Turk Kardiyol Dern Ars 2017;45(2):160-166 doi: 10.5543/tkda.2016.73557

ORIGINAL ARTICLE

Using cardiovascular imaging modalities to determine cardiac disorders before starting sports activities

Spor aktivitelerine başlamadan önce kardiyovasküler görüntüleme yöntemleri ile kardiyak bozuklukların tanınması

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ABSTRACT

Objective: We re-examined children who had previously been declared eligible to participate in competitive sports activities for cardiac disorders, using cardiac investigation protocol.

Methods: Total of 250 children (224 males [89.6%], and 26 females [10.4%]) between the ages of 8 and 17 years who had just started or were already engaged in sports activities were included in the study. Participants had detailed physical examination evaluated by a pediatric cardiologist. Those with findings suggesting cardiac disorder in their history and/or physical examinations, and/or 12-channel electrocardiography (ECG) were examined with echocardiography (ECHO), 24-hour Holter monitoring, and exercise test.

Results: Mean duration of participation in sports activities was 13 months. Among all, 10.4% of the children had abnormalities on ECG. ECHO demonstrated cardiomyopathy in 1, mitral valve prolapse in 2, tricuspid insufficiency in 2, and mitral insufficiency in 1 participant. Holter monitoring revealed non-sustained ventricular tachycardia attacks in 1, and supraventricular tachycardia in another child. Three were ultimately disqualified from partaking in competitive sports.

Conclusion: Sports and medical communities must work together to establish study protocols to prevent sudden death related to sports and to make these activities safer for athletes. Pediatric cardiology consultation for young athletes before they start sports activities is needed.

ÖZET

Amaç: Daha önce kardiyak sorunlar için spor yarışmalarına katılmaya uygun oldukları bildirilen çocukları, kalp araştırması protokolü kullanarak yeniden inceledik.

Yöntemler: Yaşları 8–17 arasında olan, sportif etkinliklere daha önce veya yeni başlamış 250 çocuk (224 erkek [%89.6] ve 26 kız [%10.4]) çalışmaya dahil edildi. Katılımcılar detaylı bir fizik muayeneden geçirildi ve bir çocuk kardiyoloğu tarafından değerlendirildi. Öykü, fizik muayene veya 12 derivasyonlu elektrokardiyografi'nin (EKG) herhangi birinde kalp hastalığını düşündürecebilecek bulgusu olan hastalar ekokardiyografi, 24 saatlik Holter kaydı ve egzersiz testi ile araştırıldı.

Bulgular: Spor faaliyetlerine ortalama katılım süresi 13 ay idi. Tüm katılımcıların %10.4'ünde EKG patolojisi mevcuttu. Ekokardiografi katılımcıların birinde kardiyomiyopati, ikisinde mitral valv prolapsusu ve birinde mitral yetersizliği ortaya koydu. Holter moniterizasyonu katılımcıların bir tanesinde süreksiz ventriküler taşikardi ve birinde supraventriküler taşikardiyi açığa çıkardı. Üç katılımcı profesyonel spor aktivitelerinden uzaklaştırıldı.

Sonuç: Spor ve tıp komiteleri atletler için spor etkinliklerini daha güvenli yapmak, spora bağlı ani ölümleri engellemek için birlikte çalışarak çalışma programları oluşturmalıdırlar. Genç sporcuların spor faaliyetlerine başlamadan önce çocuk kardiyolojisi konsültasyonu yapılması gerekir.

Sudden cardiac death (SCD) in athletes usually occurs during training or competition when the athletes exert excessive effort. SCD in athletes has been defined as "deaths occurring 30 seconds – 6 hours after exercise" by the World Health Organization.[1]

Annual rate of athlete deaths due to SCD was found to be 1/217 000 in the United States.[2] Rate of sudden death due to cardiac causes was reported as 1–3/100 000 in all athletes.[3] Another study reported incidence of SCD as 2.1/100,000 in athletes younger

Received: October 03, 2016 *Accepted:* December 28, 2016
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than 35 years of age.[4] In the Lausanne Recommendations, it was reported that more than 90% of sudden deaths in athletes were due to a preexisting cardiac disease.[5] Various cardiovascular investigation strategies have been defined to prevent cardiac-related deaths. In 2004, the Lausanne Recommendations proposed preparticipation screening protocol for athletes in order to decrease incidence of SCD.[6]

In this study, young athletes were re-examined for cardiac disorders after they had been declared to be eligible to participate in sports using cardiac investigation protocol as outlined in the Lausanne Recommendations. Present study also includes a review of cardiovascular investigation methods.

METHODS

Total of 250 children between ages of 8 and 17 years who were beginning or already engaged in sports activities were included in the present study. Detailed personal history of all participants was obtained, including family history, as outlined in the Lausanne Recommendations. Detailed physical examination was performed and evaluated by a pediatric cardiologist, as well as 12-channel electrocardiography (ECG) results. Those with acute or chronic disorder or with history of medication use or smoking were excluded. Those with findings suggesting cardiac disorder in their history, and/or physical examination, and/or 12-channel ECG were examined with echocardiography (ECHO), 24-hour Holter monitoring, and exercise test. This study meets the ethical standards of the journal.

In case of positive family history of myocardial infarction at young age in close relatives, or family history of cardiomyopathy (CMP), Marphan syndrome, long QT syndrome (LQTS), Brugada syndrome, severe arrhythmia, coronary artery disease, or other cardiovascular disease, family history was regarded as positive. Personal history was considered positive in presence of chest pain suggesting angina, syncope, or syncope-like symptoms, arrhythmia, palpitations, dyspnea, or tiring quickly.

Positive physical examination findings included musculoskeletal and ocular findings supporting Marphan syndrome, weakening or delayed femoral artery pulse, mid-systolic or late systolic clicks, second heart sound alone or becoming stable in expiration, diastolic or systolic murmurs ≥degree 2/6, irregular heart rhythm, or blood pressure >95th percentile. Study participants with those findings had secondary investigation.

Abbreviations:

ARVD	*Arrhythmogenic right ventricular dysplasia*
CMP	*Cardiomyopathy*
ECG	*Electrocardiography*
ECHO	*Echocardiography*
HCMP	*Hypertrophic cardiomyopathy*
LQTS	*Long QT syndrome*
MVP	*Mitral valve prolapse*
RBBB	*Right bundle branch block*
SCD	*Sudden cardiac death*
SVT	*Supraventricular tachycardia*

Electrocardiography

Twelve-channel ECG was regarded as positive with presence of 1 or more findings defined by the European Society of Cardiology (Table 1).[4] Non-invasive tests including ECHO, 24- hour Holter, and exercise test, were conducted in event of positive findings in baseline examinations. Catheterization and angiography were invasive tests to be implemented when diagnosis could not be made using non-invasive methods.

Statistical analysis

Data were evaluated using SPSS software version 16.0 (IBM Corp., Armonk, NY, USA). As first step, normal distribution of sample was analyzed with Shapiro-Wilk test. Normal distribution was represented using mean and standard deviation, whereas skewed distribution was expressed as median and minimum-maximum. Unpaired t-test or Mann-Whitney U test was used for comparison of 2 groups. Categorical variables in proportions or percentages were analyzed with chi-square test or Fisher's exact test, as appropriate. Associations between variables were assessed with Pearson's correlation analysis or Spearman's rank correlation analysis. P value <0.05 was considered to be statistically significant.

RESULTS

Total of 250 licensed, competitive athletes of median age 13 years (range: 8–25 years) who had medical reports indicating "may perform sports activities, healthy" were included in the study. There were 224 males (89.6%), and 26 females (10.4%). Mean duration of participation in sports was 13 months. There were 205 (82%) football players, 25 (10%) basketball players, 17 (6.8%) volleyball players, and 3 (1.2%) swimmers.

Data about the family history of the athletes are presented in Table 2 and were collected as outlined in the Lausanne Recommendations. Data obtained from

Table 1. Positive findings on 12-channel electrocardiogram[4]

P wave	Left atrial enlargement: Negative portion of the P wave in lead V1 ≥0.1 mV in depth, and ≥0.04 s in duration
	Right atrial enlargement: peaked P wave in leads II and III or V1 ≥0.25 mV in amplitude
QRS complex	Frontal plane axis deviation: right ≥+120° or left –30° to –90°
	Increased voltage: amplitude of R or S wave in in a standard lead ≥2 mV, S wave in lead V1 or V2 ≥3 mV, or R wave in lead V5 or V6 ≥3 mV abnormal Q waves ≥0.04 s in duration or ≥ 25% of the height of the ensuing R wave or QS pattern in two or more leads
	Right or left bundle branch block with QRS duration ≥0.12 s
	R or R′ wave in lead V1≥ 0.5 mV in amplitude and R/S ratio ≥1
ST-segment, T-waves and QT interval	ST-segment depression or T-wave flattening or inversion in two or more leads
	Prolongation of heart rate corrected QT interval >0.44 s in males and >0.46 s in females
	Premature ventricular beats or more severe ventricular arrhythmias
Rhythm and conduction abnormalities	Supraventricular tachycardias, atrial flutter, or atrial fibrillation short PR interval (<0.12 s) with or without without 'delta' wave
	Sinus bradycardia with resting heart rate ≤40 beats/min
	First (PR ≥0.21 s), second or third degree atrioventricular block

physical examination, ECG, and ECHO are summarized in Table 3.

Among all the children (n=250), 10.4% had abnormalities on ECG (n=26). There were ventricular extrasystoles in 7, pathological axis in 6, and right bundle branch block (RBBB) in 13 athletes.

ECHO was performed on 73 study participants and results demonstrated CMP in 1, mitral valve prolapse (MVP) in 2, tricuspid insufficiency in 2, and moderate mitral insufficiency in 1. Holter monitoring was conducted for 9 patients and revealed non-sustained ventricular tachycardia attacks in 1 young athlete and short supraventricular tachycardia attacks in another.

One participant who had ventricular extrasystoles on ECG had ECHO findings that suggested aortopulmonary window. Catheter angiography was scheduled as third line examination. Patient was diagnosed with ruptured sinus of Valsalva, and subsequently underwent surgery. This athlete as well as 1 diagnosed with CMP and another with MVP and short supraventricular tachycardia attacks on Holter monitoring were prohibited from further participation in competitive sports; all 3 were male. Correlation of symptoms with ECG and ECHO findings is presented in Table 4.

Four of 17 study participants with dyspnea had abnormalities on ECG, and 1 of 11 in that group had abnormal ECHO findings. Comparison of those with and without dyspnea with ECG and ECHO findings did not reveal any statistically significant results (p>0.05 for each).

Table 2. Clinical characteristics of athletes (n=250)

Parameter	
Age (years)	13 (8–25)
Sex (Male/Female)	224/26
Finding, n (%)	
Dsypnea	17 (6.8)
Chest pain	22 (8.8)
Getting tired quickly	4 (1.6)
Palpitations	16 (6.4)
Syncope	3 (1.2)

Family history of heart disease before 45 years of age 25.[1]

Table 3. Physical examination, electrocardiography, and echocardiography findings of the subjects (n=250)

Finding	n	%
Murmur	23	9.2
Dysrhythmia	7	2.8
Electrocardiography abnormalities	26	10.4
Echocardiography abnormalities (n=73)	7	9.5
Holter abnormalities (n=9)	2	22.2

Only 1 (4.5%) of 22 children with chest pain had ECG abnormalities, and 1 of 19 (5.2%) who had ECHO performed had abnormal findings. Participants with and without chest pain had similar ECG and ECHO results.

Among those with palpitations (n=16), 6 (37.5%) had positive ECG findings, and 1 (11.1%) of 9 athletes had pathological ECHO findings. There was statistically significant difference between patients with and without palpitations in ECG and ECHO findings (p=0.02, p=0.021, respectively). However, there was no significant correlation between palpitations and ECHO findings (p>0.05).

Study participants who had symptom of tiring quickly had normal ECGs. Two who had ECHO did not have any abnormal findings.

Incomplete RBBB was determined on ECG in 1 one of the athletes who had history of syncope. One of 2 in that group who had ECHO was diagnosed with tricuspid insufficiency.

Two (8%) patients who had family history of cardiac disease (n=25) in close relatives before 45 years of age had abnormal findings on ECG. Findings were normal in all 23 who subsequently had ECHO. Comparison of those with and without positive family history for cardiac disease at young age did not reveal any significant differences for ECG or ECHO findings (p>0.05 for each).

Five (21.7%) children with heart murmur had abnormal findings on ECG. Three (15.7%) of 23 had pathological ECHO findings. There was no significant difference in ECG or ECHO findings between those with heart murmur and those without (p>0.05 for each).

DISCUSSION

There is great heterogeneity among cardiac screening programs conducted for athletes before they join sports activities and such screening is performed in only a few countries.[7]

There is structural cardiac abnormality in approximately 90% of SCD cases.[5] Atherosclerotic heart disease is most common cause of sudden death in athletes over 35 years of age; however, congenital or hereditary heart disease are typically cause of SCD in athletes between 12 and 35 years of age.[5,8] Risk of SCD is highest in athletes between ages of 15 and 19 years of age.[8]

Maron et al. reported that 90% of SCD cases were male.[9] National records in the USA indicated that non-traumatic athlete deaths were 5 times more frequent in males.[10] Another study reported death rate 9 times higher in males.[3] In the present study, it was determined that males were more frequently engaged in competitive sports, and all those disqualified from competitive sports following study were male. Higher

Table 4. The correlations of the symptoms in athletes with ECG and ECHO findings (n=250)

Findings	ECG findings positive	ECG findings negative	*p*
Dyspnea (n=17)	4	13	0.66
Chest pain (n=22)	1	21	0.70
Palpitations (n=16)	6	10	0.02
A positive family history (n=25)	2	23	0.56
Murmur (n=23)	5	18	0.54
Findings	**ECHO findings positive**	**ECHO findings negative**	***p***
Dyspnea (n=17)	1	10	0.68
Chest pain (n=22)	1	18	0.58
Palpitations (n=16)	1	8	0.61
A positive family history (n=25)	0	10	0.64
Murmur (n=23)	3	20	0.25

ECG: Electrocardiography; ECHO: Echocardiography.

death rate in young males may be related to greater participation of males in competitive sports. In addition, male gender is risk factor in itself, as phenotypic expressions and prevalence of diseases that may cause SCD have higher expressions in males between the ages of 12 and 35 years. SCD may occur due to genetic abnormality affecting key proteins of the heart. LQTS, Brugada syndrome, hypertrophic cardiomyopathy (HCMP), arrhythmogenic right ventricular dysplasia (ARVD), catecholaminergic polymorphic ventricular tachycardia, and dilated CMP are well-known monogenic diseases causing tendency for SCD.

Age is important parameter for predicting underlying heart disease. Corrado et al.[4] studied sudden death in athletes and non-athletes of 12 to 35 years of age and suggested that sports activities increased risk of sudden death 2.5 times in this age group. Authors reported that CMP was most frequent cause of sudden death in athletes, followed by hidden cardiovascular disease, including premature coronary artery disease and congenital abnormalities of coronary arteries. In our study, dilated CMP was diagnosed in 1 participant in this age range.

HCMP was determined to be leading cause of SCD in the USA; however, most common cause was found to be ARVD in Italy, since deaths related to HCMP were minimized (2%) after implementation of standardized sportsman screening programs.[3,8] Most SCD in athletes younger than 35 years of age is result of congenital heart disease, most frequently HCMP (36%) and congenital coronary artery abnormalities (17%). ARVD, myocarditis, MVP with arrhythmia, Wolf-Parkinson-White syndrome, dissection of aorta, and acquired coronary artery disease are less frequent causes.[11] Rate of coronary artery disease was 80% in athletes studied who were older than 35 years of age. Maron et al.[7] reported that SCD was related to HCPM in 46% of cases and coronary artery disease in 19%. Another study reported that 40% of sudden deaths occurred before 18 years of age, and 33% occurred before 16 years of age. Male/female ratio was determined to be 9/1.[2] Occurrence of deaths at young age indicates that cardiovascular screening is necessary for children before starting sports activities, as well as for those who are engaged in sports without previous cardiovascular screening.

Most frequent cause of sudden death in athletes is arrhythmia. Bradyarrhythmia and atrial and ventricular extrasystoles are common in athletes, and usually benign. Supraventricular arrhythmias such as atrial fibrillation, nodal reciprocal reentrant tachycardia and Wolf-Parkinson-White syndrome are rare. Ventricular tachycardia that develops secondary to HCMP, ARVD, LQTS, and congenital coronary artery disease are less frequent, but more dangerous.[12] Ventricular fibrillation is the most frequently seen tachyarrhythmia determined before SCD. Ventricular tachyarrhythmia is the most frequent heart rhythm causing cardiac arrest in cases with preexisting atrioventricular or intraventricular conduction defects. In our study, we found abnormal ECG findings in only 10.4% of cases. RBBB was most frequent disorder, and there was no SCD related to arrhythmia.

Common characteristics of cardiac screening programs used in the USA and European Union are performing physical examination and obtaining personal and family history. Use of advanced methods, such as ECG and ECHO, is a source of debate.

In the USA, preparticipation screening programs only include history and physical examination. On the other hand, 12-channel ECG is used in addition to history and physical examination in Italy. An Italian study reported that HCMP was responsible for SCD in only 2% of athletes; however, it was responsible for 7.3% of SCDs in young people who were not athletes. When results of Italian study are compared with results of Burke, from the USA, it can be seen that rate of HCMP was similar in SCD not related to sports; however, rate of HCMP in SCD associated with sports is quite different in the 2 studies (24% vs 2%).[13] This may be interpreted as rate of sudden death related to HCMP may be minimized by determining athletes with HCMP through screening programs and disqualifying them from participating in sports activities thereafter.[14]

It has been reported that HCMP was responsible for 40% of deaths in athletic field in the USA.[6,15–17] Although ECHO is the most basic method in diagnosis of HCMP, its use in large populations is expensive and impractical.[6] Twelve-channel ECG is cost-effective, alternative method for population-based imaging. Italian studies have demonstrated that use of ECG in addition to history and physical examination of young athletes resulted in precise diagnosis of HCMP. In an Italian study, rate of HCMP in athletes was found to be 0.07% based on history, physical examination, and

12-channel ECG,[17] and this rate is compatible with rate determined in the USA (0.1%) where ECHO was used. This indicates that ECG used in Italian program is as sensitive as ECHO in HCMP screening.

In Italy, rate of athletes who needed ECHO after positive history, abnormal physical examination findings, or abnormal ECG was 8.9%; this rate was 29.2% in our study. Presence of more symptoms and positive family history, as well as higher rate of ECG abnormalities in our study compared to other studies may provide explanation.

Use of ECHO in addition to basic protocol does not substantially change effectiveness of imaging in diagnosis of HCMP. Pelliccia et al. performed ECHO in athletes with normal ECG, and did not diagnose any case of HCMP.[7] This supports hypothesis that sensitivity of ECG is as high as ECHO in diagnosis of HCMP. In the present study, we did not diagnose HCMP with ECG or ECHO in any of the athletes.

We found rate of cardiac disorders lower than other studies in the literature, and this may be related to previous examination of our cases by a pediatrician, and approval to participate ın sports activities. It has also been demonstrated that since rate of cardiac disease determined by pediatric cardiologist is very small (3/250) when Lausanne Recommendations are fully implemented by pediatricians and family physicians, they can conduct assessment for participation in sports and physical activities first without need for additional evaluation by pediatric cardiologist.

Conclusion

Sports and medical communities must work together to establish study protocols to prevent sudden deaths related to sports activities and to make sports safer for athletes. Results of this study indicate need to include 12-channel ECG in screening program for athletes before they begin sports activities due to proven good performance in determining HCMP and preventing sudden deaths. Periodic physical examination, beginning before participating in sports activities, as well as review of medical history and family history of the athlete, ECG, ECHO, and health and safety precautions implemented during and after sports activities may prevent sports-related SCDs. Invasive tests, including contrast ventriculography, coronary angiography, endomyocardial biopsy, and electrophysiological studies, may be used to confirm suspicion of heart disease in uncertain cases, as recommended by Working Group on Cardiac Rehabilitation and Exercise Physiology of the European Society of Cardiology.

Financial support

This research received no specific grant from any funding agency commercial, or not-for-profit sectors.

Conflict-of-interest issues regarding the authorship or article: None declared

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Key words: Arrhythmias; echocardiography; physical activity; sports.

Anahtar sözcükler: Aritmi; ekokardiyografi; fizik aktivite; spor.