BALNEO RESEARCH Balneo and PRM Research Journal
English Edition
e ISSN 2734 - 8458 p ISSN 2734 - 844X ROMANIAN ASSOCIATION OF BALNEOLOGY

Observational Clinical Studies

Evaluating the Impact of Vitamin D Deficiency on Paediatric ECG Parameters: A Cross-Sectional Study

Diana Jecan-Toader [1,2], Ioan-Alexandru Minciuna [3,4], Raluca Tomoaia [3,4], Radu Rosu [3,4], Gabriel Gusetu[3,4], Andreea-Liana Bot (Rachisan)[1,5], Alexandru Jecan[6,7], Marius Colceriu[2], Gabriel Cismaru*[3,4] and Simona Cainap [2,8]

Citation: Jecan-Toader D., Minciuna I.A., Tomoaia R., Rosu R., Gusetu G., Bot (Rachisan) A.L., Jecan A., Colceriu M., Cismaru G., Cainap S. - Evaluating the Impact of Vitamin D Deficiency on Paediatric ECG Parameters: A cross-sectional study *Balneo and PRM Research Journal* ***2026**, 17(2): 1044*

Academic Editor(s):
Constantin Munteanu

Reviewer Officer:
Viorela Bembea

Production Officer:
Camil Filimon

Received: 23.02.2026
Published: 10.07.2026

Reviewers:
Alexandra Sporici
Andreea Iulia Vladulescu Trandafir

Publisher's Note:
Balneo and PRM Research Journal stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.

1 Department 2—Medical Sciences, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania;
2 2nd Pediatric Clinic, Emergency Clinical Hospital for Children, Cluj-Napoca, Romania;
3 5th Department of Internal Medicine, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania;
4 Cardiology Department, Rehabilitation Hospital, Cluj-Napoca, Romania;
5 Department of Pediatric Nephrology, Emergency Clinical Hospital for Children, Cluj-Napoca, Romania;
6 Department of Orthopedics and Traumatology, Iuliu Hatieganu University of Medicine and Pharmacy, Cluj-Napoca, Romania;
7 Leon Daniello Pneumology Hospital, Cluj Napoca, Romania;
8 Department of Mother and Child, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania

* Correspondence: gabi_cismaru@yahoo.com

Abstract: Vitamin D deficiency is a widespread health issue within the paediatric population. In recent years, vitamin D deficiency has been linked to numerous cardiovascular diseases such as atherosclerosis, arterial hypertension, myocardial infarction, and heart failure. Previous studies have also suggested a possible relationship between low vitamin D levels and electrocardiographic changes in both adult and paediatric populations. In this retrospective study, we investigated whether circulating 25-OH-vitamin D levels are associated with changes in ventricular repolarisation, depolarisation, and atrial conduction on ECG in the paediatric population. A total of 66 patients aged 1 to 17 years were divided into three groups according to serum 25-OH Vitamin D levels: sufficient (>30 ng/ml; n=21), insufficient (20-30ng/ml; n=30) and deficient (<20 ng/ml; n=15). QRS duration, QTc interval, Tpe interval, Tpe/QTc ratio, JTp interval, Tpe/JTp ratio, JTc interval, PR interval, P-wave duration, LAP interval and P-wave dispersion were assessed. Associations between vitamin D status and the measured ECG parameters were analyzed using one-way analysis of variance (ANOVA). No statistically significant associations were found between vitamin D level and any of the evaluated ECG parameters ($p>0.05$). These findings do not support an independent association between vitamin D status and ECG changes related to ventricular repolarisation, depolarisation, or atrial conduction in the paediatric population.

Keywords: vitamin D, electrocardiogram, ventricular depolarization, ventricular repolarization, atrial conduction, pediatrics

1. Introduction

Vitamin D is part of the fat-soluble vitamin family, with functions similar to those of steroid hormones [1]. Traditionally, vitamin D has been recognised as an important modulator of skeletal and mineral metabolism, but recent studies have demonstrated its involvement in numerous physiological patterns beyond what was commonly thought. Vitamin D receptors are found in most human cells, including

cardiovascular cells such as myocytes and endothelial cells [2,3]. Located in the nucleus of cells, vitamin D receptors exert both genomic and non-genomic effects. The genomic transcriptional effects are described as regulating the expression of thousands of genes, while non-genomic effects are represented by rapid responses involving several second messengers and transmembrane transport of ions [4,5]. These mechanisms have the potential to explain why vitamin D deficiency has been linked to numerous cardiovascular diseases such as arterial hypertension, left ventricular hypertrophy, atherosclerosis, myocardial infarction, heart failure and endothelial dysfunction [2,6,7].

It has been hypothesised that vitamin D might have a role in the pathogenesis of arrhythmias. Due to its antioxidant properties, it may influence the production of reactive oxygen species in the atria and therefore, the formation of a proarrhythmic substrate [8]. This assumption is supported by studies reporting an association between vitamin D levels and left atrial ECG abnormalities, including atrial fibrillation [8,9]. Furthermore, research in animal models has shown an association between vitamin D levels and QT interval, independent of calcium levels [10]. Past studies have also reported associations between vitamin D levels and ventricular conduction delay, along with ventricular repolarisation [11,12]. However, much of the available clinical evidence is derived from observational studies, and the reported associations may therefore be influenced by residual confounding related to comorbidities, medication use, and overall cardiovascular risk. Furthermore, data in the paediatric population remain limited, and adult findings cannot be directly extrapolated to children because age-related differences in cardiac electrophysiology, autonomic maturation, vitamin D metabolism, and arrhythmia mechanisms may modify the relationship between vitamin D status and ECG parameters. Since ventricular repolarisation abnormalities have been associated with ventricular arrhythmias and sudden cardiac death, identifying the factors contributing to such abnormalities is critical for effective intervention [13].

Considering the growing evidence linking vitamin D deficiency and arrhythmias in adults, the scarcity of studies conducted in the paediatric population, as well as the high prevalence of vitamin D deficiency in children [14], it is crucial to identify whether vitamin D deficiency increases the arrhythmic risk in children. The present study sought to evaluate whether low vitamin D levels are associated with changes in ventricular repolarisation, depolarisation, and atrial conduction as seen on ECG that could predispose children to arrhythmias.

2. Materials and Methods

A total of 121 subjects aged 1 to 17 years were retrospectively selected from all consecutive patients who visited a tertiary paediatric hospital between January 2018 and January 2022. Inclusion criteria required that all included subjects were paediatric patients over one year old who underwent an electrocardiogram (ECG) and had their serum 25-hydroxy-vitamin D levels measured during the same visit. Exclusion criteria consisted of congenital or acquired heart disease, arterial hypertension, obesity (defined as body mass index at or above the 95th percentile for age and sex), serum electrolyte disturbances, thyroid dysfunction, diabetes mellitus, renal failure, liver disease, malabsorption disorders, neurological illness, collagen tissue disease, chronic inflammatory disease, current vitamin D supplementation as documented in the medical records or the use of drugs known to cause changes in ECG. Serum electrolyte disturbances were defined as electrolyte values outside the age-adjusted reference ranges used by our institution's clinical laboratory. After applying the exclusion criteria, a total of 66 subjects remained eligible for inclusion.

Data regarding gender, age, medical history, anthropometric measurements, physical findings and levels of 25-OH-vitamin D, total and ionic calcium, magnesium, and phosphorus were extracted from patient files and anonymized. Serum 25-hydroxy-vitamin D levels were measured from fasted blood samples using the LI-

AISON 25 OH Vitamin D TOTAL Assay Kit (DIASORIN Inc. Stillwater, MN, USA), as per our institutional protocol. The ECGs were also collected from the patient files and anonymized.

In our institution all ECGs are performed as standard 12 lead electrocardiograms obtained in the supine position after 10 minutes of rest, set at an amplitude of 10 mV/mm amplitude and a speed of 25 mm/second using the Edan SE-12 EXPRESS device. The ECGs were reviewed using a magnifying glass by two independent cardiologists who were blinded to the levels of serum vitamin D of the subjects. Ventricular depolarisation as seen on the ECG was assessed using QRS duration (QRSd). QRSd was defined as the duration from the start to the end of the complex. Ventricular depolarisation and repolarisation ware evaluated using corrected QT interval (QTc), while Tpeak to Tend (Tpe), Tpe/QTc, J to Tpeak (JTp), Tpe/JTp and JTc were used to evaluate the ventricular repolarisation. The QTc interval was evaluated by measuring the QT interval and correcting it using Bazzett's formula. Tpe was measured from the peak of the T wave to its end and JTp from the J point to the peak of the T wave. JTc was defined as the difference between QTc and the duration of QRS. Atrial conduction markers evaluated were P wave duration (PWD), PR interval, left atrial P wave duration (LAP), and P wave dispersion (Pd). PWD was measured from the earliest initial deflection from the isoelectric line in any lead to the time of the latest activation in any lead. PR interval was defined as the duration from the start of the P wave to the start of the QRS complex. LAP was measured from the point of highest negative deflection (-dV/dt max) in lead V1 or V2 to the end of the P-wave in any of the leads and Pd was defined as the difference between the longest P wave and the shortest one.

For statistical analysis, subjects were categorized according to their serum 25-hydroxy vitamin D levels as deficient (less than 20 ng/ml), insufficient (20-30 ng/ml), and sufficient (greater than 30 ng/ml). The thresholds were chosen in accordance with the recommendations from most authorities [15]. Given the age-dependent variation of ECG parameters in children, age was compared between study groups to assess potential confounding effects.

Depending on the type and distribution of the data, variables were expressed as either mean ± SD, median [IQR] or frequencies. Normality was tested using the Kolmogorov–Smirnov test. All patients were divided into three groups, according to the value of vitamin D levels (sufficient, deficient, insufficient). Baseline parameters in the three groups were compared using the one-way analysis of variance (ANOVA) when the variables were continuous and the Chi square test in the case of categorical data. Levene's test for the equality of variances was performed before each presented comparison and a logarithmic transformation was performed when p was <0.05. After each analysis of variance, a post-hoc test using Sheffe's method was performed for pairwise comparison of subgroups. Statistical analysis was performed using MedCalc Statistical Software 19.6.1 (MedCalc Software Ltd., Ostend, Belgium; http://www.medcalc.org; 2020). A p-value of <0.05 was considered significant. The ethics committee approved the study protocol.

3. Results

The study included 66 subjects, between the ages of 1 and 17, with a mean age of 10 years. Of these, 27 (40,9%) were male and 39 (59,1%) were female. Baseline characteristics of all patients are presented in Table 1. Most patients were admitted for investigation of an innocent murmur (30.30%), followed by tachycardia (21,21%), auscultatory arrhythmia (16,66%), syncope (9,09%), ponderal hypotrophy (7,57%), and bradycardia (4,54%). Additionally, two patients were admitted for dyslipidemia, and one patient each for hydronephrosis, pharyngitis, acrocyanosis, thoracic pain, and reevaluation after poststreptococcal arthritis.

Variables	Mean	Median	SD	25 - 75 P
Age (years)	10.170	11.000	4.1299	6.000 to 14.000
Weight (kg)	36.494	34.500	16.2424	22.000 to 49.000
Height (cm)	140.121	143.500	24.2909	118.000 to 158.000
BMI (kg/m2)	17.40	16.500	3.0696	15.400 to 19.500
Ionized calcium(mmol/l)	1.2 (0.04)	1.190	0.04338	1.170 to 1.220
Total calcium (mg/dl)	9.913	9.935	0.3441	9.760 to 10.160
Phosphorus (mg/dl)	4.593	4.720	0.6155	4.150 to 5.000
Magnesium (mg/dl)	1.932	1.950	0.1701	1.830 to 2.050
25-OH Vitamin D (ng/ml)	27.638	26.500	10.8708	20.100 to 31.400
Heart rate (beats/minute)	82	80.000	17.6243	70.000 to 93.000
QRSd (msec)	78.485	80.000	8.8130	70.000 to 80.000
QTc (msec)	413.727	414.500	21.0848	397.000 to 428.000
Tpe, (msec)	85.455	80.000	9.7951	80.000 to 90.000
JTp (msec)	193.636	200.000	26.9265	180.000 to 210.000
Tpe/JTp	0.448	0.438	0.07077	0.400 to 0.500
Tpe/QTc	0.207	0.203	0.02667	0.189 to 0.224
JTc (msec)	335.242	337.000	24.3184	318.000 to 352.000
PR (msec)	135.455	130.000	22.5444	120.000 to 140.000
PWD (msec)	88.182	90.000	10.5114	80.000 to 90.000
Pd (msec)	40.909	40.000	8.7226	40.000 to 50.000
LAP (msec)	29.773	30.000	7.5667	20.000 to 40.000

Table 1. Baseline characteristics of all patients

The three study groups were compared for age, gender, weight, height, BMI, ionized calc ium, total calcium, phosphorus, magnesium, and 25-OH-vitamin D levels. No significant differences were found between the study groups, except for 25-OH-vitamin D level (Table 2).

	Deficient (N=15)	Insufficient(N=30)	Sufficient(N=21)	p
Age (years)	10.733 ± 3.8631	10.542 ± 4.1388	9.238 ± 4.3348	0.45
Gender (M/F)	5/10	11/19	11/10	0.42 (Chi2 = 1,723)
Weight (kg)	38.000 ± 17.4233	37.170 ± 15.5440	34.452 ± 16.9646	0.78
Height (cm)	141.400 ± 23.8202	142.333 ± 23.3420	136.048 ± 26.5414	0.65
BMI (kg/m2)	17.740 ± 3.9946	17.270 ± 2.8417	17.410 ± 2.7630	0.89
Ionized calcium(mmol/l)	1.194 ± 0.03291	1.200 ± 0.04709	1.194 ± 0.04599	0.87
Total calcium (mg/dl)	9.903 ± 0.3003	9.907 ± 0.3567	9.928 ± 0.3698	0.97
Phosphorus (mg/dl)	4.648 ± 0.5915	4.481 ± 0.5893	4.715 ± 0.6677	0.38
Magnesium (mg/dl)	1.957 ± 0.09728	1.914 ± 0.2131	1.940 ± 0.1434	0.7
25-OH Vitamin D (ng/ml)	15.753 ± 2.6859	25.007 ± 3.0974	39.886 ± 9.5540	**< 0.001**

Table 2. Demographic characteristics and laboratory findings of the 3 study groups

The ECG parameters of the three study groups, including heart rate, QRS duration, corrected QT interval, Tpe interval, JTp interval, Tpe/JTp ratio, Tpe/QTc ratio, JTc interval, PR interval, P-wave duration, P-wave dispersion and left atrial P-wave interval, are presented in Table 3. The statistical analysis performed found no significant difference between the three compared groups.

	Deficient (N=15)		Insufficient(N=30)		Sufficient(N=21)		
	Mean	SD	Mean	SD	Mean	SD	p
Heart rate (beats/minute)	87.667	19.3895	81.167	19.4211	79.143	12.8736	0.34
QRSd (msec)	79.333	10.3280	78.333	9.1287	78.095	7.4960	0.91 0.91
QTc (msec)	422.467	20.8322	411.867	21.7648	410.143	19.4481	0.18
Tpe, (msec)	86.667	11.1270	86.000	8.5501	83.810	10.7127	0.64
JTp (msec)	187.333	26.3131	194.333	30.0211	197.143	22.8348	0.20
Tpe/JTp	0.469	0.07397	0.451	0.07324	0.429	0.06283	0.24
Tpe/QTc	0.206	0.03026	0.210	0.02721	0.204	0.02405	0.77
JTc (msec)	343.133	25.9391	333.533	25.6417	332.048	20.8770	0.36
PR (msec)	138.667	17.6743	133.000	16.8462	136.667	31.6754	0.70
PWD (msec)	90.667	11.6292	87.333	9.8027	87.619	10.9109	0.59
Pd (msec)	39.333	7.9881	41.667	8.7428	40.952	9.4365	0.7
LAP (msec)	32.667	7.0373	28.167	7.4837	30.000	7.7460	0.17

Table 3. The ECG characteristics of the three study groups

4. Discussion

In the current study, we sought to determinewhether vitamin D levels are associated with ventricular depolarisation, ventricular repolarisation, and atrial conduction on ECG in the paediatric population. Our statistical analysis did not reveal any significant association between circulating 25-OH-vitamin D levels and paediatric ECG abnomalities.

Atrial fibrillation, the most common arrhythmia in adults, carries a significant burden on society, caused by its important thromboembolic risk and impact on cardiac function. Observational studies have identified an increased incidence of atrial fibrillation during winter months, following a seasonal pattern similar to that of vitamin D deficiency in the northern hemisphere [16,17]. This led to the hypothesis that atrial fibrillation is associated with low levels of vitamin D. It was proposed that vitamin D may inhibit fibrosis formation in the atria through its antioxidant properties [8]. This assumption was supported by a study by Canpolat et al. which identified a direct correlation between low levels of vitamin D and more extensive left atrial fibrosis [18]. Furthermore, an experimental study on rabbit hearts found a direct electrophysiological effect of 1,25-OH-vitamin D on the left atrium, preventing and terminating atrial fibrillation [8,19]. Observational studies have also linked vitamin D deficiency to atrial fibrillation [20,21]. Moreover, Anees et al. reported an association between vitamin D deficiency and an increased risk of left atrial, defined as an increased PR interval and P-wave duration [9], that could predispose adults to atrial

fibrillation [22]. Although our study involved paediatric participants, a demographic where atrial fibrillations is uncommon, we sought to investigate whether vitamin D deficiency might affect the same ECG markers associated with atrial fibrillation in adults, potentially serving as indicators of future risk. However, our study did not identify any statistically significant associations between vitamin D levels and any of the left atrial parameters studied (PR interval, PWD, Pd, and LAS). It is plausible that the mechanism through which vitamin D deficiency acts on the risks of developing atrial fibrillation requires a longer time to manifest. However, in light of recent large studies and meta-analyses that found no association between circulating vitamin D levels and atrial fibrillation, the relationship between vitamin D and atrial ECG abnormalities remains improbable [23,24].

Regarding ventricular depolarization, our data did not identify statistically significant associations between vitamin D levels and QRS duration. This contrasts with the results reported by Tuliani et al., who identified associations between vitamin D deficiency and major electrocardiographic abnormalities, including ventricular conduction defects in adults [11]. However ,it is important to note that the study used a higher threshold for vitamin D deficiency (<40 ng/ml) that the one used in our study. The difference in thresholds, along with the employment of a different study population, might account for the contrasting results.

There have been reports of improvement in the burden of ventricular extrasystoles following vitamin D supplementation [25–27]. This led to the hypothesis that vitamin D could influence ventricular repolarisation. Ventricular repolarisation abnormalities have historically been associated with malignant ventricular arrhythmias and sudden cardiac death [28]. Considering the severity of these conditions, identifying at-risk patients has become a central point of research in the field. This warranted the development of the first clinical markers for arrhythmic risk: the QT and the corrected QT (QTc) intervals. Normal QTc values are considered as between 360 and 460 ms for both sexes, as per the European Society of Cardiology guidelines [29]. Proarrhythmic risk has been associated with extreme value, being later defined as Short QT Syndrome and Long QT Syndrome [30]. Both QT and QTc have the limitation of being one-dimensional in predicting arrhythmogenesis. This led to the development of new QT variability evaluating methods, such as QT dispersion (QTd), Tpeak to Tend (Tpe), Tpe/QTc, J to Tpeak (JTp), Tpe/JTp and corrected JT (JTc) distance [30].

Our study did not identify any statistically significant associations between circulating vitamin D levels and various measures of ventricular repolarization, including the QTc interval, the Tpe interval, the JTp interval, the Tpe / JTp ratio, the Tpe / QTc ratio, and the JTc interval. Our findings do not support an association between ventricular repolarisation and vitamin D levels. Contrastingly, a study by Kaya H. on 400 adults, divided into two equal groups based on their vitamin D levels, concluded that vitamin D levels were inversely correlated with ventricular repolarisation markers, including Tpe interval, Tpe/QTc ratio and Tpe/QT ratio. It is important to note that the serum vitamin D threshold for deficiency used in this study was below 10 ng/ml, much lower than in our methodology [31]. Similarly, a smaller study by Bekdas et al. found an inverse association between vitamin D levels and ventricular repolarisation parameters, such as Tpe, JTc and Tpe/QT. However, this study also employed different vitamin D level thresholds as the ones used in our methodology, defining insufficiency as levels under 12 ng/ml, deficiency between 12 to 20 ng/ml, and sufficiency above 20 ng/ml [32]. It is possible that ventricular repolarization may only be affected when vitamin D levels fall below the thresholds utilized in our study. Bagrul et al. conducted a study on 150 adolescents divided into three equal study groups according to their 25-OH-vitamin D levels similar to those used in our methodology, and identified a relationship between low circulating vitamin D levels and increased Tpe interval, Tpe/QTc ratio and Tpe/Jtpeak ratios. However, the study did found no significant differences with respect to QTd and QTc values, aligning with

our findings regarding QTc [12]. It is noteworthy that their study included only children aged 10 and above, unlike ours, which included paediatric patients aged 1 to 17, potentially influencing the results. Furthermore, a recent study has evaluated the effects of vitamin D deficiency on the indices of cardiac electrophysiological balance (iCEB and iCEBc), defined as QT/QRS and QTc/QRS ratios. These markers not only assess ventricular repolarization but also ventricular depolarization, offering a global view of the ventricular electrical activity. The study revealed an inverse relationship between vitamin D levels and QT/QRS and QTc/QRS ratios [33]. While our research did not specifically investigate these markers of ventricular electrical activity, we did examine their components. Our results did not reveal any significant associations between vitamin D levels and QRS or QTc intervals. However, it's noteworthy that this does not disprove the possibility of a relationship between vitamin D and the indices of cardiac electrophysiological balance, but further investigations are necessary to study the potential associations between these markers and vitamin D levels.

It is therefore possible that the arrhythmogenic effects of vitamin D deficiency emerge only at lower circulating levels than the ones used in our study. Furthermore, while vitamin D has been more extensively linked to arrhythmias in adults, its effect in the paediatric population remains underexplored. The few studies conducted in children have involved small sample sizes and yielded conflicting results.

Considering the contradictory evidence in the literature and the public health importance of the issue, caused by the high prevalence of vitamin D deficiency, as well as the dangerous risks of arrhythmias, large experimental studies in paediatric subjects are needed to effectively determine if vitamin D is independently correlated with ECG anomalies. This may be particularly relevant in children with chronic diseases, neuromuscular disorders, reduced mobility, or prolonged recovery periods, in whom vitamin D deficiency may be more frequent and musculoskeletal, functional, and cardiovascular vulnerability may coexist. Such studies should particularly examine whether lower serum values of vitamin D have an effect on ECG markers.

The limitations of the present study include its small samples size and single-center retrospective observational nature. Furthermore, the retrospective design did not allow adjustment for seasonal variation in vitamin D measurements, which may represent a potential source of residual confounding. Given the exploratory nature of the analysis and the number of ECG parameters assessed, the possibility of type I error inflation due to multiple comparisons cannot be excluded. Although all ECGs were independently reviewed by two cardiologists, interobserver agreement was not formally evaluated using kappa statistics or intraclass correlation coefficients. Finally, an experimental model would better study the relationship between vitamin D and ECG abnormalities predisposing to arrhythmias.

5. Conclusions

In summary, our study did not identify any relationship between 25-hydroxy-vitamin D levels and multiple ECG markers, including P-wave duration,PR interval, left atrial P-wave duration, P-wave dispersion, QRS duration, corrected QT interval, Tpeak to Tend, Tpe/QTc, J to Tpeak, Tpe/JTp ratios, and JTc duration in children. These findings suggest that vitamin D status was not associated with measurable alterations in atrial conduction, ventricular depolarization, or ventricular repolarization parameters within our study population. However, given the relatively small sample size and retrospective design, the possibility of an association cannot be definitively excluded. Furthermore, considering the divergent and scarce evidence found in paediatric literature, large prospective studies should be conducted to elucidate this relationship. Achieving a global consensus on this issue could lead to public health strategies aimed at preventing and decreasing the incidence of arrhythmias in children, a particularly vulnerable population.

Author Contributions: Conceptualization, Diana Jecan-Toader and Simona Căinap; Data curation, Ioan-Alexandru Minciună, Radu Roșu and Gabriel Gușetu; Formal analysis, Raluca Tomoaia and Gabriel Cismaru; Investigation, Diana Jecan-Toader, Andreea-Liana Bot (Rachisan), Alexandru Jecan and Marius Colceriu; Methodology, Diana Jecan-Toader and Ioan-Alexandru Minciună; Supervision, Simona Căinap; Writing – original draft, Diana Jecan-Toader and Raluca Tomoaia; Writing – review & editing, Gabriel Cismaru and Simona Căinap.

Funding: This research received no external funding.

Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of the Clinical Emergency Hospital for Children, Cluj-Napoca – 8226 from April 2022.

Informed Consent Statement: Patient consent was waived due to the retrospective nature of the study, as well as because of the analysis of anonym clinical data.

The study was conducted in accordance with the applicable laws and regulations of the country in which it was performed.

Data Availability Statement: o The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Norman PE, Powell JT. Vitamin D and cardiovascular disease. Circ Res. 2014 Jan 17;114(2):379-93. doi: 10.1161/CIRCRESAHA.113.301241.
2. Andrukhova O, Slavic S, Zeitz U, Riesen SC, Heppelmann MS, Ambrisko TD, Markovic M, Kuebler WM, Erben RG. Vitamin D is a regulator of endothelial nitric oxide synthase and arterial stiffness in mice. Mol Endocrinol. 2014 Jan;28(1):53-64. doi: 10.1210/me.2013-1252.
3. Tishkoff DX, Nibbelink KA, Holmberg KH, Dandu L, Simpson RU. Functional vitamin D receptor (VDR) in the t-tubules of cardiac myocytes: VDR knockout cardiomyo-cyte contractility. Endocrinology. 2008 Feb;149(2):558-64. doi: 10.1210/en.2007-0805.
4. Norman AW. Minireview: vitamin D receptor: new assignments for an already busy receptor. Endocrinology. 2006 Dec;147(12):5542-8. doi: 10.1210/en.2006-0946.
5. Hii CS, Ferrante A. The Non-Genomic Actions of Vitamin D. Nutrients. 2016 Mar 2;8(3):135. doi: 10.3390/nu8030135.
6. Savastio S, Pozzi E, Tagliaferri F, Degrandi R, Cinquatti R, Rabbone I, Bona G. Vitamin D and Cardiovascular Risk: which Implications in Children? Int J Mol Sci. 2020 May 16;21(10):3536. doi: 10.3390/ijms21103536.
7. Surdu AM, Pînzariu O, Ciobanu DM, Negru AG, Căinap SS, Lazea C, Iacob D, Săraci G, Tirinescu D, Borda IM, Cismaru G. Vitamin D and Its Role in the Lipid Metabo-lism and the Development of Atherosclerosis. Biomedicines. 2021 Feb 9;9(2):172. doi: 10.3390/biomedicines9020172.
8. Thompson J, Nitiahpapand R, Bhatti P, Kourliouros A. Vitamin D deficiency and atrial fibrillation. Int J Cardiol. 2015 Apr 1;184:159-162. doi: 10.1016/j.ijcard.2015.02.012.
9. Anees MA, Ahmad MI, Chevli PA, Li Y, Soliman EZ. Association of vitamin D deficiency with electrocardiographic markers of left atrial abnormalities. Ann Noninva-sive Electrocardiol. 2019 May;24(3):e12626. doi: 10.1111/anec.12626.
10. Sood S, Reghunandanan R, Reghunandanan V, Gopinathan K, Sood AK. Effect of Vitamin D Deficiency on Electrocardiogram of Rats. Indian J. Exp. Biol. 1995, 33, 61–63.
11. Tuliani TA, Shenoy M, Deshmukh A, Rathod A, Pant S, Badheka AO, Levine D, Afonso L. Major electrocardiographic abnormalities and 25-hydroxy vitamin D deficiency: insights from National Health and Nutrition Examination Survey-III. Clin Cardiol. 2014 Nov;37(11):660-6. doi: 10.1002/clc.22329.
12. Bagrul D, Atik F. Association of vitamin D deficiency with ventricular repolar-ization abnormalities. Kardiol Pol. 2019 Aug 23;77(9):853-858. doi: 10.33963/KP.14888.
13. Monitillo F, Leone M, Rizzo C, Passantino A, Iacoviello M. Ventricular repolar-ization measures for arrhythmic risk stratification. World J Cardiol. 2016 Jan 26;8(1):57-73. doi: 10.4330/wjc.v8.i1.57.
14. Cashman KD, Dowling KG, Škrabáková Z, Gonzalez-Gross M, Valtueña J, De Henauw S, Moreno L, Damsgaard CT, Michaelsen KF, Mølgaard C, Jorde R, Grimnes G, Moschonis G, Mavrogianni C, Manios Y, Thamm M, Mensink GB, Rabenberg M, Busch MA, Cox L, Meadows S, Goldberg G, Prentice A, Dekker JM, Nijpels G, Pilz S, Swart KM, van Schoor NM, Lips P, Eiriksdottir G, Gudnason V, Cotch MF, Koskinen S, Lamberg-Allardt C, Durazo-Arvizu RA, Sempos CT, Kiely M. Vitamin D deficiency in Europe: pan-demic? Am J Clin Nutr. 2016 Apr;103(4):1033-44. doi: 10.3945/ajcn.115.120873.

15. Bischoff-Ferrari, H.A. Optimal Serum 25-Hydroxyvitamin D Levels for Multi-ple Health Outcomes. Adv. Exp. Med. Biol. 2014, 810, 500–525, doi:10.1007/978-1-4939-0437-2_28.
16. Frost L, Johnsen SP, Pedersen L, Husted S, Engholm G, Sørensen HT, Rothman KJ. Seasonal variation in hospital discharge diagnosis of atrial fibrillation: a population-based study. Epidemiology. 2002 Mar;13(2):211-5. doi: 10.1097/00001648-200203000-00017.
17. Murphy NF, Stewart S, MacIntyre K, Capewell S, McMurray JJ. Seasonal varia-tion in morbidity and mortality related to atrial fibrillation. Int J Cardiol. 2004 Nov;97(2):283-8. doi: 10.1016/j.ijcard.2004.03.041.
18. Canpolat U, Aytemir K, Hazirolan T, Özer N, Oto A. Relationship between vit-amin D level and left atrial fibrosis in patients with lone paroxysmal atrial fibrillation un-dergoing cryoballoon-based catheter ablation. J Cardiol. 2017 Jan;69(1):16-23. doi: 10.1016/j.jjcc.2016.06.012.
19. Hanafy DA, Chang SL, Lu YY, Chen YC, Kao YH, Huang JH, Chen SA, Chen YJ. Electromechanical effects of 1,25-dihydroxyvitamin d with antiatrial fibrillation activi-ties. J Cardiovasc Electrophysiol. 2014 Mar;25(3):317-23. doi: 10.1111/jce.12309.
20. Demir M, Uyan U, Melek M. The effects of vitamin D deficiency on atrial fibril-lation. Clin Appl Thromb Hemost. 2014 Jan;20(1):98-103. doi: 10.1177/1076029612453762.
21. Ozcan OU, Gurlek A, Gursoy E, Gerede DM, Erol C. Relation of vitamin D defi-ciency and new-onset atrial fibrillation among hypertensive patients. J Am Soc Hypertens. 2015 Apr;9(4):307-12. doi: 10.1016/j.jash.2015.01.009.
22. Smith JW, O'Neal WT, Shoemaker MB, Chen LY, Alonso A, Whalen SP, Soli-man EZ. PR-Interval Components and Atrial Fibrillation Risk (from the Atherosclerosis Risk in Communities Study). Am J Cardiol. 2017 Feb 1;119(3):466-472. doi: 10.1016/j.amjcard.2016.10.016.
23. Vitezova A, Cartolano NS, Heeringa J, Zillikens MC, Hofman A, Franco OH, Kiefte-de Jong JC. Vitamin D and the risk of atrial fibrillation--the Rotterdam Study. PLoS One. 2015 May 1;10(5):e0125161. doi: 10.1371/journal.pone.0125161.
24. Huang WL, Yang J, Yang J, Wang HB, Yang CJ, Yang Y. Vitamin D and new-onset atrial fibrillation: A meta-analysis of randomized controlled trials. Hellenic J Cardi-ol. 2018 Mar-Apr;59(2):72-77. doi: 10.1016/j.hjc.2017.11.006.
25. Cismaru G, Lazea C, Cainap S, Iacob D. Vitamin D as a Substitute of Catheter Ablation in a Paediatric Patient with High Burden Premature Ventricular Contractions: A Case Report. J Clin Diagn Res. 2021, doi:10.7860/JCDR/2021/48292.14987.
26. Kiuchi MG, Ramalho e Silva G, Rodrigues Paz LM, Chen S, Hoye NA, Lima Souto GL. Influence of Vitamin D Levels on the Treatment of Premature Ventricular Com-plexes in Patients with Chronic Kidney Disease. IJC Metab Endocr. 2017;14:53–58, doi:10.1016/j.ijcme.2017.01.002.
27. Cismaru G, Pop D, Zdrenghea D, Rosu R. Vitamin D Supplementation Re-placed Catheter Ablation in a Patient with Frequent Premature Ventricular Contractions. J. Cardiovasc Emergencies. 2021; 7:57–61, doi:10.2478/jce-2021-0005.
28. Castro-Torres Y, Carmona-Puerta R, Katholi RE. Ventricular repolarization markers for predicting malignant arrhythmias in clinical practice. World J Clin Cases. 2015 Aug 16;3(8):705-20. doi: 10.12998/wjcc.v3.i8.705.
29. Priori SG, Blomström-Lundqvist C, Mazzanti A, Blom N, Borggrefe M, Camm J, Elliott PM, Fitzsimons D, Hatala R, Hindricks G, Kirchhof P, Kjeldsen K, Kuck KH, Her-nandez-Madrid A, Nikolaou N, Norekvål TM, Spaulding C, Van Veldhuisen DJ; ESC Sci-entific Document Group. 2015 ESC Guidelines for the management of patients with ven-tricular arrhythmias and the prevention of sudden cardiac death: The Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC). Eur Heart J. 2015 Nov 1;36(41):2793-2867. doi: 10.1093/eurheartj/ehv316.
30. Tse G, Yan BP. Traditional and novel electrocardiographic conduction and re-polarization markers of sudden cardiac death. Europace. 2017 May 1;19(5):712-721. doi: 10.1093/europace/euw280.
31. Kaya H. Effect of vitamin D on novel ventricular repolarization indices. Int Arch Intern Med. 2019;3(2):017. doi:10.23937/2643-4466/1710017.
32. Bekdas M, Inanir M, Ilhan Z, Ildes E. Effects of serum vitamin D level on ven-tricular repolarization in children and adolescents. Bratisl Lek Listy. 2021;122(11):816-820. doi: 10.4149/BLL_2021_130.
33. Guzelcicek A, Kilinc E, Fedai H, Dedeoglu NF, Toprak K, Tascanov MB, Ocak M, Gungorer B, Kose D. Relationship Between Vitamin D Level and Index of CardioElec-trophysiological Balance in Children. Comb Chem High Throughput Screen. 2024;27(14):2096-2100. doi: 10.2174/1386207326666230816094807.