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SCREENING AND COMPARISON OF ELITE MALE AND FEMALE SOCCER PLAYERS FOR ECG CHANGES ASSOCIATED WITH SUDDEN CARDIAC DEATH

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ABSTRACT

Young athletes are twice as prone to sudden cardiac death (SCD) when compared to young non-athletes. The paper aimed to determine the differences in ECG changes between elite male and female soccer players and how ECG changes can be used to predict the possibility of SCD. In this cross-sectional study, 148 elite soccer players (77 men, 71 women), 14-21 years, were randomly selected. Following the anthropometric measurements, an ECG was used to determine the parameters related to sudden death. The American Heart Association questionnaire (AHA) was used to determine the demographic information and health history of participants. Related to normal ECG findings, a significant difference was found between genders for right ventricular hypertrophy, sinus bradycardia and sinus arrhythmia. Increased QRS voltage and early repolarisation/ST-segment elevation were seen only in men. Related to incomplete Right Bundle Branch Block (RBBB), no significant differences were observed between genders in terms of T wave inversion in V1-V3, ectopic atrial or junctional rhythm, and 1°AV (atrioventricular) node block. Related to abnormal ECG findings, there were significant differences between genders for T wave inversion and T wave inversion local. Using an ECG during screening and check-ups is vital for all soccer players.

***Keywords*:** Athletes; Cardiac death; ECG; Soccer; Sudden cardiac death.

INTRODUCTION

Since sudden cardiac death (SCD) is counterintuitive in an apparently healthy athlete, this topic has attracted increasing attention (Bakkum *et al*., 2011; Emery & Kovacs, 2018). The increasing incidence of SCD in athletes necessitates the need for adequate pre-screening and identification of athletes at risk for cardiac events/disease (Bakkum *et al*., 2011). While exercise training does result in physiological adaptations to the cardiac structure, such as "athlete's heart", such adaptation, whether positive or negative, are visible on an electrocardiogram (ECG) and vary according to age, gender and ethnicity, as well as the type

and intensity of exercise training (Parry-Williams & Sharma, 2018). Some of these athletic changes may coincide with those seen in inherited cardiac conditions, such as cardiomyopathies and channelopathies (Etheridge *et al*., 2018). These incidences are amongst the major causes of SCD during sport participation (Flannery & La Gerche, 2019). A significant proportion of these conditions can be detected with an ECG.

In some instances, further investigations are required where the ECG does not enable differentiation between physiology and pathology. These could involve the use of echocardiography, cardiac magnetic resonance imaging (MRI) and exercise stress testing (Costantini *et al*., 2019). Erroneous diagnoses can have serious implications and may result in unfair disqualification from sport or false reassurance. Hence, it is essential that physicians are knowledgeable about the electrical manifestations that can occur, as a result of physical training, and more specifically from different sports, such as soccer (Parry-Williams & Sharma, 2018; Abela & Sharma, 2019).

There are structural differences between male and female athletes' hearts in terms of size of cardiac chambers, cardiac output and cardiac muscle contractility (Krysztofiak *et al*., 2018; Malhotra & Sharma, 2018). When interpreting an ECG, it is imperative that physicians are aware of these differences (Berge *et al*., 2019; Hedman *et al*., 2019). Various clinical trials, literature reviews, epidemiological studies and outcomes data have identified gender differences in the incidence of SCD (Rodriguez *et al*., 1992; Larsen & Kadish, 1998; Gowda *et al*., 2006). These studies have also shown that gender differences may be an independent risk factor for certain cardiac arrhythmias (Bai *et al*., 2018).

PURPOSE OF STUDY

To our knowledge, there is not a specified cross-sectional study that has focused on ECG parameters and how they relate to SCD in elite soccer players in both genders. Besides focusing on gender-differences, this study also aimed to investigate how ECG parameters relate to cardiac repolarisation, which could be an essential predictor of SCD.

METHODOLOGY

Ethical considerations

The study was conducted in compliance with and in the spirit of the declaration of Helsinki and approved by the ethics committee of the University of Tehran, Iran.

Design and participants

In this cross-sectional study, 77 male and 71 female (14-21 years of age) elite soccer players were randomly selected using randomised block design among national registered soccer players in Iran. Inclusion criteria included elite soccer players with five years of professional soccer experience, at least one year in the Premier League of Iran and physically active. Prior to participation in this study, participants completed a consent form (Kargarfard *et al*., 2018).

Participants in this research were elite and registered individuals who were members of Tehran Province Premier League clubs and had been assessed for doping since such athletes are not permitted to take illegal supplements, according to World Anti-Doping Agency (WADA) laws. Players were not excluded for using commonly used supplements not against WADA laws. Players were excluded if they had any congenital cardiac disease, diagnosed

hypertension (a cut-off point of 130-139/80-89 was determined as hypertension Stage 1, and a cut-point of ≥140/≥90 was determined as hypertensive Stage 2 (Han *et al*., 2020)), any known congenital heart diseases, such as electrical conducting disorders, Wolf-Parkinson-White (WPW), any valvular heart disease and pathological conditions that could result in ECG abnormalities. The reason for excluding these diseases in this study is their effect on the ECG. Players with a history of taking ergogenic supplements were also excluded.

A physician completed the American Heart Association questionnaire (AHA) (Williams *et al*., 2019) and the players regarding their own and first-degree family medical history. A resting ECG followed, completed by an experienced physiologist, under the supervision of a Sports Medicine Specialist and analysed by a cardiologist.

Instruments and procedure

Resting heart rate and resting blood pressure were measured while seated after a five-minute rest with the auscultatory method according to the standards established by the American College of Sports Medicine (Pescatello *et al.,* 2014).

Stature and body mass were measured, and body mass index (BMI) was calculated for all players (Pescatello *et al*., 2014; Shariat *et al*., 2017). Body mass measurements were measured on a medical scale (Mettle DT Digital, Mettler-Toledo AG, Ch-8606 GreiFensee, Switzerland), while stature was measured using a standard wall-mounted stadiometer according to the methods proposed by the International Society for the Advancement of Kinanthropometry (ISAK) (Marfell-Jones *et al*., 2012; Shaw *et al*., 2016; Kargarfard *et al*., 2017). After five minutes of inactivity in the supine position, a 12-lead resting ECG (German Order-No.11-2200 electrocardiograph) was conducted. All abnormal automated ECG interpretations were manually reviewed for accuracy by the same expert cardiologist specialising in sports medicine.

Data analysis

After checking the normality of data, the t-test was used to analyse the data using SPSS version 19 software and statistical significance set at $p \leq 0.05$. The t-test for independent groups was used to compare the results of the males and the females.

RESULTS

Seventy seven (77) elite male and 71 elite female soccer players between 14 and 21 years of age (age: 18.65±1.48 years; BMI: 21.05±2.68kg/m^2; height: 168.6±8.6 cm) were recruited. There was no significant difference in BMI between male and female players (t=1.112; df (degrees of freedom)=146). Ranges of heart rate per minute in all athletes were between 46 and 112 (resting HR) and were significantly higher among female players (t=2.123; df=146).

In terms of normal ECG findings, previously described in Drezner *et al.* (2017) (Table 1), the study found increased QRS voltage for left ventricular hypertrophy in 24 males and none among the females. Right ventricular hypertrophy was found in 25 males and 3 females, and there was a significant difference between the genders (t=3.165; df=146). Early repolarisation/ST-segment elevation (J point elevation, ST elevation, J waves or terminal QRS slurring in the inferior and/or lateral leads) was observed among 24 males. While sinus and bradycardia (≤60bpm) is a common finding in athletes, profound sinus bradycardia (≤30bpm) (Wilcox *et al*., 2013) was seen in 27 males and 14 females. There was a significant difference between the genders in terms of profound sinus bradycardia (t=3.212; df=146; $p \leq 0.05$).

Table 1. **NORMAL ELECTROCARDIOGRAPHY FINDINGS**

Variable	Definition	Total	Males	Females	p-value (t-value)
Increased QRS voltage for LVH	Isolated QRS voltage criteria for left (total %)	24	24	0	<0.001* (3.40)
	SV1, mm	10.1±4.2	11.5±4.5	8.6±3.2	<0.001** (3.62)
	RV5-V6, mm	15.6±6.4	19.6±5.6	11.2±4.0	<0.001** (3.30)
	SUM(SV1 + RV5 or RV6 >35 mm)	25.7±9.2	31.2±8.0	19.8±6.2	<0.001** (4.13)
RVH	Right ventricular hypertrophy (total %)	28	25	3	<0.001* (3.78)
	RV1, mm	2.9±1.7	3.7±1.6	2.2±1.4	<0.001* (4.11)
	SV5-V6, mm	3.3±2.6	4.2±3.0	2.4±1.6	<0.001* (3.63)
	SUM(RV1 + SV5 or SV6)	6.2±3.1	7.9±3.1	4.5±1.7	<0.001* (3.86)
	RV1 + SV5 or SV6 >11 mm	13	13	0	<0.001* (3.56)
	R or R' wave in lead V1≥5mm in amp	21	18	3	0.001*
	R/S ratio ≥ 1	0	0	0	-
Incomplete RBBB	rSR' pattern in lead V1 and a qRS pattern in lead V6 with QRS duration <120 ms	13	7	6	0.891 (1.21)
Early repolarisation/ ST segment elevation	J point elevation, ST elevation, J waves or terminal QRS slurring in inferior and/or lateral leads	24	24	0	<0.001* (3.71)
T wave inversion V1-V3	T wave inversion V1-V3 in athletes less than age less than 16	2	1	1	0.696 (1.11)
Sinus bradycardia	≤30 bpm	27	23	4	<0.001* (3.70)
Sinus arrhythmia	Heart rate variation with respiration: Rate increases during inspiration and decreases during expiration	41	27	14	0.037* (2.1)

Table 1. **NORMAL ELECTROCARDIOGRAPHY FINDINGS *(cont.)***

Variable	Definition	Total	Males	Females	p-value (t-value)
Ectopic atrial or junctional rhythm	P waves are a different morphology compared with the sinus P wave, such as negative P waves in the inferior leads ('low atrial rhythm')	7	5	2	0.292 (1.32)
Junctional escape rhythm	QRS rate is faster than the resting P wave or sinus rate and typically less than 100 beats/min with narrow QRS complex unless the baseline QRS is conducted with aberrancy	0	0	0	-
1°AV block	PR interval 200–400 ms	2	1	1	0.954 (1.42)
2°AV block Mobitz type I (Wenckebach)	PR interval progressively lengthens until there is a non-conducted P wave with no QRS complex; the first PR interval after dropped beat is shorter than last conducted PR interval	0	0	0	-

Data=Mean±SD * Statistically significant=p≤0.05 mm=millimetre ms=milliseconds AV=Atrioventricular LHV=Left Ventricular Hypertrophy RVH=Right Ventricular Hypertrophy RBBB=Right Bundle Branch Block

Sinus arrhythmia (irregular heartbeat, either too fast or too slow, including respiratory sinus arrhythmia) was seen in 27 men and 14 women, and there was a significant difference between the genders (t=2.011; df=146; p≤0.05). Although T-wave inversion in leads V1–V4 in some athletes (black athletes) may represent ethnic variation, inverted T-waves may represent the first and only sign of such inherited heart muscle diseases. Inverted T-waves may precede the detection of any structural changes in the heart. As such, in terms of ECG findings not consistent with athlete heart changes (Table 2), T wave inversion was found in 11 players (8 males and 3 females).

A significant difference (t=3.145; df=146; p=0.040) was seen between the genders related to T wave inversion (≥1mm in depth in two or more contiguous leads; excludes leads aVR, III and V1). In the current study, T-wave inversion location (Anterior: V2-V4, excludes: black athletes with J-point elevation and convex ST-segment elevation followed by TWI (T-Wave Inversion) in V2-V4; athletes age <16 years with T-Wave Inversion (TWI) in V1-V3; and biphasic T-waves in only V3 was also observed among the same 11 players.

***Table 2*. ABNORMAL ELECTROCARDIOGRAPHY FINDINGS**

Variable	Definition	Total	Men	Women	p value (t value)
T wave inversion	≥1mm in depth in two or more contiguous leads; excludes leads aVR, III and V1	11	8	3	0.040* (1.99)
T wave inversion Location	Anterior: V2-V4 excludes: black athletes with J-point elevation and convex ST segment elevation followed by TWI in V2-V4; athletes age <16 with TWI in V1-V3; and biphasic T waves in only V3	11	8	3	0.040* (2.31)
	Lateral: I and AVL, V5 and/or V6 (only one lead of TWI required in V5 or V6)	0	0	0	-
	Inferolateral: II and AVF, V5-V6, I and AVL	0	0	0	-
	Inferior: II and AVF	0	0	0	-
ST segment depression	≥0.5 mm in depth in two or more contiguous leads	0	0	0	-
Pathologic Q wave	Q/R ratio ≥0.25 or ≥40 ms in duration in two or more leads (excluding III and aVR)	0	0	0	-
Complete LBBB	QRS ≥120 ms, predominantly negative QRS complex in lead V1 (QS or rS) and upright notched or slurred R wave in leads I and V6	0	0	0	-
QRS≥140ms duration	Profound non-specific intraventricular conduction delay	0	0	0	-
Ventricular pre excitation	PR interval <120 ms with a delta wave (slurred upstroke in the QRS complex) and wide QRS (≥120 ms)	0	0	0	-

Table 2. **ABNORMAL ELECTROCARDIOGRAPHY FINDINGS *(cont.)***

Variable	Definition	Total	Men	Women	p value (t value)
Prolonged QTC interval	QTc ≥470 ms (Male), QTc ≥480 ms (Female)	1	0	1	0.296 (1.41)
	QTc ≥500 ms (marked QT prolongation)	1	0	1	0.296 (1.18)
Profound sinus bradycardia <30	<30 beats per minute or sinus pauses ≥3 s	0	0	0	-
PR interval≥400ms	≥400 ms	0	0	0	-
2° AV block Mobitz Type II	Intermittently non-conducted P waves with a fixed PR interval	0	0	0	-
3° AV block	Complete heart block	0	0	0	-
≥2 PVCs	≥2 premature ventricular contractions per 10 s tracing	1	0	1	0.333 (1.22)
Atrial tachyarrhythmias	Supraventricular tachycardia, atrial fibrillation, atrial flutter	0	0	0	-
Ventricular arrhythmias	Couplets, triplets and non-sustained ventricular tachycardia	0	0	0	-

* Statistically significant (p≤0.05)
LBBB=Left Bundle Branch Block PVCs=premature ventricular contractions AV=Atrioventricular
QTc=corrected QT interval (*estimates* the QT interval at a standard heart rate of 60 bpm)

DISCUSSION

The research aimed to determine the differences in ECG changes between elite male and female soccer players and how ECG changes can be used to predict the possibility of cardiac abnormalities that correlate with SCD based on international criteria for defining T-wave inversion (Drezner *et al*., 2017). In this regard, the findings of this study showed significant differences in the ECG changes between elite male and female soccer players.

With regard to normal ECG findings, the study found significant differences between genders for right ventricular hypertrophy, sinus bradycardia and sinus arrhythmia. Increased QRS voltage and early repolarisation/ST-segment elevation were seen only among males, and this was in line with findings of previous studies (Yarnoz & Curtis, 2008; Rijnbeek *et al*., 2014). Normal ECGs are different in men and women due to the presence of sex hormones or physiological and athletic adaptations (Gowda *et al*., 2006). In addition, related to incomplete RBBB, T-wave inversion V1-V3, ectopic atrial or junctional rhythm, and 1°AV block, there were no significant differences between genders.

While it is not the structural differences and normal ECG variants between males and females that predict SCD, based on the abnormal ECG findings, there were significant differences between genders for T-wave inversion according to the definition of international criteria (Drezner *et al*., 2017). Related to ventricular arrhythmia, females are more prone than males, which is likely due to the electrophysiological differences between genders (Makkar *et al*., 1993; Fung *et al*., 2000).

In this regard, the interplay of anatomic, structural, hormonal, autonomic and genetic factors could be the reason for differences between men and women related to the different ECGs (Gowda *et al*., 2006). It is clear that the incidence of cardiac diseases and the related frequency, could be related to gender and its interaction with age (Mercuro *et al*., 2010; Moss, 2010). It has previously been demonstrated that a shorter early repolarisation could be the reason for that shorter QTc (corrected Q-T interval) in males in comparison to females (Vicente *et al*., 2014). The effect of testosterone on calcium could explain this difference in repolarisation (Vicente *et al*., 2014).

The larger hearts of males could result in a longer QRS duration (Hnatkova *et al*., 2016; Lee *et al*., 2017). However, previous studies on gender differences in T_{peak}-T_{end} are inconclusive (Braschi *et al*., 2017; Macfarlane, 2018), and current results are in accordance with those reporting that males have longer late repolarisation than females at resting heart rates. T_{peak} - T_{end} is defined as the interval between the peak of the T-wave and the end of the T-wave, representing the dispersion of repolarisation (Tse & Yan, 2017).

In support of the present study's findings, previous research has shown that the QRS widths are not only different between genders, but also between different races, such as Black and Caucasian (Hnatkova *et al*., 2016), demonstrating QRS can be influenced by factors, such as genetics and race. All the participants in this study were of Caucasian descent, and the issue of race was not examined and cannot be considered.

The findings of this study confirmed that the QRS complex and the area under the T-wave are different between genders. In this regard, the QRS complex amplitude is larger in males and especially pronounced in the precordial leads and, as such, many clinical arrhythmias are related to differences (Ravens, 2018). Furthermore, the frequency of underlying structural heart disease (Westerman & Wenger, 2016; Ravens, 2018) and progesterone and estrogen concentrations that fluctuate during the menstrual cycle can explain this phenomenon.

CONCLUSION

Most victims of exercise-related SCD have no premonitory symptoms (Siscovick, 1993), but thankfully, various ECG methods exist to predict SCD in soccer players (Bakkum *et al*., 2011; Flannery & La Gerche, 2019). Problematically, ECG changes in athletes are common and usually reflect structural and electrical remodelling of the heart as an adaptation to regular physical training. In rare cases, abnormalities of an athlete's ECG may be an expression of an underlying heart disease putting the athlete at risk of SCD during sporting events. As pathological ECG abnormalities not only cause alarm, but also require action with additional testing to exclude (or confirm) the suspicion of a lethal cardiovascular disorder, appropriate interpretation of an athlete's ECG is necessary to prevent unnecessary diagnosis (Corrado *et al*., 2009). As such, the present study provides novel information as to the electrical manifestations that can occur in both male and female soccer players as a result of physical training.

Conflict of interest

The authors reported no potential conflict of interest.

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