ORIGINAL ARTICLE

Eastern Journal of Medicine

East J Med 26(2): 322-328, 2021
DOI: 10.5505/ejm.2021.28034

Athletes´ Heart: QTC Dispersion and Speckle Tracking Echocardiography

Remzi Sarikaya[1*], Dilek Giray[2]

[1]Van Education and Research Hospital, Health Science University, Department of Cardiology, Van
[2]Van Education and Research Hospital, Health Science University, Department of Pediatric Cardiology, Van

ABSTRACT

The purpose of this study is to evaluate structure and function of the left ventricle by echocardiographic methods and to investigate the relationship between QT and corrected QT dispersion that calculated in 12-lead-electrocardiography.

This study included 38 basketball players (17 male / 21 female) and age-sex matched 40 healthy untrained controls. The left ventricle systolic and diastolic functions were evaluated by conventional, Doppler and 2-dimensional speckle tracking echocardiography methods. Electrocardiography was performed to the subjects and QT and corrected QT dispersion were calculated.

Left ventricular internal diameter at end diastole and end systole, and also end diastolic and end systolic volumes were statistically higher in athletes in the M-mode echocardiographic examination ($p<0.05$). There were no significant differences between the global longitudinal strain values of the groups ($p>0.05$). The intervals of QT and corrected QT were statistically lower in athletes whereas, dispersions of QT and corrected QT intervals were statistically higher in the athletes ($p<0.05$). There was a significant positive correlation between the global longitudinal strain values and the corrected QT interval ($p=0.043$; $r=0.494$), and negative correlation between dispersions of QT and corrected QT ($p=0.001$; $r=-0.804$ and $p=0.001$; $r=-0.764$ respectively) in athletes.

Calculation of QT and corrected QT dispersion, that is a feasible and simple method, will provide additional information to aid the differential diagnosis of physiological and pathological adaptation especially when there is a significant decrease in strain values of left ventricle.

Keywords: Athletes´ heart, corrected QT dispersion, QT dispersion, speckle tracking echocardiography

Introduction

Continuous and regular exercise causes changes in the structure and function of the heart. This condition, accompanied by a decrease in heart rate, hypertrophy in the heart muscle, and ventricular enlargement, is called athletes' heart (1). These adaptive mechanisms in the ventricles are reversible and help to provide cardiac output while systolic and diastolic functions are preserved and more effective oxygen extraction to exercising muscles (2). Changes in heart size and hemodynamics previously have been studied with two-dimensional, Doppler and 2D speckle-tracking echocardiography (3,4). Significant changes have been reported in athletes exercising intensely in the left ventricular radial and basal circumferential strain compared to healthy individuals with a sedentary lifestyle (3,5).

The effect of ventricular hypertrophy on QT dispersion was also investigated in athletes. QT dispersion is obtained by calculating the difference between the longest and shortest QT intervals on 12-lead-electrocardiography and provides assessment of the regional repolarization differences in the ventricular muscle. Increased QT dispersion is useful in predicting life-threatening arrhythmia and sudden death risk (6). However, echocardiographic measurements and calculated electrocardiographic parameters are not evaluated together in the literature.

The aim of this study is evaluation of the left ventricular structure and function in adolescent age group elite athletes using conventional, Doppler, and 2D Speckle tracking echocardiography methods and to investigate their relationship with QT and corrected QT dispersion calculated in 12-lead-electrocardiography.

Materials and Method

A group of 38 basketball players admitted to the cardiology and pediatric cardiology unit between January 2019 and December 2019 for scanning before the health report was included in this study. The study group was chosen among athletes who exercised at least 6 hours/week on average for two years. Age and gender-matched 47 untrained adolescents were selected as the control group admitted to the outpatient clinic in the same period with the diagnosis

***Corresponding Author:** Remzi Sarikaya, Van Education and Research Hospital, Turkish Republic Ministry of Health, Department of Cardiology, Süphan Mahallesi, Havayolu Kavşağı, Edremit, Van 65300, Turkey
E mail: drremzisarikaya@gmail.com, Tel: +90 (546) 472 17 35, Fax: +90 (432) 212 19 54
ORCID ID: Remzi Sarikaya: https://orcid.org/0000-0003-4774-3103, **Dilek Giray:** https://orcid.org/0000-0001-7660-4052
Received: 12.12.2020, **Accepted:** 22.02.2021

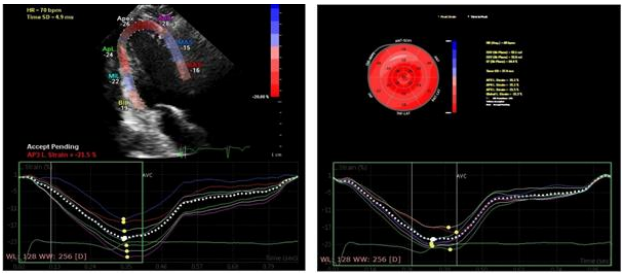

Fig. 1. Myocardial strain assessment of left ventricle by using 2D speckle-tracking imaging. The longitudinal global strain was calculated by averaged peak systolic value of six segments

of innocent murmur and normal echocardiographic examination. Control group was chosen among healthy adolescents with a sedentary lifestyle.Exclusion criteria for the patient and the control groups were cardiac, chronic infectious or inflammatory diseases, anemia, or other hematological problems. Approval of the Local Ethics Committee and form of informed consent have been acquired for the study protocol.

Echocardiographic Evaluation: The transthoracic two-dimensional echocardiography was done by using a GE Vivid E9 ultrasound machine. In the parasternal long-axis view, end-systolic and end-diastolic diameters of the left ventricle, the diastolic posterior wall thickness of the left ventricle and thickness of the interventricular septum were measured by 3 and 6 MHz transducers in 2D and M-mode evaluation. In the apical four-chamber view, the ejection fraction of the left ventricle was measured by modified Simpson's method (7). Left ventricular diastolic function was assessed by tissue Doppler echocardiography method, early diastolic filling velocity (mitral inflow E) and late diastolic velocity of atrial contraction (A-wave velocities) (E/A), and E/A ratio was calculated (8).

Speckle-Tracking Imaging and Strain: End expiratory cardiac cycles in the apical four-chamber view in standard echocardiographic imaging were obtained. Data from three serial cardiac cycles were averaged, and statistical analysis was done using Echo PAC software (version 8.0, GE healthcare PC 2008) as described previously (9). The tracking points were adjusted from an end-systolic frame manually. A self-acting tracking algorithm outlined the entire myocardial wall, and the analysis of myocardial wall motion was performed by speckle tracking. The left ventricle's longitudinal, the global strain was calculated by the average of the six segments' peak systolic values in the four-chamber view. The strain curves of poor-quality imaged segments were excluded from analyses (Fig. 1).

Electrocardiography: 12-lead electrocardiography with 25 mm/sec at standard leads was taken at rest with Nihon Kohden cardiofax GEM device for both groups. The QT interval was calculated from the beginning of the Q wave to the T wave end. If there was a U wave, the T wave's endpoint was defined using the tangent method on the descending branch of the T wave and determining the intersection with the baseline. Bazett's formula ($QTc = QT / \sqrt{RR\ (s)}$) was used to correct each QT interval for the patient's heart rate simultaneously in all leads (10). Three QT intervals were measured for every derivation, and they averaged. QT dispersion values were obtained by subtracting the shortest QT interval from the most prolonged QT interval of any 12 electrocardiographic leads. The corrected QT dispersion values were also obtained by subtracting the shortest corrected QT interval from the longest corrected QT interval of any 12 electrocardiographic leads.

Statistical Analysis: Data were analyzed using SPSS software version 20.0 for Windows (SPSS Inc, Chicago, Illinois). Descriptive statistics were expressed as means and their standard deviations. The normal distribution of numerical data was checked with the Shapiro Wilk test. Then, Mann Whitney-U test was used for variables that did not show the normal distribution, and an independent t-test was used for other parameters. Spearman's correlation analysis determined the relationships between the parameters. Statistical significance was considered as $p<0.05$.

Results

This study included 38 athletes (17 F, 21 M) and 47 healthy untrained adolescents (24 F, 23 M). The mean age of the study group was 14.6±1.2 years and 14.8±0.914 years in controls. The athletes were chosen among the adolescents who have exercised at least 6 hours/week on average for two years. Adolescents in the study group had a regular exercise history of 2.5 ± 0.75 years and were trained at least ten months per year. Distributions of age and gender were similar in between the groups ($p> 0.05$). Also, there was no significant difference in the mean systolic and diastolic blood pressures of the groups ($p> 0.05$). The average resting heart rate was significantly lower in athletes than that of controls ($p =0.006$). Table 1 shows the demographic data of the groups.

End-diastolic and end-systolic diameters of the left ventricle and the end-diastolic and end-systolic volumes of the left ventricle were statistically higher in the M-mode athlete's echocardiographic examination ($p<0.05$).

Doppler echocardiography values were similar in between the groups except for early atrial filling wave

Table 1. Demographic data of the groups

	Controls (n=47)	Athletes (n=38)	p
Age (year)	14.6±1.2	14.8±0.9	0.574
Sex			
Female (%)	24 (51.1%)	17 (44.7%)	0.351
Male (%)	23(48.9%)	21(55.3%)	0.346
Height (cm)	155±6.5	156±4.6	0.245
Weight (kg)	50.3±2.1	51.4±1.9	0.167
Systolic blood pressure (mmHg)	105.4±10	109.2±9	0.095
Diastolic blood pressure (mmHg)	64.3±9	68.6±8	0.051
Resting heart rate (beats/min)	85±8	64±10	0.006

Table 2. Conventional echocardiographic values of the groups

	Controls (n=47)	Athletes (n=38)	p
IVSd (mm)	8.1±1.7	8.3±1.2	0.265
LVIDd (mm)	39.7±5.9	45.1±3	0.001
LVIDs (mm)	25±4.3	28.4±2.4	0.003
LVPWd (mm)	8±1.6	8.1±1.2	0.060
EDV (mm3)	69.2±25	94.2±15	0.009
ESV (mm3)	26.5±10	31.6±6.8	0.005
EF (%)	64.6±5.4	65.6±4.2	0.193
Mitral E (m/s)	1.03±0.16	0.90±0.12	0.013
Mitral A (m/s)	0.54±0.9	0.52±0.9	0.550
Mitral E/A	1.9±4	1.8±4	0.052
TDIE (cm/s)	12.5±3.2	12.7±1.8	0.300
TDIA (cm/s)	5.9±1.3	6.2±1.8	0.379
TDIE/A	2.14±0.5	2.1±0.5	0.714
Strain (%)	21.2±1.2	20.8±2.2	0.257

IVSd; Interventricular septal thickness at end diastole; LVIDd: Left ventricular internal diameter at end diastolic; LVIDs: Left ventricular internal diameter at end systolic; LVPWd: Left ventricular posterior wall thickness at end diastole; EDV: End diastolic volume; ESV: End systolic volume; EF: Left ventricular ejection fraction; E wave: Early atrial filling wave velocity; A wave: Atrial contractility wave velocity; TDIE: Tissue Doppler imaging septal E'; TDIA: Tissue Doppler imaging septal A'.

velocities. The global strain values of the athletes were lower than that of controls, but this difference was not significant (p>0.05) (Table2). There was no significant difference in the left ventricular global strain values of male and female athletes.

In the electrocardiographic evaluation, rhythm abnormality was not detected in eighter athletes nor controls. The intervals of QT and corrected QT were statistically lower in athletes whereas, dispersions of QT and corrected QT intervals were statistically higher in the athletes (p<0.05) (Table 3).

Spearman's correlation analysis was performed between Doppler echocardiographic measurements and electrocardiographic parameters, and no correlation was detected (Table 4).

Spearman's correlation analysis demonstrated a significant positive correlation between the strain values and the corrected QT interval (p=0.043; r=0.494), while a significant negative correlation between the strain values and the dispersions of QT and corrected QT intervals (p=0.001; r=- 0.804 and p=0.001; r=- 0.764 respectively) in athletes. There was no such correlation in healthy controls (Table 4).

Discussion

Cardiac adaptation called 'athlete's heart' is hypertrophy and enlargement of the myocardium as a result of repeated exercise stimulation. Cardiac output increases in repetitive heavy exercises with some adaptive mechanisms such as increased left ventricular internal diameter and wall thickness,

Table 3. Electrocardiography parameters of athlete and control groups

	Controls (n=47)	Athletes (n=38)	p
QT (ms)	394±28.2	322±33.7	0.005
QTC (ms)	404±11	375±25	0.001
QTd (ms)	42.6±7.1	58.7±10.6	0.001
QTCd (ms)	43.8±7.6	62.5±13	0.001
PR (ms)	130±15	128±13	0.671

QT: Interval of QT; QTd: Dispersion of QT interval; QTC: Corrected QT interval; QTCd: Dispersion of corrected QT interval; PR: ınterval of PR interval

Table 4. Correlations between the Doppler echocardiography and strain values and other electrocardiographic parameters of athlete and control groups

	Mitral E p (r)	Mitral A p (r)	E/A p (r)	TDIE p (r)	TDIA p (r)	TDIE/A p (r)	Strain p (r)
Controls							
QT	0.587 (0.081)	0.120 (0.230)	0.369 (0.134)	0.320 (0.148)	0.728 (0.052)	0.172 (0.203)	0.694 (0.059)
QTC	0.249 (0.172)	0.583 (0.082)	0.485 (0.104)	0.619 (0.074)	0.053 (0.284)	0.217 (0.183)	0.373 (0.133)
QTd	0.055 (0.327)	0.414 (0.122)	0.655 (0.067)	0.383 (0.130)	0.614 (0.075)	0.581 (0.81)	0.058 (0.303)
QTCd	0.585 (0.082)	0.784 (0.041)	0.417 (0.121)	0.962 (0.621)	0.812 (0.063)	0.568 (0.086)	0.289 (0.158)
Athletes							
QT	0.193 (0.216)	0.252 (0.191)	0.854 (0.031)	0.245 (0.193)	0.181 (0.222)	0.376 (0.148)	0.159 (0.233)
QTC	0.155 (0.235)	0.962 (0.008)	0.245 (0.193)	0.255 (0.189)	0.779 (0.054)	0.660 (0.074)	0.043 (0.494)
QTd	0.275 (0.182)	0.893 (0.022)	0.655 (0.075)	0.403 (0.140)	0.039 (0.304)	0.797 (0.220)	0.001 (-0.804)
QTCd	0.627 (0.081)	0.328 (0.163)	0.279 (0.180)	0.590 (0.090)	0.019 (0.378)	0.241 (0.195)	0.001 (-0.764)

QT: Interval of QT; QTd: Dispersion of QT interval; QTC: Corrected QT interval; QTCd: Dispersion of corrected QT interval; TDIE: Tissue Doppler imaging septal E'; TDIA: Tissue Doppler imaging septal A'.

increased blood volume, cardiac pressure, and heart rate (11). The cardiovascular benefit of regular exercise has been demonstrated, and most sports activities are assumed to be good for health (12). Despite the fact, the effects of competitive sports and heavy exercise program and duration of action is uncertain. The importance of athletes' hearts is the risk of sudden cardiac death associated with exercise. Therefore, the adaptive mechanisms should be known to help screen before sports events, and precautions should be taken at high-risk athletes. The previous studies in the literature included data in either echocardiographic imaging or electrocardiographic assessment concerning young elite athletes (13-14). We evaluated left ventricular dimensions in the present study by conventional echocardiographic imaging and functions by Doppler and speckle-tracking imaging and correlated them with the electrocardiographic measures.

The study revealed a significant increase in end-diastolic and end-systolic internal diameters of the left ventricle with the M-mode echocardiography, showing an increase in left ventricle cavity dimensions as a result of a repeated exercise in young elite athletes as previously described (13-15). Moreover, we showed an increase in end-diastolic and end-systolic volumes in athletes when compared to controls. The sports are subdivided into two major groups according to the development of the heart's adaptive changes. The first group is aerobic, or endurance sports described by a large muscle group's involvement with dynamic exercising and presented 'eccentric left ventricle hypertrophy'. The second group is resistance or strength-trained sports, which

are experienced by repetitive increases in peripheral vascular resistance and cardiac afterload and presented 'concentric left ventricle hypertrophy' (16). However, some sports involve heterogeneity in endurance and resistance-trained cardiovascular demands, as in basketball. This study's results, which showed an increase in both size and volume in athletes' heart, are thought to be the consequence of the nature of the sport that causes both eccentric and concentric hypertrophy.

Left ventricular fractional shortening and ejection fraction have been used to define the left ventricle systolic function using conventional echocardiography. Previous studies have a controversy on these indices of systolic function. It is reported that fractional shortening or ejection fraction of the left ventricle was not different between athletes and matched controls in some studies, while significantly higher or lower in athletes in some others. But these values were still within normal limits, suggesting that there is a normal left ventricular systolic function in athletes (11,14,17). This study did not find the difference in ejection fraction of left ventricle between the groups. We supposed that ejection fraction is not enough to show the adaptation of left ventricular systolic function in elite athletes.

In this study, we found early diastolic filling (mitral E) wave of the athletes was lower than the controls. This result can be attributed to the lower heart rate prolonging the diastolic filling period and to the resting hyperdynamic state of the athlete's heart.

However, we did not find any difference in other Doppler velocities, demonstrating improved diastolic left ventricle function.

Left ventricular global longitudinal strain is accepted a more sensitive measure of systolic function than left ventricular ejection fraction in early determination of subclinical LV dysfunction (18). However, there is heterogeneity in the results of the studies in the literature. Some studies demonstrated higher left ventricular global longitudinal strain in athletes compared to controls (19), while others did not show any difference (20,21) or others showed lower values in the athletes (13,22). Moreover, in a study, Giraldeau et al. reported that female athletes have also been found to have higher left ventricular global longitudinal strain than male athletes (23). This controversy may result from variation in left ventricle structure secondary to the training type and intensity. This study found no significant difference in global longitudinal strain values neither in between athletes and controls, nor between in genders.

Athlete's heart may be related with modifications in rhythm and conduction, morphological alterations of the QRS complex, and repolarization variances. Electrocardiographic changes reported in athletes are sinus bradycardia, sinus arrhythmia, sinus pause, PR, QRS and QT interval prolongation, right bundle branch block and ST elevation (24). Some of the previous studies showed no significant difference in intervals of QT, corrected QT, dispersions of QT, and corrected QT between athlete and control groups (6,25,26). On the other hand, some studies reported that dispersion of QT and corrected QT were increased in athletes and that the increase was significantly related to the duration of athletic activity. Moreover, it has been suggested that QT dispersions can be used as a non-invasive method in the evaluation of pathological left ventricular hypertrophy and severe ventricular arrhythmia risk in athletes (27-29). In this study, we found that the dispersion of QT and corrected QT values of the athletes were significantly higher than in controls. The dispersion of QT states segmental differences in repolarization of ventricles. The longer the recovery time, the greater the risk of arrhythmia and sudden death (25). In this study, we revealed that athletes might be prone to ventricular arrhythmias more than healthy untrained populations due to their physiological and structural changes and heterogeneous repolarization in ventricular myocardium. Furthermore, we correlated QT intervals, corrected QT, and dispersions of QT and corrected QT with the Doppler echocardiographic imaging velocities. We did not find a correlation in either group.

In this study, there was a significant positive correlation between corrected QT and negative correlation between QT and corrected QT dispersions and left ventricular longitudinal strain values studied for the first time in athletes. It is known that the reduction in longitudinal strain is an early sign of left ventricular dysfunction. We did not find a significant difference in strain values between the athletes and controls, so we assumed a significant reduction in left ventricular longitudinal strain values in athletes is an uncommon feature. However, when there is an increase in corrected QT interval, and/or a decrease in the dispersion of QT and corrected QT values, a significant decrease in strain values cannot be considered as a physiological adaptation and accurate evaluation of the myocardial function, helping us distinguish compliance from illness.

Limitations: The major limitation of our study was the relatively low number of cases in each group. In addition, our results could not be interpreted to all elite athletes because our study was limited to a heterogeneous group of athletes and a small number of subjects. Besides, the subjects enrolled were not followed for a long time; they all were evaluated once

before their competitive participation. Thus, the transient changes in left ventricular dynamics, such as during a training season or dynamics of unconditioning remain unknown.

Heavy exercise does not increase the mortality rate in athletes, but can trigger the development of malignant arrhythmias, especially in those with structural heart disease and conduction disorders. Because there is no clear pathognomonic sign available in evaluating young elite athletes, examining the patients with clinical signs and symptoms, family history, electrocardiographic evaluation and echocardiographic imaging is recommended. Calculation of QT and corrected QT dispersions accompanied by reduced strain values of the left ventricle can be used to define athletes at high risk of sudden death.

References

1. Ribeiro I, Botanico J, Pelliccia A, Maron BJ. Physiologic left ventricular cavity dilatation in elite athletes (3) (multiple letters). Ann Intern Med 1999; 131: 546.
2. Morganroth J, Maron BJ, Henry WL, Epstein SE. Comparative left ventricular dimensions in trained athletes. Ann Intern Med 1975; 82: 521-524.
3. Beaumont A, Grace F, Richards J, Hough J, Oxborough D, Sculthorpe N. Left Ventricular Speckle Tracking-Derived Cardiac Strain and Cardiac Twist Mechanics in Athletes: A Systematic Review and Meta-Analysis of Controlled Studies. Sport Med 2017; 47: 1145-1170.
4. Forsythe L, George K, Oxborough D. Speckle Tracking Echocardiography for the Assessment of the Athlete 's Heart : Is It Ready for Daily Practice ? Curr Treat Options Cardiovasc Med 2018; 20: 83.
5. Vitarelli A, Capotosto L, Placanica G, et al. Comprehensive assessment of biventricular function and aortic stiffness in athletes with different forms of training by three-dimensional echocardiography and strain imaging. Eur Heart J Cardiovasc Imaging 2013; 14: 1010-1020.
6. Akalin F, Topçu B, Odabaş I, Çotuk B, Mengütay S. QT dispersion in young athletes (Sporcu çocuklarda QT dispersiyonu). Turk Pediatr Ars 2007; 42: 117-120.
7. Cain PA, Ahl R, Hedstrom E, et al. Age and gender specific normal values of left ventricular mass, volume and function for gradient echo magnetic resonance imaging: A cross sectional study. BMC Med Imaging 2009; 9: 2.
8. Laaksonen MS, Kalliokoski KK, Luotolahti M, et al. Myocardial perfusion during exercise in endurance-trained and untrained humans. Am J Physiol - Regul Integr Comp Physiol. 2007; 293: 837-843.
9. Leitman M, Lysyansky P, Sidenko S, et al. Two-dimensional strain-A novel software for real-time quantitative echocardiographic assessment of myocardial function. J Am Soc Echocardiogr 2004; 17: 1021-1029.
10. Garson A. How to measure the QT interval-What is normal? Am J Cardiol 1993; 72: 6.
11. Pluim BM, Zwinderman AH, Laarse A Van Der, Wall EE Van Der. The Athlete ' s Heart A Meta-Analysis of Cardiac Structure and Function. Circulation 2000; 101: 336-344.
12. Blair SN, Iii HWK, Paffenbarger RS, Clark DG, Cooper KH, Gibbons LW. Physical Fitness and All-Cause Mortality. JAMA 1989; 262: 2395-2401.
13. Luca A De, Stefani L, Pedrizzetti G, Pedri S, Galanti G. The effect of exercise training on left ventricular function in young elite athletes. Cardiovascular Ultrasound 2011; 1-9.
14. Charfeddine S, Mallek S, Triki F, Hammami R, Abid D, Abid L, Kammoun S. Echocardiographic analysis of the left ventricular function in young athletes: a focus on speckle tracking imaging. The Pan African Medical Journal 2016; 25: 171.
15. Sharma S, Maron BJ, Whyte G, Firoozi S, Elliott PM, McKenna WJ. Physiologic limits of left ventricular hypertrophy in elite junior athletes: Relevance to differential diagnosis of athlete's heart and hypertrophic cardiomyopathy. J Am Coll Cardiol 2002; 40: 1431-1436.
16. Naylor LH, George K, Driscoll GO, Green DJ. The Athlete ' s Heart A Contemporary Appraisal of the ' Morganroth Hypothesis'. Sports Med 2008; 38: 69-90.
17. Fagard R. Athlete'S Heart. Heart 2003; 89: 1455-1461.
18. Smiseth OA, Torp H, Opdahl A, Haugaa KH, Urheim S. Myocardial strain imaging: How useful is it in clinical decision making? Eur Heart J 2016; 37: 1196-1207.
19. Simsek Z, Hakan Tas M, Degirmenci H, et al. Speckle tracking echocardiographic analysis of left ventricular systolic and diastolic functions of young elite athletes with eccentric and concentric type of cardiac remodeling. Echocardiography 2013; 30: 1202-1208.
20. Galanti G, Toncelli L, Tosi B, et al. Evaluation of left ventricular remodelling in young Afro-Caribbean athletes. Cardiovascular Ultrasound 2019; 1-8.
21. Bjerring AW, Landgraff HEW, Leirstein S, et al. Morphological changes and myocardial function assessed by traditional and novel echocardiographic methods in preadolescent athlete's heart. European Journal of Preventive Cardiology 2018; 25: 1000-1007.
22. Richand V, Lafitte S, Reant P, et al. An Ultrasound Speckle Tracking (Two-Dimensional Strain) Analysis of Myocardial Deformation in Professional Soccer Players Compared With Healthy Subjects and Hypertrophic

Cardiomyopathy. Am J Cardiol 2007; 100: 128-132.
23. Giraldeau G, Kobayashi Y, Finocchiaro G, et al. Gender Differences in Ventricular Remodeling and Function in College Athletes , Insights from Lean Body Mass Scaling and Deformation Imaging. Am J Cardiol 2015; 116: 1610-1616.
24. Sharma S, Whyte G, Elliott P, et al. Electrocardiographic changes in 1000 highly trained junior elite athletes. Br J Sports Med 1999; 33: 319-324.
25. Turkmen M, Barutcu I, Esen AM, et al. Assessment of QT interval duration and dispersion in athlete's heart. J Int Med Res 2004; 32: 626-632.
26. Halle M, Huonker M, Hohnloser SH, Alivertis M, Berg A, Keul J. QT dispersion in exercise-induced myocardial hypertrophy. Am Heart J 1999; 138: 309-312.
27. Yildirim S, Binnetoğlu FK, Battal F, Aylanç H, Kaymaz N, et al. Relation between QT Variables and Left Ventricular Geometry in Athletes and Obese Children. Acta Med Port 2016; 29: 95-100.
28. Lawan A, Ali MA, Bauchi SSD. QT Dispersion In Dynamic And Static Group Of Athletes. Nigerian Journal Of Physiological Sciences 2006; 21: 5-8.
29. Dimitriu L, Mihai F, Dimitriu AG. The study of QT and QTc intervals dispersion in sporting teenagers. Archives of Cardiovascular Diseases Supplements 2020; 12: 126.