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Electrocardiographic and Echocardiographic Assessment of Young Black Female Footballers: Comparison with Sedentary Female Individuals

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ABSTRACT

Background: Regular, high-intensity, and prolonged physical activity causes clinical, electrical, morphological, and functional alterations in the cardiovascular system. This circulatory alterations observed in athlete heart are particularly important in black athletes as data has shown that they are more likely to experience sudden cardiac death. Comparative electrocardiographic and echocardiographic data between women who play organized football and women who are inactive are scarce particularly in African nations.

Objective: To compare the cohort group's electrocardiogram and echocardiogram to inactive women in order to determine the range of physiological adaptation in highly trained young Nigerian female football players.

Materials and Methods: This was a cross-sectional study performed among 30 high-level female footballers and 30 untrained women as control group. Study participants were assessed with a health questionnaire and targeted cardiovascular examination, 12 lead ECG and two-dimensional echocardiography were done. Ethnicity was self-assigned. A 95% confidence interval was used while statistical significance was set at $P < 0.05$.

Results: The prevalence of at least one abnormal ECG findings was significantly higher among the footballers than the control. The footballers group had more arrhythmias recorded (sinus bradycardia)

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compared to the control group. The prevalence of at least one ECG abnormality in the patient was 35.8% and comparable with the control group of 28.0% (p=0.249) and were all minor abnormalities. The mean corrected QT prolongation (QTc) of the football group was significantly higher than the control. The mean left ventricular mass index, left ventricular internal diameter in diastole, left atrial diameter andthe prevalence of increased mitral E/A ratio (supernormal diastolic function) and any valvular abnormalities among the female footballers were significantly higher than the control. However, the mean ejection fraction of the footballers (64.72±6.55%) and that of the controls (63.63±6.87%) were not significantly different (p=0.530).

Conclusion: Specific physiological adaptations were found more among the footballers. Though most of the findings were benign, the study has demonstrated the need for regular screening and follow-up of footballers to enable early detection of potential life-threatening ECG and Echocardiographic changes.

Keywords: ECG, Echo, Physiological adaptations, footballers

INTRODUCTION

Though helpful, frequent, high-intensity, and prolonged physical activity causes clinical, electrical, morphological, and functional alterations in the cardiovascular system [1]. The benign circulatory alterations observed in elite athletes are referred to as "athlete's hearts" [1]. An athlete's heart's anatomical and electrical manifestations are significantly influenced by their ethnicity [2]. The difference between an athlete's heart and cardiac pathology is especially important in this group because data from the USA show that adolescent black athletes are more likely to experience sudden cardiac death (SCD) [3, 4]. Since 85% to 90% of non-traumatic sudden deaths on sports fields are cardiovascular in nature, typically due to cardiac arrhythmias, guidelines for screening for cardiovascular abnormalities have been developed for sporting practice [5, 6, 7, 8]. There is a dearth of information about cardiac status of sportsmen in general and football players in particular in African nations, although sports competition and practice are becoming more and more professionalized. Due to the fact that football is primarily played by men and that many women view it as a sport for men, women are underrepresented in football sports. [9]. Comparative electrocardiographic and echocardiographic data between women who play organized football and women who are inactive are scarce. The purpose of this study was to compare the cohort group's electrocardiogram and echocardiogram to inactive women in order to determine the range of physiological adaptation in highly trained young Nigerian female football players.

MATERIALS AND METHODS

This was a cross-sectional study performed in Ado-Ekiti, Ekiti State Nigeria over a period of 3 months among high-level female footballers and a group of untrained women. Included were all aged 18 years and above consenting female subjects either as sportswomen (footballer) or as control (those not involved in serious /organized physical activity) after informing participants of the purpose of the study. Consenting female footballers were recruited from the state-owned football club while for the control group, young females volunteerswere randomly selected among the students of Ekiti State University, Ado-Ekiti, Nigeria. Inclusion criteria for the female footballers was age 18- 45 years, training duration for at least 10 hours weekly for at least one year. The control group consisted of those who did not engage in regular physical activity. Subjects with diagnosed hypertension, diabetes or symptoms suggestive of underlying cardiovascular disease, history of treatment or previous diagnosis of any chronic disease such as chronic renal failure, chronic liver or lung disease, sickle cell disease and/or regular consumption of alcohol or tobacco were excluded. Study participants were assessed with a health questionnaire and height and weight measurements were taken to the nearest 0.1 cm and 50 g respectively. Body mass index (BMI) was

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calculated as weight/height^2 in units of kg/m2. Targeted cardiovascular examination, 12 lead ECG and two dimensional echocardiography were done. Ethnicity was self-assigned.

ELECTROCARDIOGRAPHY

The 12-leadselectrocardiogram was recorded using Zoncare ZQ 1203G. Every participant was laying supine in a noise-free setting while the resting 12-lead ECG was recorded at a paper speed of 25 mm/sec and vertical calibrations of 1 mV=10 mm after 5 minutes of rest. Standardization of leads and specification was done according to the recommendations of the American Heart Association/American College of Cardiology (AHA/ACC) [10, 11, 12 ,13]. The ECG parameters determined include heart rate, rhythm, cardiac axis, amplitude and duration of the P wave, PR intervals, QRS duration, QRS amplitude, ST segment, T wave, and observed mean QT. Amplitudes were recorded to the nearest 100th of a millivolt and duration to the nearest millisecond. Left ventricular hypertrophy (LVH) was determined using Sokolow-Lyon criteria [14]. Observed QT (QTo) was measured from the beginning of the QRS complex to the visual return of the T-wave to the iso-electric line using lead II and the preceding R-R interval was also determined. QTc was calculated by applying Bazett's formula QTc = QTo /√R-R [15]. At least three consecutive cycles were measured and then averaged. A QTc value of 460 ms was considered to be abnormally prolonged for female gender [16]. ECG abnormalities were divided into minor and major abnormalities based on Novacode criteria [17].

ECHOCARDIOGRAPHY

Transthoracic 2D derived M-mode and conventional pulsed wave echocardiography were performed via parasternal and apical windows using a Toshiba Aplio 400 ultrasound machine equipped with a 3.5MHz cardiac transducer. Standard views were obtained and cavity and wall thickness measurements were performed using established guidelines [18]. Left atrial (LA) diameter and left ventricular (LV) internal diameter were measured from the parasternal long axis view. Left ventricular wall thickness was measured in the parasternal short-axis view, at the levels of the mitral valve and papillary muscles; the greatest measurement was defined as the maximum left ventricular wall thickness (mLVWT). Left ventricular mass was calculated with the formula of Devereux [19]. LVH was defined as left ventricular mass index ≥95g/m^2. Relative LV wall thickness (RLVWT) was calculated by dividing the end diastolic LV internal diameter by twice the posterior wall thickness [20]. Eccentric LV hypertrophy defined by increased LV mass and a RLVWT < 0.42 while LV concentric hypertrophy is defined by increased LV mass and a RLVWT > 0.42 [20]. Two-dimensional continuous-Doppler and pulsed-Doppler imaging were performed using standard parasternal and apical views [21].

The velocities of early (E) and late (A)transmitral and transtricuspid flow, the E/A ratio and deceleration time of the E-wave were measured. Normal diastolic function: E/A=>1-2, DT=130-230msec while supernormal diastolic function (enhanced E/A ratio) is E/A>2, DT=130-230msec [22]. The measurements were derived from the average of three consecutive cycles and ECHO variables were determined.

Statistical Analysis

Statistical analyses were performed using IBM SPSS software, version 28 (Chicago, Illinois, USA). Variables were tested for normality using the Kolmogorov-Smirnov test. Group mean differences were tested using Student's t-test or one-way ANOVA (analysis of variance) and Mann-Whitney U test or Kruskal Wallis for normally and non-normally distributed variables, respectively. The chi-square test or Fisher's exact tests were used as appropriate to test group differences of proportions. The significance level was $p < 0.05$.

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Ethical Consideration

All consecutive presenting female football players and control who were 18 years and older who met the inclusion criteria as stated above were recruited. All recruited patients provided signed informed consent. Patients were assured that information would be maintained confidential. The questionnaire did not include any identifiers of respondents.

RESULTS

A total number of 60 subjects were included and completed study. These included thirty female football players and thirty control group. Table 1 demonstrates basic clinical parameters of the study participants. The mean age in both groups were similar, however, the mean heart rate, systolic blood pressure, diastolic blood pressure and body mass index of the control group were significantly higher than those of the footballers. Table 2 demonstrated ECG findings of the female footballers. The prevalence of at least one abnormal ECG findings was significantly higher among the footballers than the control. The footballers group had more arrhythmias recorded (sinus bradycardia) compared to the control group. Across study groups, the mean PR duration, QRS duration, mean QTc as well as the prevalence of chamber enlargement, atrioventricular conduction block, intraventricular conduction defect, abnormal ST segment and T wave abnormalities were not statistically significantly different.The mean corrected QT prolongation (QTc) of the football group was significantly higher than the control; however, the prevalence of prolonged QTc in the footballers group is 3.3% which was not statistically different from the control group. In order of decreasing frequency, the most common abnormal ECG findings among the footballers were sinus bradycardia, LVH, early repolarization changes and T wave abnormalities; while among the controls, LVH and first degree AV block were the most frequent abnormal ECG patterns.Overall among the study subjects, the ECG abnormalities observed were all minor abnormalities based on Novacode Criteria.

Table 3 showed echocardiographic parameters of the study population. The mean M-Mode ejection fraction of the footballers (64.72±6.55%) and that of the controls (63.63±6.87%) were not significantly different (p=0.530). The mean left ventricular mass index, left ventricular internal diameter in diastole and left atrial diameter of the footballers were significantly higher than the control but the mean aortic diameter was significantly lower than the control. There is significant higher prevalence of left ventricular supernormal diastolic function among the female football group compared to the control. The overall prevalence of any valvular abnormalities among the female footballers group was significantly higher than the control group. Likewise the prevalence of pulmonary and tricuspid regurgitations was significantly higher than the control. The prevalence of abnormal LV geometry was found in 20% of the footballers compared to6.7%of the sedentary women (p = 0.254). Among the 20% with abnormal LV geometry, half had LV hypertrophy while none had LV hypertrophy in the sedentary group.

DISCUSSION

This study compared the ECG and echocardiographic changes between female footballers and a control group (those not involved in regular physical activity). On ECG, the footballers' group had more arrhythmias compared to the control group while ECHO showed thatvalvular abnormalities were observed more with the footballers' group. Bradycardia was significantly higher among footballers (36.7%). This could be due to the effect of training on heart rate through vagal hypertonia. None of the footballer subjects had tachycardia. This is similar to the observation by Sangare*et al* in a study done among female football athletes in Bamako, Mali [23]. Exercise can induce an AV-block at rest, which indicates a rise in parasympathetic tone and a fall in sympathetic tone. A study conducted among athletes in Greece by Papadakis et al among male athletes of African/Afro-Caribbeanorigin in the United Kingdom (UK) and

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France found 11.2% of them with first degree Av block while Sheikh reported 8.9% of first degree AV-block among adolescent African/Afro-Caribbean ethnicity, of whom majority were male (74.5%) [24, 25]. These rates were greater than ours (3.3%) and the fact that the majority of the subjects in those studies were male and had higher vagal tones than female subjects may account for this discrepancy [26]. There was no significant difference between the duration of QRS in sportswomen and the control (P = 0.435). This is similar to the study done by Sangaré et al [23]. The prevalence of left ventricle hypertrophy (LVH) according to the Sokolow-Lyon index was similar to what was found in the control group. Rawlins reported that the prevalence LVH was 8.2% among black female athletes in UK and France and this is similar to the prevalence of 10% in our study [2]. The mean BazettQTc duration among the female football group was significantly higher than the sedentary group ($p<0.004$). Athletes frequently have bradycardia, which causes the QT interval to be significantly prolonged. The prolonged QTc has been linked to delayed repolarization brought on by an increase in left ventricular mass in athletes or a maladaptation of Bazett's formula to low heart rates [27]. However, the prevalence of prolonged QTc was not statistically significant between the groups. Early repolarization was observed in 10% of the footballers while none had it reported among the control group. Early repolarization changes are generally common in black athletes and it is reported in as much as 40% among male athletes of African/Afro-Caribbean origin [24]. The negative T wave accounted for 6.6% among the female footballers which was not statistically significant from the control**.** Although the overall prevalence of ECG abnormalities was significantly higher among the female footballers, they were all benign.

The mean diameter of the left ventricle and left atrium were larger in the footballers' group when compared to control. These findings are similar to reports from other studies [23, 24, 25]. The Echocardiographic findings revealed that the mean left ventricular (LV) mass index was significantly higher in the footballers' group compared to the control. The LV geometry was abnormal in 20% of female footballers compared to 6.7% of the control. This is similar to the prevalence of 17.3% of abnormal LV geometry reported by Yeo *et al*. among the female cohort of Singapore athletes [28]. Power/static activities subject the cardiovascular system to elevated blood pressure during short bursts, which results in a chronic adaptation characterized by concentric remodelling, however, endurance sports are characterized by eccentric remodelling[29]. Soccer is a mixture of both endurance and power components hence the ECHO findings showed some concentric and eccentric cardiac remodeling. Active athletes are considered to benefit from regular training because it increases the left ventricular cavity's volume, which increases the flow of oxygen to the working muscles during exercise [30].

An increase in the left atrium, left ventricular mass index, and left ventricular internal diameter (LVID) in this study can be explained by the heart's physiological response to repeated, severe exercise. As a result of the continuous and intense increase in cardiac output that occurs during physical exercise, chamber dilatation may be associated to volume overload [31]. This discovery aligns with the findings of other researchers [23, 32, 33]. All our study participants had good left ventricular function, and the left ventricular ejection fraction of the footballers was comparable with that of the control. Female footballers demonstrated significant increased mitral E/A ratio (supernormal diastolic function) due to an increase of E velocity and decrease of A velocity. Kneffel et al. reported that athletes' increased E/A was associated with a lower resting heart rate [34]. It has been documented that endurance-trained athletes have improved LV diastolic function, and elevated LV filling index values distinguish the athletic heart from other pathologic hypertrophy such as hypertrophic cardiomyopathy [35].

Active sport participants without a known heart condition and structurally normal valves have been shown to have a much greater overall prevalence of functional valve regurgitation rate than sedentary controls [36, 37]. Comparing athletes to their non-athlete counterparts, there is a notable dilatation of the valvular annulus and an increase in the tenting of the atrioventricular valves, which has been linked to significantly greater functional valvular regurgitation [38].It's interesting to note that tricuspid regurgitation affected 60% of the

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female football players. Athletes frequently experience mild TR, which is accompanied by physiological dilation of the inferior vena cava, which readily collapses upon inspiration. All levels of exercise are still appropriate for those with mild TR, no RV dysfunction, resting sPAP> 50 mmHg, and right atrial pressure > 20 mmHg [39].

The limitations of the study include the cross-sectional nature of this study, which prevented us from drawing any inferences about causality. In addition, its small sample limits the generalizability of the study findings.

CONCLUSION

Young female footballers exhibited similar ECG and ECHO findings compared with their non-physically-active counterparts. Specific physiological adaptations, such as sinus bradycardia, lower systolic and diastolic blood pressure, prolonged mean QTc duration, left ventricle mass index, left atrial enlargement, left ventricular dilatation and functional valvular regurgitation were found more among the footballers. Though most of the findings were benign, the study has demonstrated the need for regular screening and follow-up of footballers to enable early detection of potential life-threatening ECG and Echocardiographic changes.

Authors contribution: Study concept and design was done by Ojo OE, analysis and interpretation of data by Ojo OE, Fadare JO, Olofinbiyi BA, Adegoke BO, Soje M and Oguntola BO, drafting the article or revising it critically for important intellectual content by OJO OE, Fadare JO, and Soje M, final approval of the version to be published Fadare JO, Olofinbiyi BA, Olaoye BO and Oguntiloye OO, agreement to be accountable for the accuracy and integrity of all aspects of the work were done by all authors.

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Table 1. Demographic and clinical characteristics of both study groups

Characteristics	Patients (30) Mean ± SD	Control (30) Mean ± SD	P-value
Age (years)	20.33±3.20	21.73±2.82	0.077
HR (beats/mi)	63.53±9.10	79.27±11.52	<0.001*
SBP	101.33±9.73	117.37±9.87	<0.001*
DBP	61.67±9.86	72.10±7.81	<0.001*

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Weight	57.80±7.91	66.53±8.64	<0.001*
Height	162.53±5.90	162.26±5.96	0.862
BMI(Kg/m^2)	21.87±2.40	25.34±3.63	<0.001*

HR- Heart rate, SBP-systolic blood pressure, DBP- diastolic blood pressure, BMI- body mass index*-significant

Table 2: ECG characteristics of study population

Variables	**Patients (30) Mean ± SD**		**Control (30) Mean ± SD**		**Statistical indices**
PR interval	169.47 ± 19.49		167.87 ± 22.88		0.772
QRS	86.63 ± 7.91		88.13 ± 6.84		0.435
QTc	413.53 ± 23.54		397.00 ± 19.01		0.004
	Patients (30)		**Controls (30)**		
Abnormal ECG findings	N	%	N	%	
	16	53.3	7	23.3	χ2 =5.711
Arrhythmias	11	36.7	2	6.7	¥p = 0.005*
Sinus tachycardia	0	0	1	1.7	¥p = 1.000
Sinus bradycardia	11	36.7	1	3.3	¥p = 0.002*
PAC	1	3.3	0	0	¥p =1.000
PVC	0	0	0		NC**
Atrial flutter	0	0	0	0	NC**
Atrial fibrillation	0	0	0	0	NC**
Junctional rhythm	0	0	0	0	NC**
Chamber enlargement	4	13.3	3	10	¥p =1.000
Left atrial enlargement	1	3.3	0	0	¥p =1.000
LVH	3	10	3	10	¥p =1.000
Right atrial enlargement	0	0	0	0	NC**
RVH	0	0	0	0	NC**
Atrioventricular (AV) block	1	3.3	2	6.6	¥p = 1.000
First degree AV block	1	3.3	2	6.6	¥p = 1.000
Second degree AV block	0	0	0	0	NC**
Third degree AV block	0	0	0	0	NC**
Intraventricular blocks (IVCD)	0	0	1	3.3	¥p = 1.000
LBBB	0	0	0	0	NC**
RBBB	0	0	0	0	NC**
LAFB	0	0	0	0	NC**
LPFB	0	0	0	0	NC**
Bifascicular block	0	0	0	0	NC**
Non specific IVCD	0	0	1	3.3	¥p = 1.000
ST segment changes (elevation or depression)	3	10	0	0	¥p = 0.237
Prolonged QTc	1	3.3	0	0	¥p = 1.000

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T wave abnormalities	2	6.6	1	3.3	¥p = 1.000
Novacode criteria					
Normal findings	14	46.7	23	76.7	$\chi 2$ = 5.711 P = 0.017
Minor abnormalities	16	53.3	7	23.3	$\chi 2$ =5.711 p =0.017
Major abnormalities	0	0	0	0	NC**

Key: PAC- Premature atrial contraction, PVC- Premature ventricular contraction, LVH- Left ventricular hypertrophy, RVH- Right ventricular hypertrophy, LBBB- Left bundle branch block, RBBB- Right bundle branch block, LAFB- Left anterior fascicular block, LPFB- Left posterior fascicular block, *- significant, NC** = Chi square statistics not computed as both cases and controls have the same proportions, ¥= Fisher's exact test, $\chi 2$= chi square

Table 3: ECHO characteristics of study population

Variables	**Patients (30) Mean ± SD**		**Control (30) Mean ± SD**		**Statistical indices**
LAD	3.21±0.35		2.96±0.32		0.008
AoD	2.66±0.21		2.82±0.29		0.017
LVIDd	4.51±0.41		4.19±0.47		0.008
LVMI	73.36±12.04		65.35±14.52		0.024
RWT	0.37±0.06		0.41±0.06		0.007
Ejection fraction	64.72±6.55		63.63±6.87		0.530
	Patients (30)		**Controls (30)**		
	N	**%**	**N**	**%**	
Any valvular regurgitation	23	76.7	10	33.33	$\chi 2$ =11.381 p =0.001
Any pulmonary regurgitation	15	50	7`	23.3	$\chi 2$ =4.593 p =0.030
Mild pulmonary regurgitation	6	20	6	20	$\chi 2$ =0.000 p =1.000
Moderate pulmonary regurgitation	9	30	1	3.3	¥p = 0.012*
Severe pulmonary regurgitation	0	0	0	0	NC**
Any tricuspid regurgitation	18	60	9	30	$\chi 2$ =5.455 p =0.020
Mild tricuspid regurgitation	13	43.3	7	23.3	$\chi 2$ =2.700

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					p =0.100
Moderate tricuspid regurgitation	4	13.3	2	6.7	¥p = 0.671
Severe tricuspid regurgitation	1	3.3	0	0	¥p = 1.000
Any mitral regurgitation	7	23.3	2	6.7	¥p = 0.146
Mild mitral regurgitation	6	20	2	6.7	¥p = 0.146
Moderate mitral regurgitation	1	3.3	0	0	¥p = 1.000
Severe mitral regurgitation	0	0	0	0	NC**
Any aortic regurgitation	2	6.7	0	0	¥p = 0.492
Mild aortic regurgitation	2	6.7	0	0	¥p = 0.492
Moderate aortic regurgitation	0	0	0	0	NC**
Severe aortic regurgitation	0	0	0	0	NC**
Left ventricular hypertrophy	3	10	0	0	¥p = 0.237
Abnormal LV geometry	6	20	2	6.7	¥p = 0.254
Left ventricular geometry					
Normal geometry	24	80	28	93.3	
Concentric remodeling	3	10	2	6.7	
Eccentric hypertrophy	2	6.7	0	0	
Concentric hypertrophy	1	3.3	0	0	
Diastolic function					
Normal left diastolic function	18	60	27	90	¥p = 0.015
Supernormal left diastolic function	12	40	3	10	¥p = 0.015
Normal right diastolic function	28	93.3	28	93.3	¥p = 1.000
Supernormal right diastolic function	2	6.7	2	6.7	¥p = 1.000