Tp-ed), and Tp-e/QT or Tp-e/QTc ratios, have been proposed as predictors of arrhythmogenic risk.[5,6] The effect of vitamin D on ventricular repolarization remains an area open to further research. Increased cardiovascular risk in vitamin D deficiency is thought to arise from heightened systemic inflammation and the resultant oxidative stress, collagen loss, and fibrosis. These changes predispose to cardiomyopathy, hypertrophy, myocardial infarction, heart failure, cardiac fibrosis, and rhythm disturbances. Additionally, endothelial and vascular smooth muscle dysfunction may increase the risk of atherosclerosis, vascular calcification, aneurysm, and hypertension.[7]

In this study, we assessed whether vitamin D levels in otherwise healthy children affect ECG parameters that predict susceptibility to ventricular arrhythmia. Our aim was to determine whether vitamin D deficiency is associated with an increased risk of arrhythmias in pediatric patients.

Materials and Methods

Study Design and Population

Our study is a prospective study including 153 healthy children aged 5–17 years who were registered between 01.10.2021 and 01.11.2022 and underwent a 13-month outpatient clinic examination at our hospital. Children with chronic diseases, acute infections, cardiovascular disorders, or regular medication use were excluded.

Participants were initially grouped based on serum 25(OH)D levels as follows:

- <20 ng/mL (low vitamin D)
- ≥20 ng/mL (normal vitamin D)

A secondary classification divided the cohort into:

- Group 1: deficiency (<12 ng/mL)
- Group 2: insufficiency (12–20 ng/mL)
- Group 3: sufficiency (≥20 ng/mL)

Vitamin D, parathyroid hormone (PTH), calcium, phosphorus, magnesium, and alkaline phosphatase levels were measured in all participants.

ECG Acquisition and Measurements

Standard 12-lead ECGs were obtained in the resting state (Electrocardiograph ECG-1350K, Nihon-Kohden; speed 25 mm/s, amplitude 10 mm/mV).

ECG evaluation included: verification of electrode placement and recording settings, rhythm assessment and P–QRS relationship, age-appropriate heart rate evaluation, PR interval, P wave and QRS axis, QT, QTmax, QTmin, QTd, QTc, QTcmax, QTcmin, QTcd, Tp-e max, Tp-e min, Tp-e, Tp-ed, Tp-e/QT, and Tp-e/QTc

QT interval: Calculated as the time from the beginning of the QRS complex to the end of the T peak. Programming was not performed in derivations for which the T peak endpoint was unknown. In cases where difficulty was encountered in determining the end point of the T peak or in cases of notched T peak, the maximum slope intercept method was recorded. The longest QT interval (QTmax), the shortest QT interval (QTmin), and the differences between them were recorded as QT dispersion.

QTc (Corrected QT value): The QT value corrected for heart rate was calculated using the Bazett formula (Bazett formula: QT duration / √R-R).

QTc dispersion: The difference between the longest QT peak (QTcmax) and the shortest QT peak (QTcmin) was recorded.

Tp-e interval: Recorded as the time from the peak of the T peak to the point where the T peak meets the isoelectric line. Programming was not performed in derivations for which the T peak endpoint was unknown. In cases where the end point of T formation was difficult to determine or in cases of notched T melting, the maximum slope intercept method was recorded. The longest Tp-e duration (Tp-e max) was calculated as the shortest Tp-e duration (Tp-e min). Tp-e dispersion was calculated and recorded as the difference between the Tp-e max and Tp-e min conditions.

Ethical Approval

This study was approved by the Ethics Committee of Sakarya University (Date: 14.10.2021, Decision No: E-16214662-050.01.04-70470-177).

Statistical Analysis

Data were analyzed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Kolmogorov–Smirnov test and presented as mean ± standard deviation or median (interquartile range), as appropriate. Categorical variables were expressed as frequencies and percentages. Group comparisons were performed using the Independent samples *t*-test, ANOVA, Mann–Whitney *U* test, or Kruskal–Wallis test, as appropriate. Categorical variables were compared using the chi-square test. Correlations were assessed using Pearson or Spearman tests, and multiple linear regression analysis was conducted to identify independent predictors. A *P* value *<.05* was considered statistically significant.

Reporting Guidelines

This study was reported in accordance with the STROBE guideline.

Results

There were no significant differences between the groups regarding age or sex. QT, QTmin, and QTcmin values were similar between the

Table 1. Ventricular Repolarization Parameters Among Vitamin D Subgroups

Parameter	<12 ng/mL	12–20 ng/mL	>20 ng/mL	*P* value
QT dispersion, ms	40 (26.8–46.4)	33.4 (26.8–43.6)	20 (13.6–20)	< .001*
QTcmax, ms	435.7 (430–441.7)	432.7 (425–436.7)	413.3 (404.7–421.7)	< .001*
Tp-e/QTc	0.19 ± 0.01	0.19 ± 0.01	0.17 ± 0.01	< .001*

*Data are presented as median (Q1–Q3) or mean ± SD. *P < .05.*

Table 2. Correlation Analysis Between Biochemical Parameters and ECG Findings

Parameter	25(OH)D *r*	*P* value	PTH *r*	*P* value
QT dispersion	−0.214	.049*	−0.019	.865
QTcmax	−0.261	.016*	0.114	.300
QT	0.010	.928	0.249	.022*
QTmax	−0.029	.791	0.215	.048*

*r, correlation coefficient. *P < .05.*

Table 3. Linear Regression Analysis Showing the Effect of Vitamin D on QTcmax

Variable	B	SE	Beta	*t*	*P* value	95% CI
Vitamin D	−0.673	0.261	−0.272	−2.579	.012*	−1.193 to −0.154

*Model: $F = 6.653$, $P = .012$, $R^2 = 0.074$. *P < .05.*

two main groups (vitamin D < 20 vs. ≥ 20 ng/mL). However, QTmax, QTd, QTc, QTcmax, and QTcd were significantly higher in the group with low vitamin D. Tp-e max, Tp-e, Tp-ed, Tp-e/QT, and Tp-e/QTc values were also significantly elevated in the low-vitamin-D group, while Tp-e min did not differ. The detailed data are provided in Supplementary Tables S1–S2. In the three-group comparison (deficiency, insufficiency, sufficiency), QTmax was significantly higher in the insufficiency group than in the sufficiency group. QTd, QTc, and QTcd were significantly higher in both the deficiency and insufficiency groups than in the sufficient group. Tp-e–related parameters (Tp-e max, Tp-e, Tp-ed, Tp-e/QT, Tp-e/QTc) were also significantly elevated in the deficiency and insufficiency groups compared with sufficiency, with no significant difference between deficiency and insufficiency (Table 1). Correlation analyses revealed a negative association between 25(OH)D and both QTd and QTcmax. PTH showed a positive correlation with QT and QTmax (Table 2). Regression analysis demonstrated that serum vitamin D independently predicted QTcmax; each 1-unit increase in vitamin D was associated with a 0.673-unit decrease in QTcmax (Table 3).

Discussion

This study was conducted to evaluate the effect of vitamin D on ventricular arrhythmia. The effect of vitamin D on cardiovascular diseases is known to occur mainly through inhibition of inflammation. Its effects on inflammation are exerted by inhibiting NF-κB activation, regulating cytokine levels (IL-6, IL-8, IL-17A, IL-10, and TGF-β), reducing the receptors of these cytokines, and inhibiting the prostaglandin pathway. Additionally, vitamin D has cardiovascular effects through renin inhibition, regulation of serum parathyroid hormone levels and lipid profile, and modulation of cardiomyocyte and endothelial cell proliferation.[8] There are simple and non-invasive atrial and ventricular parameters on the ECG that can be used to predict arrhythmia. Those associated with ventricular repolarization abnormalities include QT, QTd, QTc, QTcd, Tp-e, Tp-ed, Tp-e/QT, Tp-e/QTc, JT, and JTc parameters.[9,10]

The duration from the beginning of ventricular depolarization to the end of repolarization is termed the QT interval on the ECG. The QT interval reflects both the duration of ventricular resting and electrical activity. There is also a sex-related difference, with females having slightly longer QT intervals.[11] In our study, however, no significant sex-related difference was found in QT or QTc parameters. Considering that sex-related differences in QT interval emerge after puberty and that our study population aged 5–17 years had predominantly prepubertal mean ages in both girls and boys, the absence of sex differences was an expected finding.

In a study investigating the effects of serum vitamin D levels on ventricular repolarization in healthy children and adolescents, participants were classified into three groups: "sufficient," "insufficient," and "deficient." ECG parameters, body mass index, and systolic and diastolic blood pressures were evaluated. The insufficiency group had significantly different QT and Tp-e/QT parameters compared with the sufficient group. Moreover, serum vitamin D levels showed a negative correlation with JTc, Tp-e, and Tp-e/QT, and linear regression analysis revealed that a decrease in serum vitamin D below normal values was an independent risk factor for JTc prolongation. The authors concluded that declining vitamin D levels might cause ventricular repolarization abnormalities and increase susceptibility to ventricular arrhythmias through prolonged repolarization.[12]

Öner et al. evaluated vitamin D levels and QT dispersion in children presenting with chest pain. In this study involving 41 patients aged 7–17 years, participants were categorized into two groups: those with chest pain and those without. No significant differences were found between these groups regarding vitamin D levels or QT dispersion.[13]

The study examined the association between vitamin D deficiency and ventricular repolarization abnormalities in adolescents. Participants were grouped according to serum vitamin D levels as deficiency (<20 ng/ml), insufficiency (20–30 ng/ml), and age-matched controls (>30 ng/ml). They found that the deficiency and insufficiency groups had longer Tp-e intervals and higher Tp-e/QTc and Tp-e/JTp ratios. QTd and JTd parameters were also higher in the vitamin D–deficient group compared with the other two groups.[2]

Another study evaluated the effects of 25-hydroxyvitamin D levels on QT interval duration and dispersion in patients with type 2 diabetes. A total of 253 diabetic patients and 170 healthy controls were included. QTc and QTcd were longer in diabetic patients compared to controls. The authors concluded that diabetic patients with prolonged QTc and increased QTcd were more likely to have vitamin D deficiency.[14]

In our study, consistent with previous reports, QTmax, QTd, QTc, QTcmax, QTcd, Tp-e, and Tp-emax durations were significantly longer, and Tp-e/QT and Tp-e/QTc ratios were significantly higher in the vitamin D deficiency and insufficiency groups compared with the sufficient group. However, no significant differences were detected between the deficiency and insufficiency groups.

A negative correlation was found between serum 25(OH)D and QTd and QTcmax. These findings suggest that increased inflammation accompanying vitamin D deficiency and the subsequent rise in inflammatory cytokines may enhance oxidative stress, cause collagen loss and fibrosis, and thereby impair ventricular conduction distribution.

There are also studies examining the relationship between serum PTH levels and ECG parameters. In our study, although no relationship was found between PTH and Tp-e, Tp-e/QT, or Tp-e/QTc, PTH was associated with QTmax and QT. A positive correlation was detected between PTH and QTmax/QT, and a 1-unit increase in PTH was found to have an effect corresponding to a 0.240-unit increase in QT. In a study involving 41 patients with primary hyperparathyroidism and 40 controls, Tp-e, Tp-e/QT, and Tp-e/QTc parameters were compared, and positive correlations were observed between corrected calcium and PTH levels and these parameters. The authors concluded that both high PTH and high calcium levels may have arrhythmogenic potential.[15] We think that the mechanism underlying this effect may resemble that in vitamin D deficiency, where increased PTH contributes to systemic inflammation and subsequently prolongs ventricular repolarization.

There is a view that the normal serum 25(OH)D levels required for adequate bone mineralization should be determined based on the 25(OH)D level that does not lead to elevated PTH. Although there is more consensus on the vitamin D levels necessary for bone metabolism and calcium–phosphorus homeostasis, the threshold for cardiovascular effects remains controversial. In 2013, Atapattu and Högler reported this value as 13.6 ng/ml.[16] A more recent study by Kang et al. in 2017 determined the 25(OH)D level required for maximal PTH suppression as 18 ng/ml.[17] According to the Pediatric Endocrine Society and the 2016 global consensus recommendations, serum 25(OH)D levels in children are categorized as follows: deficiency <12 ng/ml, insufficiency 12–20 ng/ml, sufficiency 20–100 ng/ml, and toxicity >100 ng/ml.[18] In our study, we used this classification along with the definitions used in similar studies. Although no significant difference was observed between the deficiency and insufficiency groups, the significantly better parameters in the sufficient group suggest that ventricular repolarization may be more stable when vitamin D levels exceed 20 ng/mL.

The findings of our study suggest that vitamin D deficiency may induce subclinical electrical instability in children. Although no clinical arrhythmia was detected, the observed alterations in repolarization parameters may serve as early predictors of increased susceptibility to arrhythmia in later life. Evaluating data using both two- and three-group vitamin D classifications, along with the cross-sectional design and the absence of symptomatic arrhythmia data, limits the ability to demonstrate causality. The lack of long-term pediatric follow-up studies on this topic in the literature is also notable.

Limitations

This study has some limitations. Its single-center design and relatively small sample size may limit the generalizability of the results. Ventricular repolarization parameters were assessed using standard 12-lead electrocardiography, and no long-term follow-up or clinical arrhythmic outcomes were evaluated. Additionally, the observational design does not allow causal inferences.

Conclusion

In conclusion, it demonstrates that vitamin D levels are an important determinant of cardiac repolarization and that deficiency in childhood should not be overlooked. Therefore, routinely assessing vitamin D levels in children and addressing deficiencies is important not only for bone health but also for reducing cardiovascular risk.

Declarations

Animal and Human Rights Statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of Helsinki and its later amendments, or comparable ethical standards.

Informed Consent

Written informed consent was obtained from the parents or legal guardians of all participants prior to enrollment.

Data Availability

The datasets used and/or analyzed during the current study are not publicly available due to patient privacy reasons, but are available from the corresponding author on reasonable request.

Conflict of Interest

The authors declare no conflict of interest.

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None.

Author Contributions (CRediT Taxonomy)

Conceptualization: D.A., P.D.
Methodology: D.A., P.D.
Investigation: D.A., B.E., R.P.
Data Curation: D.A.
Formal Analysis: D.A., P.D.
Writing – Original Draft: D.A.
Writing – Review & Editing: P.D., B.E., R.P.
Supervision: P.D.

AI Usage Disclosure

The authors declare that no AI-assisted technologies were used.

Abbreviations

ECG: Electrocardiography
PTH: Parathyroid hormone
QT: Qt interval
QTc: Corrected qt interval
QTcd: Corrected qt dispersion
QTd: Qt dispersion
Tp-e: Tpeak–tend interval
Tp-ed: Tpeak–tend dispersion

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