Journal of Physical Education and Sport ® (JPES), Vol. 24 (issue 4), Art 118, pp. 1032 - 1039, April 2024
online ISSN: 2247 - 806X; p-ISSN: 2247 – 8051; ISSN - L = 2247 - 8051 © JPES

Original Article

Athlete´s Heart vs. hypertrophic cardiomyopathy and markers of myocardial necrosis in football players

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Published online: April 30, 2024
(Accepted for publication April 15, 2024
DOI:10.7752/jpes.2024.04118

Abstract:
High-level athletes, such as football players, undergo prolonged and intense training regimens that can lead to adaptive structural and functional changes in the heart. Athlete's heart syndrome is perhaps the most well-known of these alterations exhibiting adaptive responses such as left ventricular hypertrophy, considered benign and without intervention. However, there can be overlap between the adaptations observed in athlete's heart and pathological changes like hypertrophic cardiomyopathy, making it challenging to differentiate between the two. It is important to emphasize that football is characterized by its intermittent nature, utilizing aerobic metabolism, but also involving high-intensity actions that place significant strain on anaerobic metabolism. The present study aims to analyze cardiac structural and functional changes in high-performance football players (after finishing the competitive period and in a pre-competitive rest period) and to compare them with the patients with hypertrophic cardiomyopathy with implantable cardioverter defibrillators (N = 10). We analyzed seven elite football players who competed in the professional Portuguese league. In addition to heart rate and blood pressure measurements, data from electrocardiograms, echocardiograms, and blood samples were collected to assess biomarkers indicative of necrosis or myocardial stress, including high-sensitivity Troponin I. The results suggest that high-performance football players during the competitive phase exhibited elevated markers (non-pathological values) of myocardial necrosis, possibly due to intense physical exertion. However, the elevation of these biomarkers is significantly higher in patients with hypertrophic cardiomyopathy. The measurement of these biomarkers may aid in distinguishing between 'athlete's heart' and hypertrophic cardiomyopathy, particularly in challenging cases of differential diagnosis.
Key Words: **- athlete; cardiac; hypertrophic myocardiopathy; necrosis; troponin I; football**

Introduction

Football is an intermittent sport that mainly uses aerobic metabolism, interspersed with high-intensity actions that put a huge strain on anaerobic metabolism (Barba, F., et al.,2020; Papancrová, A., et al., 2024). The typical distance travelled by a high-level male field player during a match is 10-13 km (Di Mascio & Bradley, 2013). However, most of the distance is covered by walking and low-intensity running, and it is mainly the periods of high-intensity exercise that are most important. The amount of high-speed running is what distinguishes elite players from those who play at a lower level. Computerised time-motion analysis has shown that players perform 28% more high-intensity runs (2.43 vs. 1.90 km) and 58% more sprints (650 vs. 410 m) than professional players who are at a lower level (Mohr et al., 2003). Ingebrigtsen et al. (2012) found that the top teams in the Danish league performed 30-40% more distance running at high speed compared to teams in the middle or bottom of the league table (Ingebrigtsen et al., 2012).

In summary, it seems safe to conclude that the elite footballer has to be able to perform repeated high-intensity exercise and also that numerous factors influence the distance travelled in a game, and these can include physical ability, technical qualities, position in the game, their tactical role, the style of play, the team's possession of the ball, the quality of the opponent, the importance of the game, the seasonal period, as well as environmental factors (Bangsbo, 2014).

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Intermittent exercise, such as football, involves strong muscle contractions. In particular, concentric propulsive forces during the acceleration phase, and eccentric braking forces during the deceleration phase, are highly associated with skeletal muscle damage and the inflammatory response (Ispirlidis et al., 2008).
The study by Aslan, A., et al (2019), revealed that, even when practised recreationally by players, their practice induced increases in biomarkers in relation to short-term damage in cardiac and skeletal muscle, as well as in the inflammatory profile, this in young and middle-aged participants (Aslan et al., 2019).
Football players are usually selected for their most advantageous anthropometric characteristics, which will allow them to compete at the highest level of performance, and this is a requirement for football (Stølen et al., 2005).

There is an age-dependent prevalence of the main pathologies underlying sudden death in athletes. The main causes in young athletes (<35 years old) include hypertrophic cardiomyopathy (HCM), congenital anomalies of the coronary artery and arrhythmogenic right ventricular cardiomyopathy (ARVC) (Mavrogeni et al., 2019). It is therefore important to distinguish between athlete's heart, a physiological condition, and hypertrophic cardiomyopathy, a pathological condition.

The athlete's heart is normally characterised by LV *remodelling*, with increased parietal thickness and cavity dimensions coexisting with preservation and improvement of systolic and diastolic function (Cabanelas et al., 2013). Exposure to the ever-increasing demands of high-performance sport can turn these physiological conditions into pathological ones (myocardial fibrosis (Małek & Bucciarelli-Ducci, 2020), heart failure (Eijsvogels & Aengevaeren, 2023), changes in heart rhythm (Carbone et al., 2017)). Differentiating between these alterations, particularly hypertrophic cardiomyopathy (HCM), is of real importance for clinicians (Vilades et al., 2021).

The study by Perry, R. et al (2019), carried out with thirty-six *Australian Football League* players with 3.5 ± 2.7 years of professional training at the time of enrolment, prospectively underwent echocardiography in the 2009, 2013 and 2015 pre-seasons. In these players, there were increases in the size of the left ventricle (LV) and right ventricle (RV) and in LV mass. Players over 25 years of age showed a trend towards an increase in RV size and a decline in RV global longitudinal strain in their initial examinations. Fitness level and playing position also affected the degree of physiological athletic cardiac remodelling (Perry et al., 2019).

There are differences in the types of cardiac remodelling shown by athletes, depending on the type of exercise they do. For example, endurance athletes who have been exercising for several years show eccentric cardiac remodelling (Plews et al., 2013), while trained endurance athletes show concentric cardiac remodelling (Utomi et al., 2014). In addition to the type, level and intensity of the sporting activity practised, other factors can have an influence on cardiac changes, ranging from the level of competition, the intensity of the training, as well as intrinsic characteristics of the athlete themselves (CORÎCI et al., 2018).

Troponin is the biomarker of choice for detecting heart damage. It is a fact that high troponin levels are highly specific for heart damage (Babuin & Jaffe, 2005). In 2000, a joint committee of the European Society of Cardiology and the American College of Cardiology (ESC/ACC) issued new criteria which recognised that elevations in biomarkers were fundamental for the diagnosis of acute myocardial infarction (Antman et al., 2000) because symptoms can be atypical or non-existent and changes in the electrocardiogram can be absent or non-specific. In order to understand and quantify the extent of possible cardiac damage caused by intense/prolonged physical exercise or "chronic load", troponins, and more specifically troponin I, can be a valuable ally (Mahanty & Xi, 2020).

Hypertrophic cardiomyopathy (HCM) is a genetic disease, so relatives of affected people may be at risk. Cardiac troponin biomarkers have been shown to be elevated in HCM. Troponin I shows a positive and graded association with measures of cardiac muscle mass in people at risk of HCM, and when there is an increase in serum values, with possible heart damage (McGorrian et al., 2013). Exercise can also produce cardiac troponin elevations. Until now, exercise-induced troponin elevations have been seen as the only benign form of these biomarker elevations. However, recent studies report intriguing findings that shed new light on the underlying mechanisms and clinical relevance of exercise-induced troponin elevations. To what extent the release of troponins in athletes represents a physiological response and what the threshold is for it to become pathological is a question that remains a "grey" area (Aengevaeren et al., 2021).

The aim of our study was to analyse cardiac structural and functional changes in high-performance football players (after competition and at rest) and compare them with HCM patients with implantable cardioverter defibrillators.

Material & methods

Study design

A prospective observational study was carried out which included the cardiological assessment of footballers from the Portuguese 2nd league, aged between 19 and 30 years old, and a group of patients with HCM at high risk of sudden death and implantable cardioverter-defibrillators. The HCM patients were followed up at the Cardiology clinic of the Coimbra Hospital and University Centre.

Data was collected from the athletes in two stages. The first was carried out before the start of the championship, during the sports medical examinations, in an attempt to express a period without competition and with reduced training loads, known as "rest". The second was carried out at the end of the season, in the "window" of 48 hours following the end of the antepenultimate "matchday", known as "after the match".

All participants who took part in the study did so voluntarily and after obtaining informed consent. The study was approved by the Ethics Committee of the Faculty of Medicine of the University of Coimbra and by the National Data Protection Commission, under code 9325/2017.

Sample Characterization

The sample of football players consisted of 7 male players from a football team in Portugal's second professional football league. In general, these players train 5 days a week, twice a day, with each training session lasting an average of 1.5 hours, with the number of games per week varying between 1 and 2.

The athletes were compared with 10 MCH patients from the Cardiology Service of the Coimbra University Hospital Centre, all male, with implantable cardioverter-defibrillators (ICDs), and aged between 34 and 53.

Evaluations Performed

The participants underwent a physical examination and their medical history was taken, with the main focus on identifying cardiovascular risk factors. Their sporting history was also obtained, including years of practice and age. Cardiac screening began with the measurement of systolic blood pressure (SBP) and diastolic blood pressure (DBP) as well as heart rate (HR). Data was then also collected from a 12-lead electrocardiogram (ECG), transthoracic echocardiogram (TTE) and blood samples.

Electrocardiographic Evaluation

A 12-lead ECG was performed on all participants. The results were analysed by three cardiologists.

Echocardiographic Evaluation

Transthoracic echocardiograms were carried out by a cardiologist on all the participants using as a reference the recommendations of the consensus document and recommendations for Transthoracic Echocardiography in Portugal (two-dimensional, M-mode, colour Doppler, pulsed, continuous and tissue, myocardial deformation by Speckle-Tracking) (Evangelista et al., 2008). Measurements of the parietal thickness and dimensions of the left ventricle (LV) were observed in the parasternal long-axis window, relative wall thickness calculated by 2x LV posterior wall (LVPW)/ LV diastolic diameter (LVDD) (Jafary, 2007). LV volume was determined using the modified Simpson's rule, with images obtained in the apical window, four and two chambers. Left ventricular ejection fraction (LVEF) was obtained using the Simpson method. Tissue Doppler images of the mitral and tricuspid annuli were obtained to determine the E and E' waves and the S' wave velocities. In M mode, the tricuspid annular plane systolic excursion (TAPSE) was determined.

Blood sampling

One of the most important aspects of the research consisted of determining the individual release of troponin I in football players, comparing the 2 moments (rest and after the game) and comparing the levels of players after the game with the levels of patients with HCM.

Statistical Analysis

The values of the individual characteristics are presented as mean ± standard deviation.

Comparisons between the athlete's resting period and the post-match period were made using the paired Student's t-test or the Wilcoxon test. The paired parametric Student's t-test was used when the study populations followed normal distributions. Comparisons between athletes and HCM patients were made using the independent Student's t-test or the Mann-Whitney U-test. The independent parametric Student's t-test was used when the study populations followed normal distributions with equal variances. The Shapiro-Wilk test was used to test the normality of continuous variables. Levene's test was used to test the equality of population variances.

The Spearman or Pearson coefficients were used to assess the existence of correlation between pairs of variables. The analyses were carried out using IBM-SPSS software, version 25.0 (SPSS Inc., Chicago, Illinois). Statistical significance was established when the p-value was <0.05.

Results

Table 1 characterises the football players in terms of age, years of competition, hours of daily training and hours of sleep. The age of the players varies between 19 and 30 years, with an average of 22.6 ± 3.60 years, and the average number of years competing is 14.3 ± 2.87 years (minimum=10 and maximum=18 years).

Table 1 - Characterisation of the sample of football players for the variables: age and years of competition.

Characteristics	Min.	Max.	Mean	SD
Age (years)	19	30	22,6	3,60
Years competition	10	18	14,3	2,87

Min.=minimum; Max.=maximum; SD=standard deviation

The primary results in Table 2 show the numerical values of the echocardiographic and laboratory parameters observed at rest and after the match (within the "window" of 48 hours after the end of the match). With regard to the echocardiographic and laboratory variables, statistically significant differences were only observed for the variables Left Ventricular Ejection Fraction (LVEF) (61.1±4.82% after the game versus

68.6±2.76% at rest, p=0.006), Left Auricle Area (LAA) (21. 4±1.51mm2 after the game versus 17.1±1.62mm2 at rest, p=0.016), Left Auricle Volume (LAV) (65.9±7. 06 mm3 after the game versus 46.9±8.83 mm3 at rest, p=0.016), ratio of mitral flow diastolic velocity E to mitral annulus diastolic velocity e' (E/e') (5.0±1.21 after the game versus 4.4±1. 26 at rest, p=0.018), and Troponin I (2.6±1.13 ng/mL after the game versus 1.9±0.00 ng/mL at rest, two-tailed p=0.036), when comparing the 2 moments of the same athletes with a two-tailed paired hypothesis test. All the other echocardiographic and laboratory biomarker variables were not different between the two measurement times.

Table 2 - Analysis of clinical, echocardiographic and laboratory markers

Characteristics	After Game	Rest	Difference	TS value	pvalue
Echocardiographic					
LVEF (%)	61,1±4,81	68,6±2,76	-7,4±4,76	-4,133	**0,006**[1]
LVDD (mm)	55,3±3,86	55,0±4,58	0,3±2,31	0,135	0,893[2]
LVSD (mm)	33,9±5,08	35,0±5,02	-1,1±4,43	-0,524	0,688[2]
IVS (mm)	9,3±0,95	8,3±0,70	1.0±1.27	1,863	0,063[2]
PW (mm)	8,6±1,27	8,8±0,73	-0,2±1,38	-0,314	0,844[2]
LAA (mm2)	2,4±1,51	17,1±1,62	4,3±2,29	2,366	**0,016**[2]
DLA (mm)	35,7±3,73	37,3±2,82	-1,5±2,41	-1,527	0,172[2]
LAV (mm3)	65,9±7,06	46,9±8,83	18,9±4,87	2,366	**0,016**[2]
E' side	17,0±2,58	19,1±3,89	-2,1±3,36	-1,355	0,203[2]
E/e' ratio	5,0±1,21	4,4±1,26	0,6±0,44	2,366	**0,018**[2]
TRSEP (mm)	26,1±3,84	23,7±1,80	2,4±4,32	1,362	0,173[2]
Laboratory analysis					
Troponin (ng/mL)	10,3±20,45	5,1±7,06	5,2±13,38	1,069	0,285[1]

Data are presented as mean±standard deviation. Groups comparisons were based on a paired t-test when the difference variable (Difference=After Game - Rest) is normally distributed or on a Wilcoxon test when difference variable is non-Gaussian. TS= Teste statistics. 1pvalue for two-tailed Wilcoxon test. 2 pvalue for two-tailed paired t-test. Two-tailed pvalue <0.05 for differences between groups. HR=Heart rate; SBP=Systolic blood pressure; DBP=Diastolic blood pressure; LVEF=Left Ventricular Ejection Fraction; LVDD=Left Ventricular Diastolic Diameter; LVSD=Left Ventricular Systolic Diameter; IVS=Thickness of the Interventricular Septum; PW=Posterior wall; LAA=Left Auricle Area; DLA=Diameter of the Left Auricle; LAV=Left Auricle Volume; E/e' ratio= Ratio between diastolic velocity E of the mitral flow and diastolic velocity e' of the mitral annulus; TRSEP=Tricuspid Ring Systolic Excursion Plan

When comparing the "after match" and "at rest" values, it can be seen that the values for the parameters AAE (mean difference between post-match and at rest: -4.3±2.29mm, one-tailed pvalue =0.016), VAE (mean difference between post-match and at rest: 18. 9±4.87mm3, one-tailed pvalue=0.016), E/e (mean difference between post-match and rest: 0.6±0.44, one-tailed pvalue=0.018) are significantly higher post-match when compared to resting values. With regard to Troponin, although there was an increase "after the game", this was not statistically significant (mean difference between post-game and rest: 5.2±13.38ng /mL, one-tailed p-value=0.285). As for the LVEF value, it decreased from the "rest" phase to the "after game" phase (mean difference between post-game and rest: -7.4±4.76%, one-tailed p-value=0.031).
Table 3 shows the results of the comparison between athletes and patients with hypertrophic cardiomyopathy (HCM).

Table 3 - Comparison between athletes (after game) and patients with HCM.

Characteristics	Athletes	HMC patients	TS value	pvalue
LVEF (%)	61.1±4.81	66.6±7.51	-1.649	0.099[1]
LVDD (mm)	55.3±3.86	48.8±10.35	1.420	0.156[2]
LVSD (mm)	33.9±5.08	31.8±5.18	0.684	0.494[2]
IVS (mm)	9.3±0.95	24.6±7.08	-3.445	**<0.001**[2]
LAA (mm2)	21.4±1.51	27.5±1.65	-2.852	**0.004**[2]
DLA (mm)	35.7±3.73	51.0±10.70	-3.318	**<0.001**[1]
LAV (mm3)	65.9±7.06	57.3±35.40	1.289	0.199[2]
Troponin (ng/mL)	10.3±20.45	214.7±187.80	-3.162	**<0.001**[2]

Data are presented as mean±standard deviation. Groups comparisons were based on a t-test for two independent samples when the two samples are from populations with Gaussian distributions or on a Mann-Whitney test otherwise. Two-tailed pvalue <0.05 for differences between groups. [1]pvalue for two-tailed t-test for two independent samples. [2]pvalue for two-tailed Mann-Whitney test. Abbreviations as Table 2.

The sample of HCM patients (n = 10), all male, was characterised only by the age variable, ranging from 35 to 53 years (42.8 ± 7.32 years). The HMC patients were significantly older than the athletes (42.8 ± 7.32 years for MCH patients versus the age of the players, with an average of 22.6 ± 3.60 years), one-sided p-value <0.001).

Comparing HCM patients and athletes, it was possible to conclude that there are significantly greater structural cardiac alterations in HCM patients. The group of patients presented values of IVS (24.6±7.08mm versus 9.3±0.95mm; unilateral pvalue <0.001), LAA (27.5±1.65mm2 versus 21.4±1.51mm2; unilateral pvalue<0.004), LAD (51.0±10. 70mm versus 35.7±3.73 mm; one-sided p-value<0.001) and Troponin (214.86±187.8 ng/mL versus 10.3±20.45 ng/ mL; one-sided p-value<0.001) significantly higher when compared to the athlete group. On average, volleyball players had SIV values 15.4 mm smaller, LA area 6.1 mm2 smaller and LA diameter 15.3 mm smaller than patients with HMC. Troponin is, on average, 204.4 ng/mL higher in the group of patients with HCM.

Dicussion

Football is the most popular sport in the world and is played by men and women, children and adults with different levels of specialisation. Performance in football depends on a multitude of factors, such as technical/biomechanical, tactical, mental and physiological areas (Stølen et al., 2005). The physiological needs of high-level competitive football are to be in good physical condition in order to give maximum performance in a football match (running, holding the ball, passing and shooting). Optimising the level of physical condition is an important aspect of a team's preparation before entering a competition/match. The latest studies have shown data that a professional male footballer can reach 9 to 14 kilometres of total distance covered in a football match. And in elite women's football, a player can achieve around 8 to 12 km of total distance travelled (Yustika, 2018). The values often found for the average total distance travelled in a match are around 10 km and an above average, although not exceptional, maximum oxygen consumption of 60 ml/kg/min, suggesting a moderate overall aerobic metabolism.

A comparison between top teams and players with less able participants indicates that the components of anaerobic fitness – speed, power, strength and lactic acid system capacity can best differentiate between the 2 groups (Tumilty, 1993). Hypertrophic cardiomyopathy (HCM) is a genetically transmitted disease, clinically defined by the presence of unexplained left ventricular hypertrophy. The disease has a varied clinical course and evolution; Many patients present few or no discernible cardiovascular symptoms, while others have profound limitations when performing physical exercise and recurrent arrhythmias (Elliott & McKenna, 2004). Over the past 20 years, most data have revealed the occurrence of HCM in approximately 1 in 500 individuals. Taking into account the potential impact of these initiatives on the occurrence of diseases, different authors of different studies revisited the prevalence of HCM in the general population. They suggested that HCM is more common than previously estimated, which may increase its recognition in the medical community, allowing for more timely diagnosis and implementation of appropriate treatment options for many patients (Semsarian et al., 2015). HCM is characterized by cardiac hypertrophy, unexplained by loading conditions; non-dilated left ventricle; and a normal or increased ejection fraction. Cardiac hypertrophy is generally asymmetric, with greater involvement, most commonly, of the basal interventricular septum underlying the aortic valve. Occasionally, it is restricted to other regions of the myocardium, such as the apex, midportion, and posterior wall of the left ventricle. At the cellular level, cardiac myocytes are hypertrophied, disorganized, and separated by areas of interstitial fibrosis (Marian & Braunwald, 2017). In this study, the comparison between elite football players and patients with HCM showed that patients with this cardiomyopathy have significantly greater thickness of the IVS and also significantly greater dimensions of the LA (diameter and area). At least in relation to patients with HCM at high risk of sudden death, and when compared with football athletes, there seems to be no difficulty in making this same distinction based on echocardiographic evaluation.

When we compare the image data obtained by ECHO between athletes and patients with HCM, we can observe that the latter present significantly higher values for IVS, LAA and DLA.

The study by Perrone, M. et al (2020), with 22 amateur football players, in which blood samples to collect troponin values were collected before the start of the football game, immediately upon arrival, and the second Collection was carried out within 24 hours from the moment the game ended. Data showed a significant increase in serum troponin in amateur football players, but without any evidence of cardiac damage (Perrone et al., 2020). Another study, by Aldujeli, A. et al. (2019), reveals that the results of the study data state that the release of Troponin I is highly affected by the physical status of athletes, with the release of Troponin I being enhanced by the practice of intermittent exercise (Aldujeli et al., 2019).

The sports medical examination and, specifically, cardiovascular assessment and screening are a fundamental component in the athlete's pre-participation in screening for possible pathologies. Pre-participation medical examinations have become popular following the discoveries of the “Italian experience”, with medical history, physical examination and ECG (electrocardiogram) now mandatory for all Italian sports participants (Corrado et al., 2006). The main reason for carrying out pre-participation medical examinations is to screen for preventable causes of sudden cardiac death, which has gained much publicity in Europe recently with the collapse and successful resuscitation of Fabrice Muamba while playing for Bolton FC, as in addition to identify possible preventable causes of sudden cardiac death in athletes, pre-participation medical screening is proposed as a time to identify possible medical problems and optimize treatment (Heron & Cupples, 2014). One of the main components of carrying out the medical-sports examination is cardiac screening, of which the ECG is a

part, as it allows the identification of cardiac pathologies (Maron et al., 2015; Seto, 2003). The information obtained, and when the exam is carried out by experienced clinicians, makes this information possible to obtain reports with low false-positive rates, combining pre-participation cardiovascular screening, the inclusion of personal and family history, as well as an exam physical care and ECG. Therefore, additional tests (which may include echocardiography) are only requested if initial screening investigations show abnormal findings.
This could be the way to allow greater safety in participating in sporting events (Niederseer et al., 2021). It is important to note that cardiac screening, and in particular the ECG, reduced false positive rates to <3%. In contrast, false-positive response rates produced by cardiac screening questionnaires are 35% to 68% (Grazioli et al., 2014).

The incorporation of echocardiography is considered an accurate way to identify common cardiac abnormalities that can lead to sudden death, which may allow for faster and more effective screening (Palermi et al., 2021). It would be particularly important in the early diagnosis of some pathologies, which may be imperceptible on the ECG, particularly in their early stages.

The study by Donati, F. (2023) revealed that the inclusion of the screening echocardiogram showed additional value (around 10% more) in detecting patients with cardiovascular anomalies, which would otherwise not be diagnosed with the screening protocol. “standard” pre-participation screening (Donati et al., 2023). In our study, 57% of athletes showed changes in LVEF, LAA, LAV and E/e’; however, none of them presented criteria to recommend the interruption of sporting activity.
One of the questions that will be important to answer is what will be the limit for athletes' physical effort. The study by Lara, B., (Lara et al., 2019), which compared 63 athletes of three different distances (10 km, half-marathon and marathon), revealed that the tension imposed on the myocardium when competing in a full marathon is much greater compared to compete in shorter distances, such as the half marathon or 10km races. Although the release of cardiac troponins after exercise may not be indicative of any cardiovascular dysfunction, the higher concentration of cardiac troponins after the marathon reflects greater cardiac stress at this distance covered. The greatest cardiac stress after the marathon was present despite the greater training volume. The results of the study demonstrated how the distance covered affects the cardiac stress induced by an endurance running competition in athletes with little experience and little training history (Lara et al., 2019). As for our study with elite football players, it aims to evaluate the potential for myocardial injury associated with this sport, by determining biomarkers in two distinct phases of the sporting season (after competition and rest).

Regarding cardiac troponins, these biomarkers are detected in serum using monoclonal antibodies against the epitopes of troponin I and troponin T, these antibodies being highly specific for cardiac troponin, showing reduced interaction with skeletal muscle troponins (Regan et al., 2018).

In the football players in this study, troponin I values were higher after the game, when compared to the levels found at rest, but when we compare these values with those of patients with HCM, we found that the troponin I values found, they are substantially lower in athletes (-204).

The European Society of Cardiology and the American College of Cardiology advocate that each laboratory determine the cutoff points for each test at the 99th percentile, with a coefficient of variation of ± 10%. Using these criteria, serum troponin I values, indicative of myocyte necrosis/damage in the myocardium, range from 0.1 to 2 µg/L (Apple et al., 2002; Sandoval & Apple, 2014).

Thus, we can mention the high values in patients with HCM, as well as the significant increase in Troponins I after the game, in football athletes. It will also be important to mention that, as the main limitation of this study, we had compliance from the athletes, since, being elite athletes, their availability was limited, and for this reason, the number of athletes available to carry out cardiac screening be reduced.

Conclusions

The results obtained seem to indicate that, in athletes, and comparing the “post-competition” and “rest” phases, there is an increase in the thickness of the intraventricular septum, area of the left atrium, volume of the left atrium, as well as E/e´, with a parallel increase in troponin I, which may suggest possible cardiac damage associated with high-performance football. Regarding the left ventricular ejection fraction, there was a decrease in the “after game” phase, when compared to the “rest” phase, which could be explained by the fact that the left ventricular ejection fraction at rest is generally normal, but may be slightly reduced in healthy athletes. As it was possible to verify an increase in the dilation of the cavity, it allowed a normal systolic volume, with a lower ejection fraction.

Making the comparison “athletes vs patients with HMC”, we can say that the pathology that the latter present, causes the structural changes presented, mainly in terms of dimensions of the left auricle (area and diameter) and the IVS, to be substantially accentuated and higher than those of football players. Furthermore, the values found in high-sensitivity troponins I are much higher in magnitude than those found in athletes.
Thus, troponins I may play an increasingly relevant role in the differential diagnosis where doubts may remain between “athlete's heart” and HCM, but more and larger studies are needed.

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