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Outcomes of cardiovascular screening in professional female football players

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ABSTRACT

Objective To investigate the results of cardiac screening, prevalence of cardiac diseases and subsequent health outcomes among elite female football players in the UK.

Methods Between 2000 and 2024, 3121 elite female football players underwent the English Football Association (FA) cardiac screening programme, comprising a health questionnaire, 12-lead ECG and echocardiogram. The FA registry was interrogated for screening findings, subsequent testing, diagnosis and cardiac morbidity or mortality, including sudden cardiac arrest and death (SCA/SCD). Sex-based comparisons were performed in 2680 females screened between 2017 and 2024, matched 1:2 by age, ethnicity and screening period to 5360 male players screened through the same programme.

Results Female athletes were 19.8±5.4 years old, and the majority (87%) were White. Among 3121 female football players, 193 (6.2%) had an abnormal screening result requiring further evaluation; positive health questionnaires were present in 321 (10.3%), of which two (0.6%) athletes were true-positive for major cardiac conditions. Abnormal ECGs were present in 77 (2.5%), of which four (5.2%) were true positives. Major cardiac conditions were diagnosed in six (0.2%), including four with long QT syndrome, one with Wolff-Parkinson-White syndrome and one with bicuspid aortic valve with moderate to severe aortic regurgitation. No female had cardiomyopathy diagnosed at baseline; however, one (0.03%) was diagnosed with arrhythmogenic cardiomyopathy 18 months later. During 19 193 person-years of follow-up, no female experienced SCA/SCD. In matched analysis, major cardiac conditions were diagnosed in five females (0.2%) and 29 males (0.5%; p=0.027), with higher estimated screening expenditure per major diagnosis in females (£146 809 vs £51 888).

Conclusions Cardiovascular screening in elite female football players identified a low prevalence of major cardiac conditions, predominantly electrical disorders, with no cardiomyopathy diagnosed at baseline and no SCA/SCD during follow-up. As female participation in elite sport continues to grow, these findings support the need for sex-appropriate interpretation and adequate budgeting for pre-participation cardiovascular evaluation.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Male athletes dominate the evidence base on the diagnostic yield and clinical outcomes of pre-participation cardiac screening programmes, with women historically under-represented. Available data suggest a lower incidence of sudden cardiac death in female athletes and a lower burden of structural cardiac disease at autopsy. However, the prevalence of quiescent cardiovascular disease among actively competing professional female athletes has remained unclear.

WHAT THIS STUDY ADDS

⇒ This study provides unique insights into the prevalence of cardiac conditions among elite female athletes and directly compares them with their male counterparts, along with longitudinal outcome data. In female football players, symptom reporting was more frequent but had low true-positive yield, while major cardiac conditions were predominantly electrical.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Screening pathways should therefore emphasise careful ECG interpretation, including corrected QT prolongation and repolarisation abnormalities, alongside sex-specific echocardiographic thresholds and structured follow-up of clinically relevant findings.

INTRODUCTION

The beneficial effects of exercise are overwhelming, and physically active individuals experience lower cardiovascular morbidity and a longer life span compared with sedentary individuals.[1 2] Paradoxically, vigorous exercise may trigger fatal arrhythmias in athletic individuals with underlying cardiac disease.[3] The incidence of sudden cardiac arrest (SCA) or death in athletes ranges from 1 in 15 000 to 1 in 200 000, depending on the demographic constitution of the population.[4–8] Deaths are most prevalent in young males participating in dynamic sports such as basketball and football, and are

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frequently due to cardiomyopathies and ion channel disorders.[9 10] Most of the knowledge pertaining to the incidence of exercise-related sudden cardiac death (SCD) and the prevalence of cardiac disease in athletes is derived from studies examining predominantly male athletes. Over the past two decades, female participation in high-level sport has increased substantially, with more women competing at elite levels[11]; however, cardiovascular health in female athletes is under-reported. Several studies have reported a considerably lower prevalence of SCD in female athletes compared with male athletes, with ratios varying from 3:1 to 10:1.[12–14] Autopsy studies show a lower incidence of structural disease in female athletes compared with male counterparts.[15] The prevalence of quiescent cardiovascular disease among living professional female athletes is unknown, possibly due to historically limited access to pre-participation screening programmes. Since 2000, the English Football Association (FA) has offered a cardiovascular screening programme for female football players in the UK. This study aims to report the outcomes of a mandatory pre-participation cardiac screening programme in female football players and compare them with their male counterparts.

METHODS

Screening programme

Between 1 January 2000 and 30 August 2024, a total of 3121 consecutive female football players underwent cardiac screening as part of the FA programme. This encompassed all female athletes across professional football clubs and their academies in England. All athletes trained competitively (minimum of 4 days per week). Written consent for screening was obtained from all athletes by the team doctor, with consent from a parent or guardian for athletes under 16 years of age, in accordance with principles of FA data governance.

During cardiovascular assessment, athletes underwent a cardiac health questionnaire, 12-lead ECG and echocardiogram. Accredited cardiac physiologists visited clubs to perform the echocardiograms, which were subsequently reviewed by an expert cardiologist, who was a member of the FA-approved cardiology advisory panel.

After evaluation, a formal report was delivered to the FA cardiac screening department, where each athlete was classified in one of three categories: (a) normal; (b) abnormality detected and further evaluation needed to confirm or refute the presence of cardiac disease; or (c) cardiac disease detected. Further investigations were performed at regional specialist centres, following the detection of an abnormality; this included athletes with potential cardiovascular symptoms reported on their health questionnaires, ECG abnormalities including a short PR interval (defined as ≤120 ms), a delta wave, left bundle branch block (LBBB) and corrected QT (QTc) interval abnormalities (defined as ≥480 ms for females and ≥470 ms for males), as well as abnormal T-wave inversion (TWI) and structural abnormalities detected on echocardiogram including abnormally enlarged cardiac dimensions and/or low ejection fraction (EF) or other uncommon echocardiographic features.[4 16 17] TWI was adjudicated per International ECG Criteria, accounting for age and ethnicity: anterior TWI confined to V_1–V_3 in non-Afro-Caribbean athletes <16 years and V_1–V_4 in Afro-Caribbean athletes was considered physiological; inferior TWI presenting in all three inferior leads and lateral TWI were considered abnormal.[18] Echocardiography was analysed in accordance with the standard protocols.[19] Ventricular wall thickness, cavity dimension measurements and identification of the origins of the left and right coronary arteries were made in accordance with established guidelines.[20] Further cardiovascular investigations included exercise stress testing, 24-hour Holter monitoring, cardiovascular MRI, electrophysiological studies or genetic testing depending on the clinical indication. Athletes with a prolonged QTc interval underwent exercise stress testing, prolonged ECG monitoring and genetic testing. Athletes with a short PR interval were evaluated for features of Wolff-Parkinson-White (WPW) pattern and referred for an exercise test and/or electrophysiological (EP) studies if clinically indicated. For analytical purposes, once a cardiac disorder was detected, it was further classified as either associated with SCD or as a minor cardiac condition. Both ECG and echocardiography were independently reviewed by the first author (KY) with retrospective application of the International Criteria for ECG Interpretation in Athletes[18] and the British Society of Echocardiography guidelines,[21] who was blinded to the outcomes. Equivocal ECG and echocardiography findings were discussed with the principal investigator (AM), who made the final adjudication. Additionally, to assess inter-observer reliability, a random sample of 312 ECGs (10% of the cohort) was independently analysed by two investigators (KY and RB). There was excellent inter-observer agreement for abnormal ECG findings (kappa value of 0.88).

Diagnosis

A priori list of cardiac conditions associated with SCD included any form of cardiomyopathy, myocarditis, previous myocardial insult with residual myocardial scar, intra-myocardial mass, anomalous origin of coronary artery, WPW syndrome, long QT syndrome (LQTS), any ventricular arrhythmia, channelopathy, Brugada syndrome, severe degree of valvular pathology and significant aortopathy.

Other conditions that are not listed above include congenital heart disease, such as atrial septal defect (ASD), small ventricular septal defect (VSD), patent ductus arteriosus (PDA), valvular heart disease, such as bicuspid aortic valve (BAV), more than mild degree of valvular pathologies, mild/moderate degree of aortopathy and supraventricular arrhythmia. These are classified as minor cardiac conditions that are not associated with SCD but require long-term surveillance.

Hypertrophic cardiomyopathy (HCM) was defined as a left ventricular (LV) wall thickness of ≥15 mm in one or more myocardial segments, which could not be solely explained by loading conditions (z-score >2 in adolescents).[22] A diagnosis of dilated cardiomyopathy (DCM) was considered if the athlete had a dilated LV (LV end-diastolic dimension >51 mm) and a reduced EF (<54%); further evaluation was also considered for those with a positive family history.[20 23] Arrhythmogenic cardiomyopathy (ACM) was diagnosed according to published criteria.[24] LQTS was based on a QTc interval ≥480 msec (for females) or ≥470 msec (for males), in association with low amplitude, notched or biphasic T-waves in at least three leads, paradoxical prolongation of the QT interval with exercise, and/or a positive genetic test.[25] A diagnosis of WPW pattern was made if there were characteristic ECG changes comprising a short PR interval (≤120 ms), prolonged QRS duration (>120 ms) and the presence of a delta wave (slurred QRS upstroke/downstroke).[16] Brugada syndrome was suspected with characteristic coved ST elevation in leads V_1–V_3.[16 26]

Follow-up

All athletes with abnormal ECG findings and/or echocardiogram findings underwent further evaluation, including cardiac

MRI, exercise testing and prolonged ECG monitoring, where applicable, and were monitored yearly. Athletes with congenital valvular or septal abnormalities were monitored with annual ECG and echocardiography. Cardiac MRIs were organised at the discretion of the attending cardiologist. Athletes who were diagnosed with cardiac disorders that are associated with SCD were evaluated and counselled about the potential risks of ongoing competitive sport in the presence of the next of kin and club doctor. Athletes who chose to continue to compete following a shared decision-making process were monitored on a 6-monthly basis, whereas those who decided to restrict vigorous exercise and competitive sport to mitigate their risk were discharged into the care of the National Health Service (NHS). Decisions regarding major cardiac diseases and the associated risks were made by the FA cardiology consensus panel in conjunction with the exercise recommendations of the European Society of Cardiology.[12] Follow-up duration was estimated using available FA screening programme records, club-level information and subsequent clinical correspondence where available. Cumulative person-years were calculated by summing the available follow-up time for each athlete. Because follow-up was based on available programme and club records rather than a dedicated longitudinal competition registry, follow-up duration should be interpreted as an estimate.

Outcomes

The follow-up period for each individual athlete was based on the number of years of competition within the FA, as determined from the FA registry of athletes. Athlete deaths were identified through a database compiled from reports to the FA as well as contacting each club individually. In addition, regular internet searches were conducted using three different search engines (Google, Bing and Yahoo) and the keywords: 'athlete', 'death', 'sudden', 'cardiac', 'arrest', 'collapsed', 'heart', 'attack', 'died', 'football', 'running', 'unknown', 'college', 'saved' and 'defibrillator'. Data on progression and survival status of athletes with diagnoses of diseases associated with SCD were obtained from the relevant regional cardiologists.

Comparison between sexes

To minimise temporal bias in sex-based comparisons, a secondary matched analysis was performed. Female football players screened between January 2017 and August 2024 were eligible for this analysis. Each female athlete was matched 1:2 with male football players who were screened through the same FA cardiac screening programme during the same period and were retrospectively analysed using the same protocol. A total of 2680 female athletes were matched to 5360 male athletes. Matching was performed by age (within ±1 year), ethnicity (exact matching) and screening period (same screening year). No replacement was allowed, and each male athlete was used only once. The matched analysis was used to compare sex-based differences in screening findings, referral rates, diagnostic yield, follow-up outcomes and estimated screening expenditure. The full female cohort remained the primary population used to describe cardiovascular screening outcomes in female football players.

Ethical approval

The protocol underwent internal governance review by the FA in accordance with its research governance procedures. The study used data collected through the Football Association's established cardiovascular screening programme, with athlete consent, data governance and data use overseen in accordance with Football Association legal and governance requirements. The age of informed consent in the UK is 16 years, with parental or guardian consent sought for those under this age.

Statistical analysis

Statistical analysis was performed using Microsoft Excel for Microsoft 365 and IBM SPSS Statistics Version 31.0.1.0 (IBM Corp., Armonk, NY, USA). Normal distribution of all continuous variables was examined using the Shapiro-Wilk test. The primary analysis described screening findings, diagnostic yield and follow-up outcomes in the full female cohort. For the contemporary matched analysis, female athletes screened between 2017 and 2024 were compared with their 1:2 matched male counterparts. Categorical outcomes in the matched cohort were compared using conditional logistic regression stratified by matched set. Continuous variables were compared using linear mixed-effects models with matched set as a random effect. Fisher's exact test was used for sparse descriptive comparisons where matched-set modelling was not appropriate. Continuous variables are presented as mean±SD. A two-sided p value <0.05 was considered statistically significant.

Patient and public involvement

Patients or the public were not involved in the design, conduct, reporting or dissemination plans of our research.

Equity, diversity, and inclusion

The author group consists of four women and 21 men, including junior, mid-career and senior researchers. Although the majority

Table 1 Baseline characteristics of the full female cohort and matched cohorts

	Full female cohort (n=3121)	Matched female cohort (n=2680)	Matched male cohort (n=5360)	P value*
Age, years	19.8±5.4	20.1±5.5	20.0±5.0	0.429
Age ≥16 years	2369 (75.9%)	2087 (77.9%)	4154 (77.5%)	0.705
BSA	1.72±0.13	1.72±0.12	1.90±0.18	<0.001
Ethnicity				
White	2717 (87.1%)	2313 (86.3%)	4626 (86.3%)	1.0
Black	124 (4.0%)	109 (4.1%)	218 (4.1%)	1.0
Mixed-race	220 (7.0%)	200 (7.5%)	400 (7.5%)	1.0
Asian	55 (1.8%)	53 (2.0%)	106 (2.0%)	1.0
Other	5 (0.2%)	5 (0.2%)	10 (0.2%)	1.0

*P value represents between female and male matched cohorts.
BSA, body surface area.

Table 2 Screening findings and diagnostic yield in the full female cohort

	N (%)	Major cardiac condition diagnosed	No major cardiac condition diagnosed
Positive health questionnaire	321 (10.3)	2	319
Abnormal ECG	77 (2.5)	4	73
Abnormal anterior TWI	41 (1.3)	0	41
Inferior TWI	10 (0.3)	0	10
Lateral TWI	9 (0.3)	0	9
Abnormal screen requiring further evaluation	193 (6.2)	6	187
Major cardiac conditions	6 (0.2)	–	–
Minor cardiac conditions requiring surveillance	36 (1.2)	–	–
Findings of uncertain significance requiring surveillance	73 (2.3)	–	–
Any athlete under surveillance	115 (3.7)		
SCA/SCD during follow-up	0 (0)		

TWI territories were not mutually exclusive.
ECG, electrocardiogram; SCA/SCD, sudden cardiac arrest/sudden cardiac death; TWI, T-wave inversion.

of members of the author group practise in one country, the author group has a diverse ethnic background. Our study population included all female football players who underwent a mandatory cardiovascular screening with matched male football players who underwent the same cardiovascular screening protocol. We included football players from different socioeconomic backgrounds. However, socioeconomic backgrounds with respect to cardiovascular impact were not assessed.

RESULTS

Study population

The mean age of the 3121 female football players was 19.8±5.4 years, and the majority of female athletes (n=2369, 75.9%) were aged ≥16 years. Most female athletes were of White ethnicity (87.1%), followed by mixed-race (7.0%), Black (4.0%) and Asian (1.8%). The average body surface area (BSA) of the female cohort was 1.72±0.13 m^2.

Of the full female cohort, 2680 athletes were screened between 2017 and 2024 and were included in the matched analysis against 5360 male football players. Baseline characteristics of the full female cohort, the matched female and the matched male cohorts are shown in table 1.

Symptoms and family history in female athletes

In the full female cohort, a positive health questionnaire, defined as symptoms and/or relevant family history, was present in 321 (10.3%) athletes. Referral was reserved for symptoms or family history considered clinically concerning after clinical review. One or more cardiac symptoms were reported in 284 (9.1%) athletes. Chest pain was the most commonly reported symptom in 127 (4.1%) athletes, followed by dizziness (n=114, 3.7%), palpitations (n=90, 2.9%), loss of consciousness (n=78, 2.5%) and dyspnoea (n=29, 0.9%). A family history of either premature coronary artery disease and/or cardiomyopathy or SCD was reported in 51 (1.6%) female athletes.

ECG characteristics in female athletes

The ECG characteristics in the full female cohort are summarised in online supplemental table 1.

Abnormal ECG findings were present in 77 (2.5%) female athletes. The most commonly present abnormal ECG finding was TWI. In total, 52 (1.7%) demonstrated abnormal TWI. Anterior TWI was present in 41 (1.3%) athletes, inferior TWI in ten (0.3%) and lateral TWI in nine (0.3%) athletes. Furthermore, a prolonged QTc interval was found in 15 (0.5%) and a pathological Q wave was present in six (0.2%) athletes. Less common abnormal ECG findings were LBBB (n=4), two or more ventricular ectopic beats (n=4) and atrio-ventricular dissociation (n=1).

Echocardiographic characteristics in female athletes

Echocardiographic parameters of the full female cohort are presented in online supplemental table 2. Five (0.2%) athletes showed maximal LV wall thickness of 12 mm; however, no

Table 3 Major cardiac conditions identified in the full female cohort

Disorder age (years) ethnicity	History and examination	ECG	Echocardiography result	Exercise test result	Genetic testing	Cardiac MRI	Medication	Outcome
Suspected LQTS 13 years White	None	QTc, 485–500 ms on multiple ECGs	Normal	No paradoxical increase in QTc	KCNH2 gene variant of unknown significance	N/A	None (after discussion with EP specialist)	Continued to play after a shared decision
LQTS (type 2) 29 years White	Dizziness, palpitations	QTc, 480 ms	Normal	QTc prolonged to 502 ms during 4th min of recovery	KCNH2 gene mutation	Normal	Beta-blocker offered (declined by the athlete)	Continued to play after a shared decision
LQTS (type 2) 26 years White	None	QTc, 482 ms	Normal	QTc prolonged to 490 ms during 4th min of recovery	No gene mutations identified	N/A	None (after discussion with EP specialist)	Continued to play after a shared decision
Suspected LQTS 31 years White	None	QTc, 488 ms	Normal	No increase in QTc during 4th min of recovery	KCNH2 gene variant of unknown significance	N/A	Beta-blocker	Continued to play after a shared decision
WPW syndrome 13 years White	Palpitations	Normal (intermittent pre-excitation on prolonged monitoring)	Normal	N/A	N/A	N/A	–	Underwent ablation before returning to play
BAV with moderate to severe AR 20 years White	Murmur	Normal	Fusion of right and left cusps with moderate to severe AR	No arrhythmia and normal BP/HR response	N/A	Moderate to severe AR. Normal LV volume. No myocardial fibrosis	–	Annual surveillance

AR, aortic regurgitation; BAV, bicuspid aortic valve; BP, blood pressure; ECG, electrocardiogram; EP, electrophysiology; HR, heart rate; LQTS, long-QT syndrome; LV, left ventricle; MRI, magnetic resonance imaging; N/A, not applicable; QTc, corrected QT interval; WPW, Wolff-Parkinson-White.

female athlete demonstrated LV wall thickness >12 mm. Increased LV end-diastolic diameter >51 mm was present in 339 (10.9%) athletes, of whom seven (2.1%) had low-normal LV ejection fraction. Increased LV cavity size was interpreted in the context of indexed dimensions, global systolic function, and overall athletic remodelling and biventricular proportionality, and most cases were considered physiological. No female athlete demonstrated an LV ejection fraction <50%. Right ventricular (RV) dilatation by increased basal diameter was present in 33 (1.1%) athletes.

Subsequent evaluation after initial screening in female athletes

Retrospective application of the International Criteria did not identify any additional female athlete who would have required ECG-based further evaluation but had not been referred following the original screening assessment. Following initial screening, 193 (6.2%) female athletes were referred for further evaluation. A summary of screening findings and diagnostic yield in the full female cohort is presented in table 2. The most common referral indications were abnormal TWI in 52 (1.7%) athletes, clinically concerning symptoms or family history requiring further assessment in 51 (1.6%) and short PR interval or QTc prolongation in 38 (1.2%). Other indications included congenital structural abnormalities detected on echocardiogram such as ASD (n=12), suspected BAV (n=8), suspected aortopathy (n=5) and PDA (n=3). Four (0.1%) athletes whose echocardiogram was inconclusive for coronary origins were referred for further evaluation. LV structural abnormalities on echocardiogram, such as disproportionate ventricular dilatation in four (0.1%) and reduced LV ejection fraction in one (0.03%) athlete, were also referred for further evaluation. Additionally, less frequent indications accounted for the remaining referrals.

Major cardiac conditions in female athletes

Major cardiac conditions associated with SCA/SCD were diagnosed in six (0.2%) female athletes (table 3 and figure 1), including four athletes with LQTS, one athlete with WPW syndrome and one athlete with BAV with moderate to severe aortic regurgitation. One female with a normal screening ECG underwent a prolonged ECG monitoring due to concerning symptoms, which revealed episodes of supraventricular tachycardia and an intermittently present accessory pathway. The athlete underwent an electrophysiology study and was successfully ablated. Among the four (0.1%) female athletes diagnosed with LQTS, the baseline ECGs revealed QTc values ranging from 480 to 500 ms. All cases were further evaluated by electrophysiologists. Two athletes were advised to commence a beta-blocker. They were all deemed low risk for fatal arrhythmia and were able to continue to participate in competitive sports. One (0.03%) female player

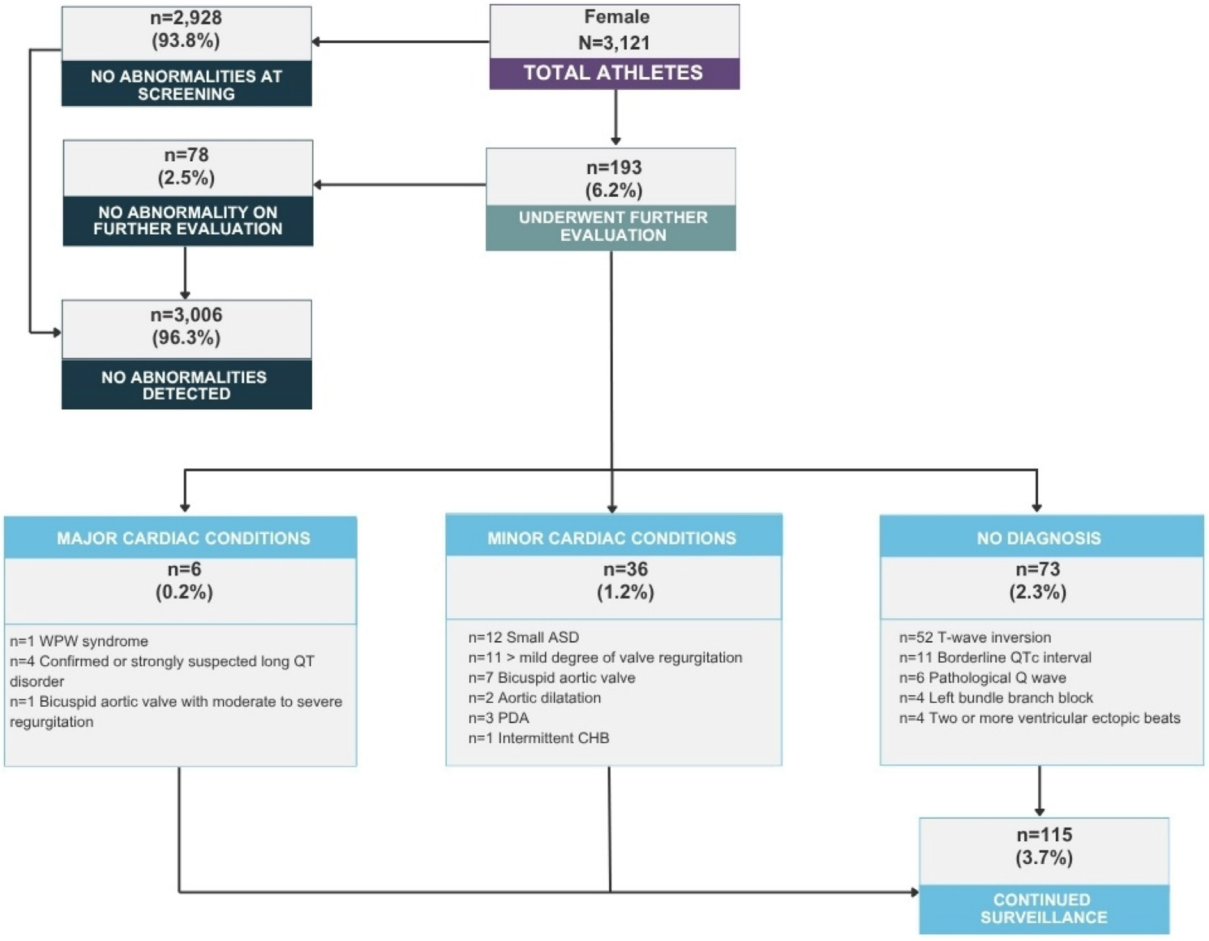

Figure 1 Outcomes of cardiovascular screening in female football players. These categories were not mutually exclusive; four athletes demonstrated both abnormal TWI and borderline QTc prolongation. ASD, atrial septal defect; CHB, complete heart block; PDA, patent ductus arteriosus; QTc, corrected QT interval; TWI, T-wave inversion; WPW, Wolff-Parkinson-White.

was diagnosed with BAV and moderate to severe aortic regurgitation on echocardiography, which was confirmed on cardiac MRI. An exercise stress test showed good functional capacity without arrhythmias or haemodynamic consequences. Cardiac MRI revealed a normal LV volume with normal systolic function and there was no evidence of myocardial fibrosis. Therefore, the player continued to compete and is under regular surveillance.

Minor cardiac conditions and surveillance findings in female athletes

Minor cardiac conditions requiring surveillance were identified in 36 (1.2%) females. These included 12 (0.4%) athletes with small ASD without evidence of RV overload or pulmonary hypertension. Three (0.1%) were diagnosed with PDA which required follow-up. 18 (0.6%) were diagnosed with valvular pathologies, including seven (0.2%) cases of BAV without dilatation of the aorta and 11 (0.4%) cases with more than mild valvular regurgitation, all of which required annual surveillance. One (0.03%) athlete demonstrated intermittent complete heart block. Additionally, two (0.1%) athletes were diagnosed with dilatation of the aortic root and/or ascending aorta requiring annual surveillance. There were two other athletes with suspected aortic dilatation on echocardiogram; however, on further cardiac imaging with cardiac MRI, aortic diameters were confirmed to be within normal range.

Findings of uncertain significance requiring annual surveillance were present in 73 (2.3%) female athletes, including 52 (1.7%) athletes with pathological T-wave abnormalities, 11 (0.4%) athletes with borderline QT prolongation, six (0.2%) with pathological Q waves, four (0.1%) with LBBB and four (0.1%) with two or more ventricular ectopic beats. These categories were not mutually exclusive; four athletes demonstrated both abnormal TWI and borderline QTc prolongation. Overall, 115 (3.7%) female athletes were placed under surveillance, including athletes with major cardiac conditions, minor cardiac conditions or findings of uncertain significance.

Sensitivity of history, ECG and echocardiogram in female athletes

Electrical diseases accounted for five (83%) female cases with cardiac conditions associated with SCD. Five (11.9%) of the total 42 female athletes diagnosed with any cardiac disorders reported one or more cardiac symptoms. The ECG had the highest sensitivity (66.7%) as a marker of major cardiac disease, while abnormal medical history had a sensitivity of 33.3%. The echocardiogram had the lowest sensitivity (16.7%).

Events during follow-up

The mean follow-up period for SCD in our female cohort was 6.2±4.3 years, corresponding to 19 193 person years. No SCA/SCD occurred among female athletes during follow-up. One female athlete was diagnosed with ACM 18 months after initial screening. This athlete demonstrated a normal resting ECG and echocardiogram at baseline, but on routine pre-participation exercise testing performed elsewhere there was a high burden of ventricular ectopy. A subsequent cardiac MRI confirmed a diagnosis of ACM.

Estimated screening expenditure in female athletes

Based on the UK NHS tariffs 2017/2018 and a similar cost analysis study from the UK,[4 27] the cost of preliminary investigation was £255 per athlete, comprising consultation (£157), ECG (£25) and echocardiography (£73). For the full female cohort, the estimated initial screening cost was £795 855. The cost of further investigating 193 female athletes was £63 886, with a total estimated outlay of £859 741. The cost to detect major cardiac condition in six female athletes was £143 290 per case, and the cost to identify any cardiac disorder among 42 female athletes was £20 470 per case.

Screening outcome comparison with male football players

In the matched analysis, 2680 female athletes screened between 2017 and 2024 were matched to 5360 male athletes screened during the same period. Matching achieved close balance for age, ethnicity and screening period (table 1). Screening findings, diagnostic yield, follow-up events and estimated screening expenditure per major diagnosis in the matched female and male cohorts are summarised in table 4.

Compared with matched male athletes, female athletes reported potential cardiac symptoms more frequently (259/2680 (9.7%) vs 254/5360 (4.7%); p<0.001). Positive family history of cardiac disease did not differ materially between groups (47/2680 (1.8%) vs 109/5360 (2.0%); p=0.39).

Clinically relevant ECG differences were concentrated in the repolarisation pattern and QTc interval. Abnormal anterior TWI was more frequent in female athletes than male athletes, but the difference was modest and did not reach statistical significance (32/2680 (1.2%) vs 46/5360 (0.9%); p=0.15). In contrast, inferior TWI was less frequent in females (6/2680 (0.2%) vs 69/5360 (1.3%); p<0.001). Although lateral TWI was uncommon in both groups, it was less frequent in female athletes (5/2680 (0.2%) vs 53/5360 (1.0%); p<0.001). Female athletes had a longer mean QTc interval than males (418.1±21.9 ms vs 405.7±23.0 ms; mean difference 12.4 ms (95% CI 11.4 to 13.4); p<0.001), although QTc prolongation meeting referral criteria was uncommon in both groups (12/2680 (0.4%) vs 25/5360 (0.5%); p=0.91). The higher mean QTc interval in females should be interpreted in the context of sex-specific diagnostic thresholds for QTc prolongation.

Referral for further evaluation occurred in 139 (5.2%) female athletes and 358 (6.7%) male athletes (p=0.008). Major cardiac conditions associated with SCA/SCD were diagnosed in five females (0.2%) and 29 males (0.5%; p=0.027). While LQTS was the most common condition diagnosed among females with major cardiac conditions, cardiomyopathy was the most

Table 4 Matched cohort comparison between females and males

	Matched females (n=2680)	Matched males (n=5360)	P value
Positive health questionnaire	292 (10.9%)	356 (6.6%)	<0.001
Any cardiac symptom	259 (9.7%)	254 (4.7%)	<0.001
Abnormal ECG	60 (2.2%)	167 (3.1%)	0.023
Abnormal TWI	38 (1.4%)	126 (2.4%)	0.004
Anterior TWI	32 (1.2%)	46 (0.9%)	0.154
Inferior TWI	6 (0.2%)	69 (1.3%)	<0.001
Lateral TWI	5 (0.2%)	53 (1.0%)	<0.001
Prolonged QTc interval	12 (0.4%)	25 (0.5%)	0.906
Referral for further evaluation	139 (5.2%)	358 (6.7%)	0.008
Major cardiac condition	5 (0.2%)	29 (0.5%)	0.027
Minor cardiac condition	30 (1.1%)	100 (1.9%)	0.010
SCA/SCD during follow-up	0 (0%)	3 (0.06%)	0.555
Cost per major diagnosis	£146 809	£51 888	–

TWI territories were not mutually exclusive.
QTc, corrected QT interval; SCA/SCD, sudden cardiac arrest/sudden cardiac death; TWI, T-wave inversion.

common diagnosis among males. No cardiomyopathy was diagnosed at baseline in females, compared with 10 cases in males. Minor cardiac conditions requiring surveillance were detected in 30 (1.1%) females and 100 (1.9%) males (p=0.010).

During follow-up, no SCA/SCD events occurred in this female subgroup and three events occurred in matched males, with two athletes successfully resuscitated. The estimated screening expenditure per major cardiac diagnosis was £146 809 in females and £51 888 in males. Estimated expenditure per diagnosis of any cardiac disorder was £20 972 in females and £11 665 in males.

DISCUSSION

This study provides one of the largest evaluations of cardiovascular screening outcomes in elite female football players in the UK. The principal findings were that major cardiac conditions associated with SCA/SCD were uncommon in female athletes, were less frequently diagnosed than in matched male athletes and were predominantly electrical rather than structural. No female athlete was diagnosed with cardiomyopathy at baseline screening, although one athlete was diagnosed with ACM during follow-up. Female athletes reported symptoms more frequently than males, but symptom reporting had a low positive diagnostic yield. In contrast, the ECG remained the most informative component of screening for major disease, particularly for QTc prolongation and abnormal repolarisation. These findings support that cardiovascular screening in female athletes should not be viewed simply as a lower-yield version of male athlete screening. Rather, female athletes appear to present a distinct screening profile requiring sex-appropriate interpretation, downstream evaluation and surveillance planning.

Female athletes reported more symptoms compared with males, but few symptomatic athletes were ultimately diagnosed with major cardiac disease. The higher symptom-reporting rate in females may reflect sex-related differences in symptom perception, reporting behaviour or questionnaire disclosure, although this cannot be determined from the present dataset. Historically, males are more likely to under-report symptoms, which may reflect a Western cultural attitude shown among male athletes and non-athletes alike.[28 29] Importantly, symptom reporting should not be dismissed, because two athletes with major cardiac conditions had a positive health questionnaire. Rather, these findings indicate that symptoms should be interpreted in a structured clinical context. As noted previously, the ECG was the most sensitive tool for detecting major pathology and major disease.[30 31]

The findings on TWI are particularly relevant to sex-specific ECG interpretation. Anterior TWI was numerically more frequent in female athletes than males, but the difference was modest and did not reach statistical significance. No female athlete with anterior TWI was diagnosed with structural heart disease after downstream evaluation. This supports the view that anterior TWI in female athletes, particularly when isolated and not accompanied by other concerning clinical features, may have a lower diagnostic yield for cardiomyopathy than in males.[32–34] By contrast, although lateral TWI was uncommon in both sexes, it was significantly less frequent in female athletes than male athletes. Lateral TWI should therefore remain a high-priority abnormality because of its established association with cardiomyopathy and other structural disease.[35 36] Inferior TWI was less common in females and was not associated with major disease in this cohort. Overall, these findings support the ongoing refinement of sex-specific ECG interpretation criteria in athletes.

The absence of HCM at baseline is one of the most striking observations. In more than 3000 female football players, only five athletes had a maximal LV wall thickness of 12 mm, none had LV wall thickness >12 mm and no athlete fulfilled diagnostic criteria for HCM. This supports previous observations that in female athletes, LV wall thickness ≥12 mm should be regarded as uncommon and should prompt further evaluation, particularly when accompanied by abnormal ECG findings, symptoms, family history, disproportionate cavity size, impaired function or abnormal CMR findings.[37 38] This study could reflect a genuinely low prevalence of overt HCM in elite female football players, but several alternative explanations should be considered. Age-related penetrance may also be relevant, as some athletes may develop diagnostic features later in adulthood.[39–45] However, age and sample size may not fully explain the absence of baseline HCM. Selection into elite football pathways may remove some athletes with early manifestations of cardiac conditions.

The diagnosis of ACM in one female athlete 18 months after a normal baseline resting ECG and echocardiogram further emphasises that screening is a time-specific assessment. Some cardiomyopathies have age-dependent or evolving expression and may not be detectable at a single screening encounter. This supports the need for longitudinal vigilance, repeat assessment when symptoms or new electrical abnormalities emerge and robust secondary prevention systems within clubs.

The lower diagnostic yield in female athletes has important resource implications. The estimated cost per major diagnosis was higher in females than males, largely because fewer major conditions were detected despite a lower referral rate. This should not be interpreted as evidence against screening female athletes. Instead, it provides data to support realistic planning as female professional football expands. Screening programmes should anticipate a lower prevalence of major disease, a predominance of electrical diagnoses, a relatively high burden of symptoms requiring clinical review and a group of athletes requiring surveillance for minor conditions or findings of uncertain significance. Screening expenditure may also vary across demographic subgroups. Age, ethnicity and ancestral origin can influence the prevalence of ECG findings such as TWI and other repolarisation patterns, which in turn may affect referral rates, false-positive findings and downstream testing. Although the matched analysis accounted for age and ethnicity, this study was not designed to estimate subgroup-specific false-positive rates or cost per diagnosis. Future studies should evaluate screening yield and downstream resource use across sex, age and ethnicity/origin groups.

Clinical implications

These findings suggest that screening pathways should be calibrated to the female athlete population rather than extrapolated from male cohorts. There should be a heightened awareness of electrical conditions among females. Female athletes report symptoms more frequently. Anterior TWI should be interpreted in context, particularly when isolated and unsupported by symptoms, additional ECG abnormalities or structural abnormalities. Echocardiographic interpretation should use sex-specific structural thresholds, with further evaluation considered in female athletes with LV wall thickness ≥12 mm, particularly when accompanied by abnormal ECG findings, symptoms, family history, disproportionate cavity size, impaired function or abnormal CMR findings. Finally, female athletes manifest a lower prevalence of diseases implicated in SCA/SCD. Consequently, the cost per case is nearly three times that of male

athletes and this should be accounted for when budgeting for cardiac screening programmes.

Limitations

This study has several limitations. The cohort was restricted to football players within one national screening programme, and findings may not be generalisable to other sports, countries or levels of competition. Although this is a large cohort of female athletes, major cardiac conditions were rare. The study was therefore underpowered to make definitive conclusions about sex-based differences in individual disease prevalence, disease subtype distribution or SCA/SCD outcomes. Diagnostic pathways and access to downstream investigations, particularly cardiac MRI, may have evolved over the 24-year study period. While the vast majority of the female cohort (86%) was screened from 2017 onwards, there may have been a more variable interpretation of results prior to 2017. Athletes screened before 2017 were more likely to undergo further investigation than those screened from 2017 onwards, likely reflecting historical differences in ECG interpretation criteria and referral thresholds.[46] Although downstream testing was performed according to clinical indication and all sex-based comparisons were restricted to a contemporary cohort matched by age, ethnicity and screening year, residual temporal variation in diagnostic practice cannot be fully excluded.

Despite internet searches and correspondence with FA-registered clubs, we may not have identified all cases of SCA/SCD in the absence of a mandatory register for SCD. Additionally, it may be possible that athletes who were initially screened may have developed quiescent cardiomyopathies, not identified in the following years.

Finally, the expenditure analysis was based on estimated UK tariffs and should not be interpreted as a formal cost-effectiveness analysis. It did not model downstream clinical outcomes, quality-adjusted life-years, emergency preparedness, staffing or administrative costs or the long-term costs of surveillance.

CONCLUSION

In this large cohort of elite female football players, cardiovascular screening identified a low prevalence of major cardiac conditions, with diagnoses predominantly comprising electrical disorders and no cardiomyopathy detected at baseline. Compared with matched male athletes, female athletes had fewer major diagnoses, more frequent symptom reporting, a different pattern of repolarisation abnormalities and higher estimated screening expenditure per major diagnosis. These findings support sex-appropriate interpretation of cardiac findings, structured surveillance of uncertain abnormalities and realistic resource planning as female professional football continues to expand.

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Patient consent for publication Not applicable.

Ethics approval This study involves human participants but was not approved by Reference: The Football Association advisory panel meeting 29/11/2023. This study involved retrospective analysis of anonymised ECG data collected as part of The Football Association cardiovascular screening programme. Participants provided informed consent for cardiovascular screening and use of anonymised data for research purposes. The study involved no additional investigations or interventions beyond routine clinical screening practice and was conducted under The Football Association governance framework. Formal Research Ethics Committee or Institutional Review Board approval was not sought as the work constituted secondary analysis of routinely collected anonymised clinical data. Participants gave informed consent to participate in the study before taking part.

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REFERENCES

1 Lee I-M, Shiroma EJ, Lobelo F, *et al*. Effect of physical inactivity on major non-communicable diseases worldwide: an analysis of burden of disease and life expectancy. *Lancet* 2012;380:219–29.
2 Reiner M, Niermann C, Jekauc D, *et al*. Long-term health benefits of physical activity--a systematic review of longitudinal studies. *BMC Public Health* 2013;13:813.
3 Thompson PD, Franklin BA, Balady GJ, *et al*. Exercise and Acute Cardiovascular Events. *Circulation* 2007;115:2358–68.

4 Malhotra A, Dhutia H, Finocchiaro G, *et al*. Outcomes of Cardiac Screening in Adolescent Soccer Players. *N Engl J Med* 2018;379:524–34.
5 Maron BJ, Doerer JJ, Haas TS, *et al*. Sudden Deaths in Young Competitive Athletes. *Circulation* 2009;119:1085–92.
6 Harmon KG, Asif IM, Klossner D, *et al*. Incidence of sudden cardiac death in National Collegiate Athletic Association athletes. *Circulation* 2011;123:1594–600.
7 Maron BJ, Gohman TE, Aeppli D. Prevalence of sudden cardiac death during competitive sports activities in Minnesota high school athletes. *J Am Coll Cardiol* 1998;32:1881–4.
8 Asif IM, Harmon KG. Incidence and Etiology of Sudden Cardiac Death: New Updates for Athletic Departments. *Sports Health* 2017;9:268–79.
9 Petek BJ, Churchill TW, Moulson N, *et al*. Survival Outcomes After Sudden Cardiac Arrest in Young Competitive Athletes From the United States. *J Am Coll Cardiol* 2025;85:1682–92.
10 Petek BJ, Churchill TW, Moulson N, *et al*. Sudden Cardiac Death in National Collegiate Athletic Association Athletes: A 20-Year Study. *Circulation* 2024;149:80–90.
11 Finocchiaro G, Westaby J, Sheppard MN, *et al*. Sudden Cardiac Death in Young Athletes. *J Am Coll Cardiol* 2024;83:350–70.
12 Pelliccia A, Sharma S, Gati S, *et al*. 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease: the Task Force on sports cardiology and exercise in patients with cardiovascular disease of the European Society of Cardiology (ESC). *Eur Heart J* 2021;42:17–96.
13 Li L, Le Douairon Lahaye S, Ding S, *et al*. Sex Differences in the Incidence of Sudden Cardiac Arrest/Death in Competitive Athletes: A Systematic Review and Meta-analysis. *Sports Med* 2025;55:697–712.
14 Han J, Lalario A, Merro E, *et al*. Sudden Cardiac Death in Athletes: Facts and Fallacies. *J Cardiovasc Dev Dis* 2023;10:68.
15 Finocchiaro G, Westaby J, Bhatia R, *et al*. Sudden Death in Female Athletes: Insights From a Large Regional Registry in the United Kingdom. *Circulation* 2021;144:1827–9.
16 Chandra N, Bastiaenen R, Papadakis M, *et al*. Sudden cardiac death in young athletes: practical challenges and diagnostic dilemmas. *J Am Coll Cardiol* 2013;61:1027–40.
17 Sharma S, Merghani A, Mont L. Exercise and the heart: the good, the bad, and the ugly. *Eur Heart J* 2015;36:1445–53.
18 Drezner JA, Sharma S, Baggish A, *et al*. International criteria for electrocardiographic interpretation in athletes: Consensus statement. *Br J Sports Med* 2017;51:704–31.
19 Oxborough D, ZaidiA, Gati S, *et al*. A guideline update for the practice of echocardiography in the cardiac screening of sports participants: a joint policy statement from the British Society of Echocardiography and Cardiac Risk in the Young. *Echo Res Pract* 2018;5:G1–10.
20 Harkness A, Ring L, Augustine DX, *et al*. Normal Reference Intervals for Cardiac Dimensions and Function for Use in Echocardiographic Practice: A Guideline from the British Society of Echocardiography. *Echo Res Pract* 2020;7:G1–18.
21 Oxborough D, Augustine D, Gati S, *et al*. A guideline update for the practice of echocardiography in the cardiac screening of sports participants: a joint policy statement from the British Society of Echocardiography and Cardiac Risk in the Young. *Echo Res Pract* 2018;5:G1–10.
22 Elliott PM, Anastasakis A, Borger MA, *et al*. 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy: the Task Force for the Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC). *Eur Heart J* 2014;35:2733–79.
23 Weintraub RG, Semsarian C, Macdonald P. Dilated cardiomyopathy. *Lancet* 2017;390:400–14.
24 Marcus FI, McKenna WJ, Sherrill D, *et al*. Diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia: proposed modification of the Task Force Criteria. *Eur Heart J* 2010;31:806–14.
25 Schwartz PJ, Ackerman MJ. The long QT syndrome: a transatlantic clinical approach to diagnosis and therapy. *Eur Heart J* 2013;34:3109–16.
26 Anselm DD, Baranchuk A. Brugada phenocopy: redefinition and updated classification. *Am J Cardiol* 2013;111:453.
27 Dhutia H, Malhotra A, Gabus V, *et al*. Cost Implications of Using Different ECG Criteria for Screening Young Athletes in the United Kingdom. *J Am Coll Cardiol* 2016;68:702–11.
28 Wang Y, Hunt K, Nazareth I, *et al*. Do men consult less than women? An analysis of routinely collected UK general practice data. *BMJ Open* 2013;3:e003320.
29 Kroenke K, Spitzer RL. Gender Differences in the Reporting of Physical and Somatoform Symptoms. *Psychosom Med* 1998;60:150–5.
30 Williams EA, Pelto HF, Toresdahl BG, *et al*. Performance of the American Heart Association (AHA) 14-Point Evaluation Versus Electrocardiography for the Cardiovascular Screening of High School Athletes: A Prospective Study. *J Am Heart Assoc* 2019;8:e012235.
31 Harmon KG, Suchsland MZ, Prutkin JM, *et al*. Comparison of cardiovascular screening in college athletes by history and physical examination with and without an electrocardiogram: Efficacy and cost. *Heart Rhythm* 2020;17:1649–55.
32 Malhotra A, Dhutia H, Gati S, *et al*. Anterior T-Wave Inversion in Young White Athletes and Nonathletes: Prevalence and Significance. *J Am Coll Cardiol* 2017;69:1–9.
33 Petek BJ, Wasfy MM. Cardiac Adaption to Exercise Training: the Female Athlete. *Curr Treat Options Cardiovasc Med* 2018;20:68.
34 Orchard JJ, Drezner JA, Raju H, *et al*. Isolated Anterior T-Wave Inversion in Elite Athletes: Prevalence and Clinical Relevance by Sex and Sporting Discipline. *JAHA* 2025;14.
35 Papadakis M, Basavarajaiah S, Rawlins J, *et al*. Prevalence and significance of T-wave inversions in predominantly Caucasian adolescent athletes. *Eur Heart J* 2009;30:1728–35.
36 Palermi S, Tardini L, Graziano F, *et al*. Interpretation and management of T wave inversion in athletes: An expert opinion statement of the Italian Society of Sports Cardiology (SICSPORT). *Int J Cardiol* 2025;422:132968.
37 Pelliccia A, Maron BJ, Culasso F, *et al*. Athlete's heart in women. Echocardiographic characterization of highly trained elite female athletes. *JAMA* 1996;276:211–5.
38 D'Ascenzi F, Biella F, Lemme E, *et al*. Female Athlete's Heart: Sex Effects on Electrical and Structural Remodeling. *Circ Cardiovasc Imaging* 2020;13:e011587.
39 Maron BJ, Douglas PS, Graham TP, *et al*. Task Force 1: preparticipation screening and diagnosis of cardiovascular disease in athletes. *J Am Coll Cardiol* 2005;45:1322–6.
40 Maron BJ, Olivotto I, Spirito P, *et al*. Epidemiology of Hypertrophic Cardiomyopathy–Related Death. *Circulation* 2000;102:858–64.
41 Maron BJ. Hypertrophic cardiomyopathy: a systematic review. *JAMA* 2002;287:1308–20.
42 Niimura H, Bachinski LL, Sangwatanaroj S, *et al*. Mutations in the gene for cardiac myosin-binding protein C and late-onset familial hypertrophic cardiomyopathy. *N Engl J Med* 1998;338:1248–57.
43 Maron BJ, Niimura H, Casey SA, *et al*. Development of left ventricular hypertrophy in adults in hypertrophic cardiomyopathy caused by cardiac myosin-binding protein C gene mutations. *J Am Coll Cardiol* 2001;38:315–21.
44 Maron BJ, Spirito P, Wesley Y, *et al*. Development and progression of left ventricular hypertrophy in children with hypertrophic cardiomyopathy. *N Engl J Med* 1986;315:610–4.
45 Maron BJ, Haas TS, Ahluwalia A, *et al*. Incidence of cardiovascular sudden deaths in Minnesota high school athletes. *Heart Rhythm* 2013;10:374–7.
46 Malhotra A, Dhutia H, Yeo T-J, *et al*. Accuracy of the 2017 international recommendations for clinicians who interpret adolescent athletes' ECGs: a cohort study of 11 168 British white and black soccer players. *Br J Sports Med* 2020;54:739–45.