Original Article

Evaluation of the Relationship between Serum 25(OH) Vitamin D Levels and Cardiac Functions in Adolescent Athletes

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Abstract

Background: Vitamin D is a prohormone necessary for the optimal functioning of the locomotor and circulatory systems in humans. As a caveat, vitamin D metabolism is crucial for maintaining musculoskeletal and cardiovascular health for overexercising people, like athletes. Our study intended to explore the relationship between serum 25-hydroxy vitamin D (25(OH) D) levels and left ventricle/right ventricle (LV/RV) systolic and diastolic function in adolescent athletes using 2D Doppler studies. **Methods:** In our cross-sectional study, 100 adolescent athletes were divided into two groups: vitamin D insufficiency (25(OH)D<20 ng/mL) and vitamin D sufficiency (25(OH)D>20 ng/mL) with 30 males and 20 females in both groups. A detailed physical examination and basic biochemical tests were performed; serum 25(OH)D levels were determined, and an echocardiographic evaluation was performed. **Results:** We found that decreased serum 25(OH)D levels were associated with impairment in many indicators of cardiac function, such as left ventricular-right ventricular-interventricular septum peak systolic velocity (Sm) and Tei index, inflow peak early diastolic velocity and inflow peak late diastolic velocity ratio (E/A), annulus early diastolic myocardial peak velocity (E′), and E/E′ ratio. **Conclusions:** To protect cardiac functions in adolescent athletes, we suggest screening serum 25(OH) D levels during certain periods, such as fall and winter, and vitamin D supplementation if necessary.

Keywords: *Adolescent, athlete, echocardiography, vitamin D*

Introduction

Vitamin D is a steroid prohormone that has important effects on the musculoskeletal system, renin-angiotensin system (RAAS), cardiovascular system, and immunity.[1] Intense exercise puts a lot of stress on the musculoskeletal and cardiovascular systems of athletes; therefore, these systems need to be in good condition. Previous research has linked low vitamin D levels in athletes to delayed fracture healing, increased risk of infection, impaired immunity, adverse effects on vital organs such as the lungs and heart affecting athletic conditioning, adverse effects on diabetic control, increased neurological disease and imbalances in pain perception, and adverse effects on performance due to psychological disorders.[2] Similarly, giving vitamin D supplements to athletes has been demonstrated to improve physical performance, lower the incidence of fractures, shorten fracture healing time, and reduce the incidence of infectious diseases.[3]

Low vitamin D levels have adverse effects on the cardiovascular system, such as increasing the risk of atherosclerosis, coronary artery disease, acute myocardial infarction, stroke, hypertension, diabetes, heart failure, and atrial fibrillation.[4,5] In addition, vitamin D insufficiency can cause myocardial systolic and diastolic dysfunction by leading to conditions such as RAAS overactivation, systemic hypertension, myocardial fibrosis, cardiac hypertrophy, inflammation, oxidative stress, and endothelial dysfunction.[4] Studies

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have reported cardiac systolic and diastolic dysfunction in vitamin D deficiency and improvements in cardiac function with vitamin D uptake.[6-8] Pulsed and tissue Doppler imaging has been used successfully for many years to define cardiac systolic and diastolic dysfunction in the early stages.[9]

Compared to later periods in life, vitamin D deficiency is observed at a higher rate in infancy and adolescence due to the rapid growth and development that occurs during those stages. Therefore, it is very important to prevent vitamin D insufficiency in athletes at this age due to its possible negative effects on their health.[10] The effects of vitamin D levels on cardiac function have been well discussed in adult athletes; however, limited research is available for adolescent athletes in the literature.[11,12] In our study, we intended to explore the correlation between the serum levels of 25-hydroxy vitamin D (25(OH)D) and cardiac function in adolescent athletes by utilizing pulsed and tissue Doppler echocardiography methods.

Methods

Participants

Adolescents aged between 12 and 17 years were studied in this research. Participants had been involved in formal football and volleyball training and competitions for more than 12 months. Participants had a minimum of 8 h of training sessions (4 h for strength and the other 4 h for conditioning and tactical work within the team) and played one or two games per week. The adolescents live at 40 degrees latitude, where the study was conducted. Adolescent athletes were excluded if they had anemia or chronic diseases involving any system. None of the participants had taken any medication or supplements, including vitamin D, over the past 12 months. Also, the participants with congenital or acquired cardiac defects on echocardiography were excluded.

Procedures

This cross-sectional study was designed with 100 adolescent athletes (60 male and 40 female volunteers) aged between 12 and 17 and years. Participants who visited the pediatric outpatient clinics for periodic examinations between September 2023 and February 2024 were included. The study was designed to be done in autumn and winter periods to minimize the seasonal differences of sun exposure on vitamin D metabolism. Serum 25(OH)D levels were determined by the chemiluminescence-immunoassay method using The Cobas 8000 e801 analyzer (Roche Diagnostics, Mannheim, Germany). Statistically, we used the "consecutive sampling method" for this study. According to the consecutive sampling method, we limited the subject size to 100 adolescents [50 athletes in the vitamin D below reference range group (serum 25(OH)D< 20 ng/mL-insufficiency; 30 males and 20 females) and another 50 athletes in the vitamin D-normal/reference range group (serum 25(OH)D > 20 ng/mL-sufficiency; 30 males and 20 females)].[13]

During the study period, 118 adolescent athletes were admitted to our outpatient clinic. Among these, we excluded 10 athletes due to selection criteria (two with anemia, one with celiac disease, three with vitamin D supplementation, two with performance-enhancing supplements, and two with mitral valve insufficiency). For the consecutive sampling method, three male and five female athletes, who were the most recent appliciants, were also excluded, and the remaining 100 athletes underwent echocardiography investigation and were included in the statistical study.

The pediatric cardiologist performed an echocardiographic evaluation without the data of the serum 25(OH)D levels of the athletes. Medical history, physical examination, and laboratory tests serum [25(OH)D, calcium, phosphate, alkaline phosphatase, parathormone (iPTH), and hematocrit] were performed. Participants with elevated serum alkaline phosphatase and iPTH levels underwent radiologic imaging to exclude the rickets. The *z*-scores of anthropometric measurements were obtained from scales prepared from Turkish children's data.[14] After resting for 20 min, systolic and diastolic blood pressure were measured, and percentiles were calculated according to scales derived from Turkish children's data.[15]

The families or legal representatives of the adolescents gave their written approval for our study. Dated 10.05.2023 and with the number 2023-8/2, the ethics committee of our hospital granted the required clearance. Our research followed the principles laid out in the Declaration of Helsinki.

Echocardiographic evaluation

Vivid S60N (GE Vingmed Ultrasound AS Strandpromenaden 45, 3191 Horten, NORWAY) and 3Sc-Rs sector probes were used for echocardiography. Every measurement was averaged across three heart cycles in a row. We performed standard 2D, M-mode, and Doppler measurements in compliance with the recommendations set out by the American Society of Echocardiography. Left ventricular (LV) M-mode measurements were obtained from a parasternal long-axis perspective. Simpson's method was used to determine the ejection fraction (EF). The Devereux formula was used to determine the left ventricle mass (LVM) and was then divided by the body surface area to generate the LVM index (LVMI).[16]

Pulsed wave Doppler and tissue Doppler measurements were obtained in a four-chamber view. Ventricular diastolic flow velocities were obtained by pulsed wave Doppler. For this purpose, inflow peak early diastolic velocity (E) and inflow peak late diastolic velocity (A) measurements were performed. In tissue Doppler examination, the sample volume was placed in the lateral annulus of both ventricles and simultaneously in the basal part of the interventricular septum (IVS). Measurements of [E/A, annulus early diastolic myocardial peak velocity (E′), E/E′] were taken as indicators of diastolic function. In addition, [EF, peak systolic velocity (Sm)] measurements were taken as indicators of systolic function. The Tei index [TX-myocardial performance index (MPI)], which evaluates systolic and diastolic function together, was also calculated by tissue Doppler imaging. Low EF and Sm are indicators of systolic dysfunction, whereas low E/A ratio on pulsed Doppler examination, low E′ value, and high E/E′ ratio on tissue Doppler examination are indicators of diastolic dysfunction. In addition, a high Tei index indicates that systolic and diastolic functions are impaired together.[9]

Statistical analysis

The Statistical Package for the Social Sciences for Windows version 26.0 was used in this study. Categorical variables were expressed as *n* (%), while continuous variables were expressed as mean ± SD (standard deviation) for normal distributions and median and IQR (inter-quantile range) for non-normal distributions. The distribution and frequency of the data were analyzed using descriptive analysis, and the frequency data were compared between two separate groups using Chi-square testing. The Kolmogorov-Smirnov test was employed to analyze normality. To compare two independent groups, the Student's *t*-test was used if normally distributed, and the Mann-Whitney U test was used if not. The relationship between serum 25(OH)D levels and echocardiographic indicators of systolic and diastolic cardiac function were calculated using Spearman's correlation coefficient (rho = *r*). A Spearman correlation coefficient of 0.00-0.19 was regarded as "very weak," 0.20-0.39 as "weak," 0.40-0.59 as "moderate," 0.60-0.79 as "strong," and 0.80-1.0 as "very strong." Statistical studies used a significance threshold of <.05.

Results

Our study consisted of 100 adolescent athletes aged 12-17 years with a mean age of 14.3 ± 1.6 years. The female-to-male ratio was similar in the vitamin D levels insufficient and sufficient groups (20 females and 30 males). Demographic characteristics, anthropometric measurements, and laboratory tests, including calcium, phosphate, and hematocrit values, were similar in both groups. While serum 25(OH)D serum levels were lower in the group with vitamin D levels below the reference range [14 (7-17) ng/mL] than in the reference range group [24 (20-44) ng/mL] ($P < .001$), alkaline phosphatase and iPTH levels were higher in the below reference group than in the other group ($P < .001$, $P < .001$). Eight participants with elevated serum alkaline phosphatase and iPTH levels underwent radiologic imaging for rickets, and all were found to be normal. The serum calcium levels of all participants were within normal limits. There was no difference between the groups in terms of the duration of formal sports training and participation in competitions [median

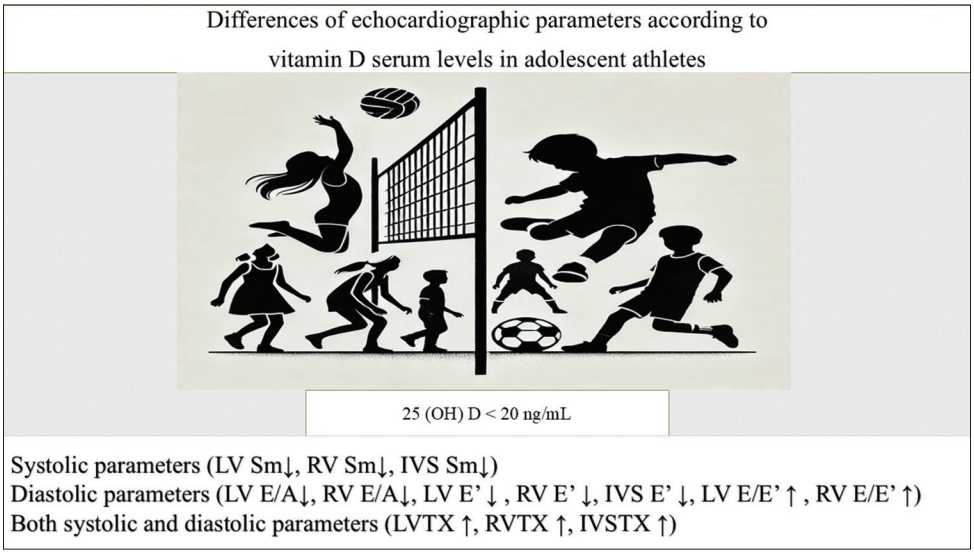

Figure 1: Schematized version of the results of our study. LV: left ventricle, RV: right ventricle, IVS: interventricular septum, Sm: peak systolic velocity, E: inflow peak early diastolic velocity, A: inflow peak late diastolic velocity, E′: annulus early diastolic myocardial peak velocity, TX: Tei index (myocardial performance index)

Table 1: Comparison of basic demographic, clinical, and laboratory findings between groups

	25 (OH) D < 20 ng/mL (*n*=50)	25 (OH) D >20 ng/mL (*n*=50)	*P*
Age, years	14.28±1.6	14.32±1,62	0.902
Height, cm	166.82±9.52	166.28±9.32	0.775
Weight, kg	57.2±8.86	57.26±10.17	0.975
BSA, m²	1.59±0.14	1.59±0.16	0.961
BMI, kg/m²	20.42±1.12	20.54±1.75	0.670
BMI *z* scores	0.28±0.06	0.26±0.04	0.800
SBP, mm Hg	122.94±4.61	123.56±6.36	0.578
SBP, percentile, median (IQR)	55 (40−69)	55 (20−81)	0.524
DBP, mm Hg	69.76±5.97	68.66±6.06	0.363
DBP, percentile, median (IQR)	55 (42−70)	56 (30−78)	0.264
HR, beats per minute	71.22±5.76	73.04±6.6	0.145
25(OH)D, ng/dL, median (IQR)	14 (7−17)	24 (20−44)	<.001*
Ca, mg/dL	9.63±0.33	9.61±0.31	0.698
P, mg/dL	4.06±0.37	3.97±0.35	0.196
Alp, IU/L, median (IQR)	220 (97−330)	167.5 (67−287)	<.001*
iPTH (pg/mL) median (IQR)	45.4 (35−65)	36.2 (20.3−45.5)	<.001*
Hct, (%)	38.6±2.02	38.4±2.96	0.694
Duration in formal sports training and competitions before the study (year), (IQR)	3 (1−8)	3 (1−9)	0.788

BSA: body surface area; BMI: body mass index; SPB: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate; 25 (OH) D: 25-hydroxy vitamin D, Ca: calcium, P: phosphate, iPTH: parathormone, Alp: alkaline phosphatase, Hct: hematocrit, **P* value is statistically significant

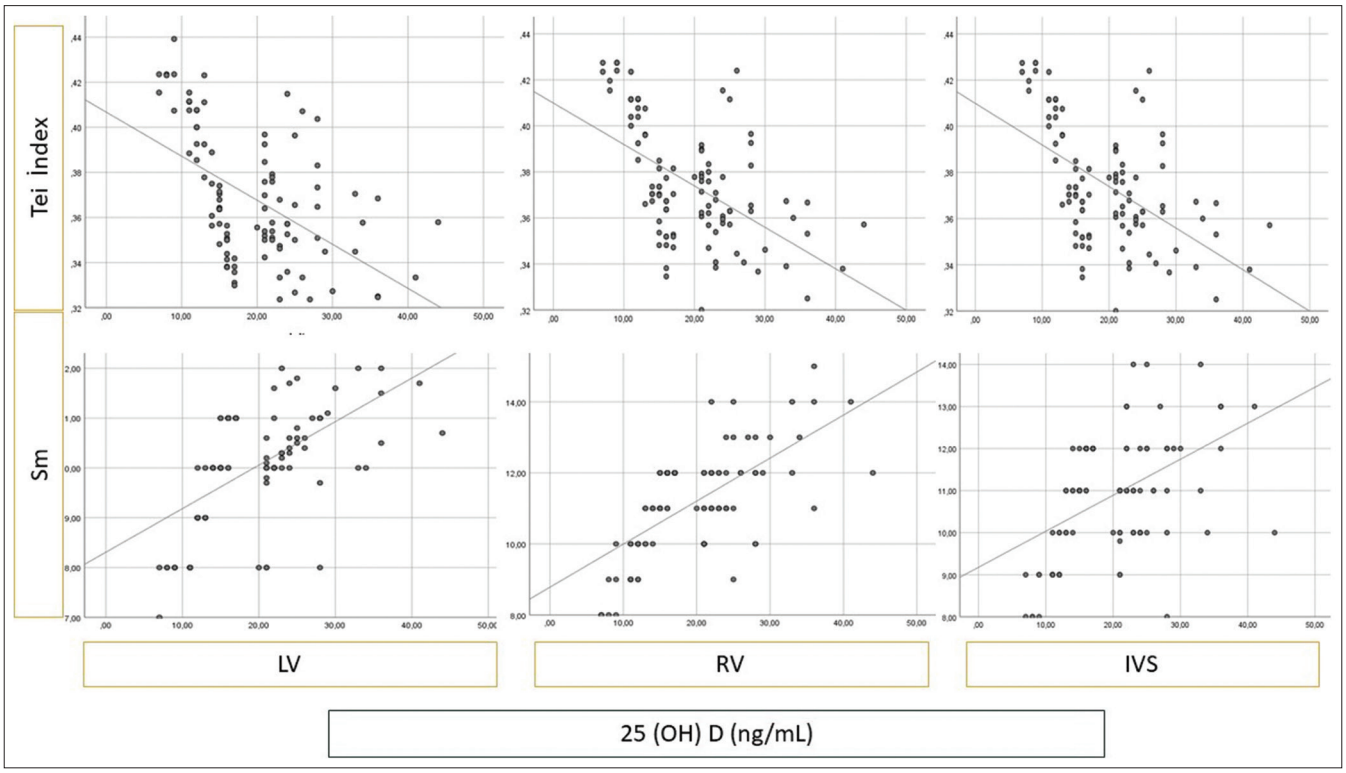

Figure 2: Correlation graphs between serum 25(OH)D levels and LV-RV-IVS Tei index and Sm. 25 (OH) D: 25-hydroxy vitamin D, LV: left ventricle, RV: right ventricle, IVS: interventricular septum, Sm: peak systolic velocity

3, IQR (1-8) years in the insufficiency group, median 3, IQR (1-9) years in the sufficiency group] [Table 1].

EF, LVMI, and M-mode echocardiographic parameters did not change between the two groups [Table 2].

On pulsed Doppler examination, LV and RV E/A ratios, indicators of diastolic function, were lower in the group with vitamin D levels below reference range (LV, *P* = .006; RV, *P* = .031). In tissue Doppler

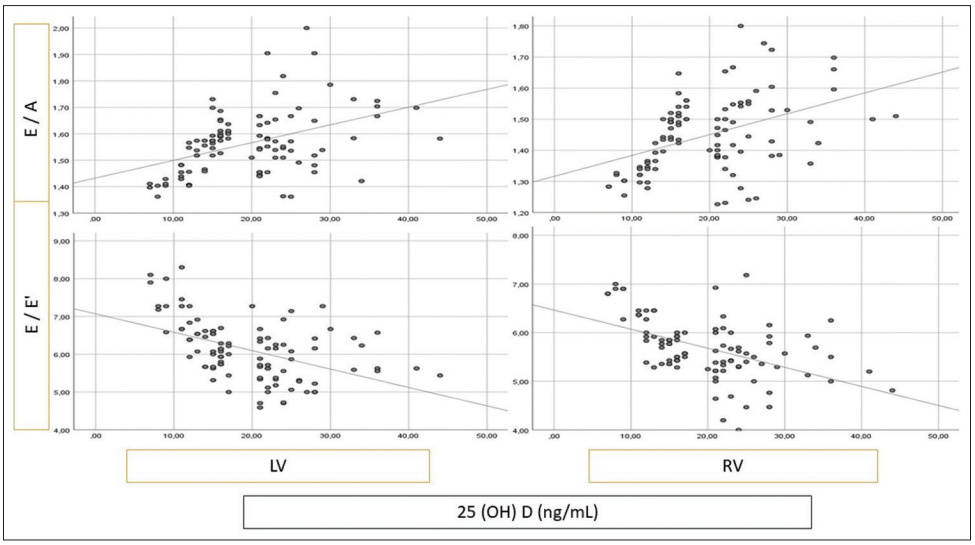

Figure 3: Correlation graphs between serum 25(OH)D levels and LV-RV E/A and E/E′ ratios. 25 (OH) D: 25-hydroxy vitamin D, LV: left ventricle, RV: right ventricle, E: inflow peak early diastolic velocity, A: inflow peak late diastolic velocity, E′: annulus early diastolic myocardial peak velocity

Table 2: Comparison of M-mode echocardiographic parameters between groups

	25 (OH) D <20 ng/mL (*n*=50)	25 (OH) D>20 ng/mL (*n*=50)	*P*
EF, %	69.84±2.61	70.12±2.34	0.573
FS, %	38.78±1.88	39.32±1.74	0.139
IVSd, mm	7.6±0.6	7.7±0.5	0.471
IVSs, mm	9.5±0.7	9.8±1	0.077
LVIDd, mm	42.4±3	43.3±3.2	0.145
LVIDs, mm	25.74±1.58	26.34±1.98	0.096
LVPWd, mm	7.6±0.7	7.7±0.8	0.812
LVPWs, mm	9.62±0.97	9.81±1.05	0.335
LAd, mm	34.28±1.97	34.88±1.96	0.131
LVMI, (g/m^2)	97.78±18.11	102.17±17.8	0.224

EF: ejection fraction, FS: fractional shortening, IVSd: interventricular septal end-diastolic dimension, IVSs: interventricular septal end-systolic dimension, LVIDd: left ventricle end-diastolic dimension, LVIDs: left ventricle end-systolic dimension, LVPWd: left ventricle posterior wall end-diastolic dimension, LVPWs: left ventricle posterior wall end-systolic dimension, LAd: left atrial dimension, LVMI: left ventricle mass index

examination, LV-RV and IVS E′ levels, which are indicators of diastolic function, were lower in the group with vitamin D levels below reference range (LV, $P < .001$; RV, $P < .001$; and IVS, $P = .005$). LV and RV E/E′ ratios, another indicator of diastolic function, were also higher in the group with vitamin D levels below reference range (LV, $P < .001$; RV, $P < .001$) [Table 3].

LV, RV, and IVS Sm levels, which are indicators of systolic function, were lower in the group with vitamin D levels below the reference range (LV, $P = .001$; RV, $P < .001$; and IVS, $P = .009$) [Table 3].

LV, RV, and IVS Tei index levels were higher in the group with vitamin D levels below the reference range ($P = .001.,001$, and. 001) [Table 3], indicating both systolic and diastolic dysfunction. Schematized versions of the results of our study are shown in Figure 1.

The Spearman correlation analysis revealed a strong correlation between serum 25(OH) D levels and RV E′ and RV Sm and a moderate correlation between LV E/A, LV E′, LV E/E′, LV Sm, LVTX, RV E/A, RV E/E′, RVTX, IVS E′, IVS Sm, and IVSTX. There was no correlation between EF and LVMI [Table 4] [Figures 2 and 3].

Discussion

Athletes need a perfect balance of properly functioning systems to cope with sports that require intense exercise. Undoubtedly, the musculoskeletal and cardiovascular systems are the most important components of this balance. Vitamin D is one of the most critical nutrients in the musculoskeletal and cardiovascular systems. It has been reported that vitamin D deficiency is very common in athletes, especially in adolescence, and therefore fractures occur more frequently in adolescent athletes with vitamin D deficiency.[17,18] Our study showed that decreased vitamin D levels have negative effects on cardiac systolic and diastolic functions in adolescent athletes.

Low serum 25(OH)D levels may lead to an increase in blood pressure due to increased vascular tone, overactivation of the RAAS, increased cardiac contractility, and hypertrophy of the cardiac muscles due to myocardial fibrosis. Vitamin D prevents hypertension and cardiac hypertrophy through genomic

Table 3: Comparison of pulsed and tissue doppler echocardiographic parameters between groups

	25 (OH) D <20 ng/mL (*n*=50)	25 (OH) D >20 ng/mL (*n*=50)	*P*
LV E, m/s	84.46±3.2	87.02±7.50	0.029*
LV A, m/s	55.24±1.85	54.66±4.1	0.364
LV E/A	1.52±0.08	1.6±0.14	0.006*
LV Sm, cm/s	9.64±1.2	10.41±0.97	0.001*
LV E′, cm/s	13.28±1.82	15.28±2.15	<.001*
LV A′, cm/s	8.67±1.04	8.96±1.03	0.151
LV E′/A′	1.54±1.51	1.71±0.2	<.001*
LV E/E′	6.45±0.73	5.77±0.69	<.001*
LV IVCT, ms	53.64±2.95	52.14±5.25	0.081
LV IVRT, ms	48.9±2.6	47.5±4.46	0.058
LV ET, ms	272.2±9.96	277.52±18.05	0.071
LVTX	0.38±0.03	0.36±0.02	0.001*
RV E, m/s	73.64±3.54	76.44±8.79	0.039*
RV A, m/s	51.86±1.4	52.08±6.06	0.803
RV E/A	1.42±0.1	1.47±0.14	0.031*
RV Sm, cm/s	10.6±1.29	11.72±1.33	<.001*
RV E′, cm/s	12.48±1.43	14.16±1.69	<.001*
RV A′, cm/s	8.56±0.79	8.86±1.08	0.116
RV E′/A′	1.46±0.15	1.61±0.15	<.001*
RV E/E′	5.95±0.49	5.44±0.61	<.001*
RV IVCT, ms	54.18±2.75	53.04±4.43	0.131
RV IVRT, ms	47.3±2.34	45.74±4.09	0.021*
RV ET, ms	265.3±7.59	270.18±17.47	0.073
RVTX	0.38±0.03	0.37±0.02	0.001*
IVS Sm, cm/s	10.5±1.34	11.22±1.35	0.009
IVS E′, cm/s	11.1±1.66	11.87±0.88	0.005*
IVS A′, cm/s	7.86±1.01	7.5±0.84	0.055
IVS E′/A′	1.46±0.37	1.6±0.2	0.017*
IVS IVCT, ms	52.4±2.9	51.7±4.56	0.362
IVS IVRT, ms	45±3.03	43.52±4.23	0.047*
IVS ET, ms	263.2±9.35	270.74±20.18	0.018*
IVSTX	0.37±0.03	0.35±0.02	0.001*

LV: left ventricle, RV: right ventricle, IVS: interventricular septum, E: inflow peak early diastolic velocity, A: inflow peak late diastolic velocity, Sm: peak systolic velocity, E′: annulus early diastolic myocardial peak velocity, A′: annulus late diastolic myocardial peak velocity, IVCT: isovolumetric contraction time, IVRT: isovolumetric relaxation time, ET: ejection time, TX: tei index (myocardial performance index). **P* value is statistically significant

Table 4: Spearmann correlation coefficient matrix between serum 25(OH) D levels and echocardiographic indicators of cardiac function in adolescent athletes (rho=*r*)

Parameter	*r*	Correlation magnitude	*P*
Structural (M-Mode)			
LVIDd	0.14	Very weak	0.173
LAd	0.15	Very weak	0.149
LVMI	0.13	Very weak	0.188
Systolic function			
EF	0.12	Very weak	0.256
LV Sm	0.58	Moderate	<.001*
RV Sm	0.65	Strong	<.001*
IVS Sm	0.47	Moderate	<.001*
Diastolic function			
LV E	0.38	Weak	<.001*
LV E/A	0.43	Moderate	<.001*
LV E′	0.52	Moderate	<.001*
LV E′/A′	0.46	Moderate	<.001*
LV E/E′	0.47	Moderate	<.001*
RV E	0.39	Weak	<.001*
RV E/A	0.43	Moderate	<.001*
RV E′	0.64	Strong	<.001*
RV E′/A′	0.57	Moderate	<.001*
RV E/E′	0.49	Moderate	<.001*
IVS E′	0.50	Moderate	<.001*
IVS E′/A′	0.43	Moderate	<.001*
Systolic and diastolic together			
LVTX	–0.52	Moderate	<.001*
RVTX	–0.53	Moderate	<.001*
IVSTX	–0.50	Moderate	<.001*

LV: left ventricle, RV: right ventricle, IVS: interventricular septum, E: inflow peak early diastolic velocity, A: inflow peak late diastolic velocity, Sm: peak systolic velocity, E′: annulus early diastolic myocardial peak velocity, A′: annulus late diastolic myocardial peak velocity, TX: tei index (myocardial performance index). **P* value is statistically significant

and nongenomic pathways.[19,20] Studies have shown that vitamin D treatment suppresses RAAS and helps regulate blood pressure.[21] Ameri *et al*.[22] reported a negative correlation between serum 25(OH) D levels and LV thickness and LVMI in a study conducted in a healthy population. In our cross-sectional study, we did not detect any relationship between serum 25(OH)D levels and blood pressure, cardiac muscle thickness, and LVMI. Such cardiac effects may be expected with prolonged exposure to vitamin D deficiency. Since our study was cross-sectional, we compared present measurements, but further long-time research is needed to decipher such relationships.

In our study, serum alkaline phosphatase levels were higher in the vitamin D deficient group compared to the other group; however, serum calcium levels were similar between the two groups. In addition, none of the participants had clinical and radiologic signs of rickets. In the early stages of vitamin D deficiency, an increase in serum alkaline phosphatase level is expected; however, the change in serum calcium level occurs in very severe deficiencies (especially in cases with rickets in infancy).[23]

In vitamin D deficiency, cardiac functions may be impaired through various mechanisms. Experimental animal studies have shown that vitamin D deficiency

impairs calcium ion transport in vascular smooth muscle cells and cardiac myocytes, causing damage and remodeling and leading to cardiomyocyte hypertrophy, interstitial tissue inflammation, and fibrosis.[24,25] Myocardial dysfunction may also occur due to the increased release of proinflammatory cytokines, suppression of anti-inflammatory cytokines, and myocardial fibrosis that may develop due to secondary hyperparathyroidism due to vitamin D deficiency.[26,27] EF measurement and tissue Doppler examination can be used to assess myocardial systolic function. In studies conducted with children who had rickets, it was reported that EF was low in the rickets group,[28] and EF improved after vitamin D treatment.[29] In studies conducted in patients with heart failure, it has been reported that serum 25(OH) D level is an independent marker in determining EF,[30] and an improvement in EF was observed with vitamin D treatment.[31,32] In their study of female basketball players, Radovanovic *et al*.[33] reported a positive correlation between serum 25(OH) D levels and LV EF (r = 0.59, P = .01) and LV Sm (r = 0.51, P = .003). For cardiac systolic dysfunction to cause a decrease in EF, significant heart failure is expected. Mild cardiac systolic dysfunction (subclinical) may not result in a decrease in EF. Therefore, in recent years, tissue Doppler imaging studies have been successfully used to detect subclinical cardiac systolic dysfunction in many systemic diseases and clinical conditions (even if EF is not affected). The Tei index or myocardial performance index is an excellent approach applied by tissue Doppler imaging that is not affected by changes in heart rate, blood pressure, preload and afterload. It evaluates systolic and diastolic cardiac functions together and is easy to perform. It has been successfully used to evaluate the degree of impairment in myocardial systolic and diastolic functions in many diseases.[9,34] In one study, it was reported that although there was no difference in EF measurement in adolescents with vitamin D deficiency, there was a decrease in LV Sm-RV Sm values and an increase in LVTX-RVTX values.[35] Another study reported that children with vitamin D deficiency and congenital heart disease showed an increase in EF measurement and a decrease in RVTX values with vitamin D supplementation.[36] In our study, although EF measurement was normal in adolescent athletes with vitamin D deficiency, LV-RV-IVS Sm values were low, and LV-RV-IVS Tei index values were high. Furthermore, serum 25(OH)D levels were strongly correlated with RV Sm and moderately correlated with LV Sm, IVS Sm, LVTX, RVTX, and IVSTX, but not with EF. Tissue Doppler may be able to detect the early stages of systolic dysfunction before the EF is affected.

Myocardial diastolic dysfunction may develop through RAAS overactivation, calcium accumulation in cardiac myocytes, and vascular smooth muscle cells, remodeling in myocardial cells, muscle hypertrophy, and many other mechanisms due to secondary hyperparathyroidism that may accompany vitamin D deficiency.[24,37] The best diastolic indicators are the pulsed Doppler E/A ratio, the tissue Doppler E′ value, and the E/E′ ratio. In two different studies conducted on adolescents and adults with vitamin D deficiency, a low E/A ratio, a low E′ value, and an increased E/E′ ratio were reported.[35,38] In their study of athletes, Radovanović *et al*. reported a correlation between serum 25(OH)D and mitral valve inflow deceleration time, which is another indicator of diastolic function (r = 0.51, P = .03).[33] In our study, we detected deteriorations in diastolic function indicators in adolescent athletes with vitamin D deficiency in accordance with the literature (LV and RV E/A ↓, LV-RV and IVS E′ ↓, and LV and RV E/E′ ↑). We also found a strong correlation between serum 25(OH) D levels and RV E′ and moderate correlations with LV E/A, LV E′, LV E/E′, RV E/A, RV E/E′, and IVS E′. The effect of vitamin D on cardiac function will be better understood with vitamin D supplementation and the long-term follow-up of these subjects.

Limitations of the study

Since we conducted our study in the fall and winter periods, lower serum 25(OH)D levels may have been obtained compared to other seasons of the year due to relatively low sun exposure. In our cross-sectional study, we found that low serum 25(OH)D levels were associated with subclinical impairment of cardiac function. However, studies on vitamin D deficiency have reported hypertension and overt echocardiographic abnormalities, but these may not have developed yet because our study was cross-sectional. Further studies with long-term follow-up of serum 25(OH)D levels and cardiac function in many adolescent athletes, including all seasons, are needed to clearly understand the effect of vitamin D on the heart of adolescent athletes.

Conclusions

In our study, we detected deteriorations in systolic (LV Sm ↓, RV Sm ↓, and IVS Sm ↓) and diastolic (LV and RV E/A ↓, LV-RV and IVS E′ ↓, and LV and RV E/E′ ↑) cardiac function indicators in adolescent athletes with serum 25(OH) D levels below 20 ng/mL. In addition, the LV, RV, and IVS Tei index, which can show systolic and diastolic functions together, was higher in the group with vitamin D levels below reference range. For this reason, to protect cardiac functions in adolescent athletes, we recommend screening serum 25(OH)D

levels in certain periods, such as fall and winter, and vitamin D supplementation if necessary.

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Author contributions

All authors have accepted responsibility for the entire content of this manuscript and approved its submission.

Ömer Güneş: Conceived and designed the evaluation and drafted the manuscript. Hakan Altın: Drafted the manuscript, collected and interpreted clinical data and reviewed the manuscript. Participated in the design of the evaluation and performed statistical analysis.

Ethical approval

The approval of the local Ethics Committee was obtained.

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Nil.

Conflict of interest

There are no conflicts of interest in connection with this paper, and the material described is not under publication or consideration for publication elsewhere.

Data availability

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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