Cardiac remodeling induced by exercise in male master athletes

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Abstract

Aims: To describe cardiac remodeling in a population of male master athletes evaluated by transthoracic echocardiography and to analyse its relationship with several exercise-related characteristics.

Methods and results: A total of 105 male master athletes aged ≥40 years old, mostly involved in endurance sports (81.0%) with a median training-volume of 66 [44; 103] METs/h/week, were studied. Left ventricular end-diastolic and end-systolic volumes were above the references in 84.8% and 75.8% athletes, decreasing in frequency when adjusted for BSA (26.3% and 23.2%). LV geometry was changed in more than half of the athletes (eccentric hypertrophy 28.3%, concentric remodelling 15.2% and concentric hypertrophy 8.1%) and several right ventricular (RV) dimensions were increased. Left atrium was dilated in 53.5% and right atrium in 37.4% athletes; only one athlete had a dilated aorta. Mean LV ejection fraction was 61±7% and global longitudinal strain -18.3±2.0%. Changes in LV geometry were more common in high intensity sports; LV dilation in athletes exercising >10 hours/week and in high intensity sports; RV dilation in athletes exercising >66 MET-hour/week and in endurance sports. In multivariate analysis high intensity sports remained an independent predictor of changes in LV geometry. There was a significant correlation between volume of exercise and cardiac structural adaptations.

Conclusions: Cardiac structural adaptations were frequent in male master athletes, more pronounced in those involved in endurance sports, with high intensity and high volume of exercise. This data reinforces the concept that the characteristics of exercise are major determinants of cardiac remodeling and should be considered during athletes' evaluation.

Introduction

Regular exercise training induces several cardiac adaptations, influenced by multiple factors.[1] These changes were described over a century ago, but the distinction between physiological findings and pathological conditions remains frequently challenging. Most studies describing cardiac adaptations exercise-induced were performed in young athletes, but in the last few decades endurance events have become more popular, with the participation of a growing number of master athletes. The influence of lifelong exercise in cardiac structure and function in older individuals is not well understood.[2,3] On the other hand, recent data in veteran or master athletes have shown a potential relationship between years of endurance exercise and the development of some cardiac pathological conditions, justifying the clarification of this issue and a deeper study of this specific population.[4–6]

Different types of exercise impose different loads on the cardiovascular (CV) system, resulting in different cardiac adaptive changes, more pronounced in endurance exercise. As a matter of fact, the heart of endurance athletes is under great stress during training and competition. Structural cardiac adaptations are more described for left ventricle (LV), but lifelong endurance exercise has been related to right ventricle (RV) enlargement, typically balanced with LV dilation.[8,9] The magnitude of these adaptations and even the values of normality by transthoracic echocardiogram (TTE) for master athletes should be

established.[10] As many older athletes perform high levels of exercise since their youth, a higher influence of regular exercise training on cardiac structure and function can be expected when athletic conditioning has been continuously maintained throughout the years.[11] Among the characteristics associated with high levels of cardiac remodeling, several are related with exercise and should be considered during the evaluation and interpretation of TTE in master athletes. In this setting, although not routinely recommended in the pre-participation screening protocols, TTE might be a useful, non-invasive and accessible tool for athletes' evaluation.[7]

The current study aimed to describe the cardiac remodeling in a population of male master athletes evaluated by TTE and to analyse its relationship with several exercise-related characteristics.

Methods

Study design and participants

This cross-sectional study included male master athletes with ≥ 40 years old participating in regular exercise for at least 4 hours/week for a minimum of 5 years. Individuals with symptoms, known CV disease, diabetes mellitus, Systematic Coronary Risk Estimation (SCORE) ≥5% and contraindication for competitive sport were excluded from this analysis. The athletes' evaluation was performed at a dedicated sports cardiology clinic, including a detailed sports and personnel clinical history, CV risk stratification according to the SCORE charts[12], family history of CV disease, documentation of demographic data, physical evaluation with blood pressure, heart rate and anthropometric data, 12-lead electrocardiogram and exercise testing.

Regarding the characterization of exercise training, the sporting disciplines were classified according to the four groups proposed by the European Society of Cardiology[13] (skill, power, mixed and endurance sports). The number of years of continuous exercise practice, the number of hours and training sessions per week, the intensity of exercise (METs for each sport according to the Compendium of Physical Activities[14]) and the level of competition, divided in competitive and non-competitive, were also registered. The volume of exercise was calculated by the Metabolic Equivalent Task Score (MET-hour/week), representing the product of intensity, frequency (number of training sessions per week) and duration of exercise (hours in each workout).

The study was approved by the ethics committees and all individuals gave their informed consent for participation.

Echocardiographic analysis

A TTE was performed using a high-quality echocardiograph Vivid E9 (GE Vingmed Ultrasound AS, Horten, Norway) in accordance with the current recommendations of the American Society of Echocardiography and the European Association of Cardiovascular Imaging.[15] The reference values proposed in these recommendations were also used as the normal values for the present analysis. Only the exams

considered complete and with quality for the accurate measurement of all the parameters were included. TTE was performed under normal hydration and more than 12 hours after the last training session.

The linear measurements of the LV were achieved by 2D echocardiography from parasternal long axis views, while LV end-diastolic volume (LVEDV), LV end-systolic volume (LVESV) and LV ejection fraction (LVEF) were obtained by biplane modified Simpson's method. Chamber dimensions and LV mass were indexed (LVMI) to body surface area (BSA). LV hypertrophy (LVH) was defined as LVMI > 115g/m^2, concentric if relative wall thickness (RWT) > 0.42 and eccentric if RWT ≤0.42; concentric remodeling was defined as LVIM ≤115g/m^2 and > 0.42. Diastolic function was assessed from apical four-chamber view, with pulsed-wave Doppler transmitral flow and tissue Doppler velocities, recorded with the sample in the mitral annulus (interventricular septum and LV lateral wall). Global Longitudinal Strain (GLS) was evaluated in the three standard apical views in a LV 17-segment model and averaged after three consecutive cardiac cycles for each view. The tracking quality was maximized with a frame rate > 55fps, avoiding foreshortening. The measurements of RV were made from end-diastolic frames. Two measurements of RV outflow tract (RVOT) diameter were made in the parasternal short-axis view: from the RV free wall to the anterior aortic wall (RVOT1) and immediately proximal to the pulmonary valve (RVOT2), and one in parasternal long-axis view (RVOTP). The apical 4-chamber view was modified by adjusting the probe to optimize RV size (RV-focused apical view), and three diameters were assessed: RV basal (RVD1), midcavity (RVD2), and longitudinal (RVD3). RV systolic function was assessed by tricuspid annular plane systolic excursion (TAPSE) and tissue Doppler-derived RV peak systolic velocity (RV *S'*).[9] The left atrium (LAV) and right atrium (RAV) volumes were evaluated in 4-chamber view, while the aorta was measured in parasternal long-axis view (Valsalva sinus, sinotubular junction and proximal ascending aorta) and in suprasternal view (aortic arch). Analyses were performed offline using a dedicated software package (EchoPac PC version BT13, GE Medical Systems, Faireld, CT, USA) by two experienced cardiologists with level III training (HD and NC).

Associations between echocardiographic findings and exercise-related characteristics

Five structural cardiac adaptations were predefined: 1 – Changes in LV geometry (concentric hypertrophy, eccentric hypertrophy or concentric remodelling); 2 - LV dilation (LVEDV index > 74ml/m^2); 3 - RV remodelling (RVD1 > 41mm); 4 - LA dilation (LAV index > 34ml/m^2); 5 - RA dilation (RAV index > 32ml/m^2). The prevalence of these findings was analyzed according to exercise-related characteristics, specifically: hours of exercise-training per week, intensity of exercise, type of sport and volume of exercise. Additionally, the volume of exercise was correlated with echocardiographic parameters.

Statistical analysis

All analysis was performed using SPSS for Mac version 26.0 (SPSS, Inc., Chicago, IL). Normality was tested with the Kolmogorov-Smirnov test. Continuous variables with normal distribution were expressed as means and standard deviation, and non-normal variables as median (interquartile range [IQR]). Categorical variables were expressed as frequencies and percentages. Statistical comparison was

performed using chi-square test or Fisher's exact test, when appropriate for categorical variables, and Mann-Whitney or Kruskal-Wallis test for continuous variables. Pearson analysis was performed to evaluate the correlations between the volume of exercise and echocardiographic parameters. Binary logistic multivariate analysis was performed for identification the independent predictors of structural remodeling. A *p* value < 0.05 was considered statistically significant.

Results

Baseline characteristics

Demographic, clinical and exercise-related characteristics are shown in Table 1. Of the 105 athletes included, 98.1% were Caucasian, with a mean age of 48±6 years and BSA of 1.9±0.1m^2. All athletes had low to intermediate CV risk, more than a half (53.3%) classified as low risk (SCORE = 0). The mean number of years with continuous exercise training was 17±10 years (31.4% >20 years), the mean hours/week was 8±5 (16.2% >10 hours), distributed in 5±1 sessions/week, and the median volume of exercise was 66 [44; 103] METs/h/week. Most athletes were involved in endurance sports (81.0%), followed by power sports (12.4%) and mixed sports (6.7%). The most frequent sports were long distance running (63.8%), cycling (10.5%) and weight lifting (9.5%). The majority of the athletes were involved in competitive sport (66.7%) and 29.5% in disciplines characterized by high intensity exercise.

Table 1
Baseline characteristics (N = 105).

Demographics and Clinical	
Caucasians, n (%)	103 (98.1)
Age, y	48±6
Height, cm	176±6
Weight, kg	76±9
BMI, kg/m^2	24.5±2.5
BSA, m^2	1.9±0.1
SBP, mmHg	125±11
DBP, mmHg	78±8
Heart rate, bpm	57±8
Low CV risk (SCORE = 0), n (%)	56 (53.3)
Exercise-related	
Hours/week, h	8±5
Sessions/week, n	5±1
Volume of exercise, METs/h/week	66 [44; 103]
Years of continuous exercise training, y	17±10
Endurance sports, n (%)	85 (81.0)
Mixed sports, n (%)	7 (6.7)
Power sports, n (%)	13 (12.4)
High intensity exercise, n (%)	31 (29.5)
Competitive level, n (%)	70 (66.7)
BMI: body mass index; BSA: body surface area; CV: cardiovascular; DBP: diastolic blood pressure; SBP: systolic blood pressure.	

Echocardiographic evaluation

Structural findings

TTE was considered complete and with adequate quality for the measurement of all parameters in 94.3% athletes. The echocardiographic findings are shown in Tables 2 and 3. LV end diastolic diameter (LVEDD) ranged from 42 to 71mm (mean 51±5mm), greater than 58mm in four athletes. The mean LVEDV was

194±41ml (69±13ml/m^2) and LVESV was 69±13ml (27±7 ml/m^2). According to the recommendations[15], 84.8% and 75.8% athletes had respectively LVEDV and LVESV above the reference values. This frequency decreased to 26.3% and 23.2% when adjusted for BSA. LV wall thickness (LVWT) ranged from 7 to 14mm (10.4±1.4mm), greater than 10mm in 49.5% athletes, but only in one exceeding 13mm (14mm), while the mean LVMI was 111±30g/m^2. Most of the athletes (51.6%) had changes in LV geometry: 28.3% eccentric hypertrophy, 15.2% concentric remodelling and 8.1% concentric hypertrophy (Fig. 1). No cardiomyopathies were identified. The Fig. 2 shows some echocardiographic findings of a 47 years-old male ultra-distance triathlete, namely structural remodelling (eccentric LV hypertrophy and dilated LA), while parameters of diastolic and systolic function are normal.

Table 2
Echocardiographic findings.

Left ventricle	
LVEDD, mm	51±5
LVESD, mm	32±9
Maximum LVWT, mm	10.4±1.4
LVMI, g/m^2	111±30
RWT	0.38±0.06
LVEDV (biplane), ml	194±41
LVESV (biplane), ml	79±23
LVEDV index, ml/m^2	69±13
LVESV index, ml/m^2	27±7
LVEF (biplane), %	61±7
GLS, %	-18.3±2.0
Peak *E* velocity, m/s	0.76±0.14
Peak *A* velocity, m/s	0.57±0.12
E/A ratio	1.4±0.4
E/e' ratio	6.6±1.7
Right ventricle	
RVOTP, mm	36±4
RVOT1, mm	35±6
RVOT2, mm	21±3
RVD1, mm	43±7
RVD2, mm	31±5
RVD3, mm	67±9
RVWT, mm	6±1
TAPSE, mm	26±4
PASP, mmHg	21±5
RV *S'*, cm/s	0.14±0.03

Left ventricle	
Atria	
LAV index, ml/m^2	36±9
RAV index, ml/m^2	30±10
Aorta	
Sinuses of Valsalva, mm	33±4
Sinotubular junction, mm	29±4
Proximal ascending, mm	32±5
Aortic arch, mm	27±4

GLS: global longitudinal strain; LAV: left atrium volume; LVEDD: left ventricular end-diastolic diameter; LVEF: left ventricular ejection fraction; LVESD: left ventricular end-sistolic diameter; LVMI: left ventricular mass index; LVWT: left ventricular wall thickness; PASP: pulmonary artery systolic pressure; RAV: right atrium volume; RV: right ventricular; RVD1: right ventricular basal dimension; RVD2: right ventricular midventricular dimension; RVD3: right ventricular longitudinal dimension; RVOT1: proximal right ventricular outflow tract dimension; RVOT2: distal right ventricular outflow tract dimension; RVOTP: right ventricular outflow tract in parasternal long-axis; RVWT: right ventricular free wall thickness; TAPSE: tricuspid annular plane systolic excursion.

Table 3
Structural and functional parameters beyond the reference values.

Left ventricle	
LVEDD > 58mm, n (%)	4 (4.0)
LVESD > 39mm, n (%)	2 (2.0)
LVWT > 10 mm, n (%)	49 (49.5)
Concentric hypertrophy, n (%)	8 (8.1)
Concentric remodelling, n (%)	15 (15.2)
Eccentric hypertrophy, n (%)	28 (28.3)
LVEDV (biplane) > 150ml, n (%)	84 (84.8)
LVESV (biplane) > 61ml, n (%)	75 (75.8)
LVEDV index > 74ml/m^2, n (%)	26 (26.3)
LVESV index > 31ml/m^2, n (%)	23 (23.2)
LVEF (biplane) < 52%, n (%)	6 (6.1)
GLS < 17%, n (%)	22 (22.7)
Right ventricle	
RVOTP > 30mm, n (%)	89 (90.8)
RVOT1 > 35mm, n (%)	43 (43.4)
RVOT2 > 27mm, n (%)	1 (1.0)
RVD1 > 41mm, n (%)	57 (57.6)
RVD2 > 35mm, n (%)	20 (20.2)
RVD3 > 83mm, n (%)	2 (2.0)
RVWT > 5mm, n (%)	46 (46.5)
TAPSE < 17mm, n (%)	0 (0)
RV S'<9.5cm/s, n (%)	4 (4.0)
Atria	
LAV index > 34ml/m^2, n (%)	53 (53.5)
RAV index > 32ml/m^2, n (%)	37 (37.4)
Aorta	

Left ventricle	
Diameter > 40mm, n (%)	1 (1.0)

GLS: global longitudinal strain; LAV: left atrium volume; LVEDD: left ventricular end-diastolic diameter; LVEF: left ventricular ejection fraction; LVESD: left ventricular end-sistolic diameter; LVMI: left ventricular mass index; LVWT: left ventricular wall thickness; RAV: right atrium volume; RVD1: right ventricular basal dimension; RVD2: right ventricular midventricular dimension; RVD3: right ventricular longitudinal dimension; RVOT1: proximal right ventricular outflow tract dimension; RVOT2: distal right ventricular outflow tract dimension; RVOTP: right ventricular outflow tract in parasternal long-axis; RVWT: right ventricular wall thickness.

Regarding the RV, some dimensions were frequently above the reference values, especially RVOTP (90.8%), RVD1 (57.6%) and RVOT1 (43.4%). Similarly to the LV, RV wall thickness was above the normality in a large proportion of the population (46.5%). The mean LAV index was 36±9ml/m^2, more than half of the athletes showing LA dilation (53.5%); the mean RAV index was 30±10 ml/m^2, dilated in 37.4% athletes.

The aortic root diameter ranged from 21 to 45mm (33±4mm at Valsalva sinuses and 32±5mm at proximal ascending aorta). Only one athlete had dimensions greater than 40mm (45mm at Valsalva sinuses and 42mm at proximal ascending aorta), confirmed in a thoracic contrast-enhanced computed tomography. Two athletes had significant valvular changes, one bicuspid aortic valve and one mitral valve prolapse, both without significant functional repercussion.

Functional findings

The LVEF ranged from 41–79% (61±7%), and it was lower than 52% in 6 athletes. All of these athletes demonstrated normal augmentation of LVEF after exercise stress testing and absence of pathological findings in further investigations. The mean LV GLS was − 18.3±2.0%, lower than 17% in 22 athletes (22.7%). No athlete had diastolic dysfunction. The mean *E/A* ratio was 1.4±0.4 (> 2 in 6 athletes) and *E/e'* was 6.6±1.7 (< 13 in all athletes). All individuals had normal values of TAPSE (26±4mm) and of RV S' peak systolic velocity (0.14±0.03cm/s).

Characteristics of exercise and marked cardiac remodelling

The association between exercise-related characteristics and structural changes are presented in Table 4. Change in LV geometry was more common in high intensity sports (70.0% Vs. 43.5%; p = 0.015). LV dilation was more common in athletes exercising > 10 hours/week (64.7% Vs. 26.8%; p = 0.003) and in high intensity sports (50.0% Vs. 26.1%; p = 0.020), while RV dilation was more common in athletes exercising > 66 MET-hour/week (71.7% Vs. 41.3%; p = 0.002) and in endurance sports (63.7% Vs. 31.6%; p = 0.011). In multivariate binary logistic analysis, high intensity sports remained an independent predictor of change in LV geometry (OR 2.91; 95% CI 1.03–8.23; p = 0.044). There was a significant linear correlation between volume of exercise and cardiac structural adaptations (Fig. 3): LVMI (r0.077; p = 0.005); LVEDV index (r0.066; p = 0.011); RVD1 (r0.084; p = 0.004); LAV index (r0.132; p < 0.001); RAV index (r0.062; p = 0.013).

Table 4
Structural cardiac remodelling according to exercise-related characteristics (%).

Hours of exercise/week	**≤10**	**>10**	***p* value**
Changes in LV geometry	51.2	52.9	0.897
LV dilation	26.8	64.7	0.003
RV dilation	56.1	64.7	0.513
LA dilation	51.2	64.7	0.310
RA dilation	32.9	58.8	0.045
Intensity of exercise	**Moderate-low**	**High**	p **value**
Changes in LV geometry	43.5	70.0	0.015
LV dilation	26.1	50.0	0.020
RV dilation	53.6	55.7	0.228
LA dilation	52.2	56.7	0.680
RA dilation	36.2	40.0	0.722
Type of sport	**Non-endurance**	**Endurance**	p **value**
Changes in LV geometry	63.2	48.8	0.259
LV dilation	47.4	30.0	0.149
RV dilation	31.6	63.7	0.011
LA dilation	52.6	53.8	0.930
RA dilation	31.6	38.8	0.561
Volume of exercise	**≤66 METs/h/week**	**> 66 METs/h/week**	p **value**
Changes in LV geometry	50.0	52.8	0.779
LV dilation	23.9	41.5	0.064
RV dilation	41.3	71.7	0.002
LA dilation	47.8	58.5	0.289
RA dilation	37.0	37.7	0.936

Changes in LV geometry: concentric hypertrophy, eccentric hypertrophy or concentric remodelling; LV dilation: LVEDV index > 74ml/m^2; RV dilation: RVD1 > 41mm; LA dilation: LAV index > 34ml/m^2; RA dilation: RAV index > 32ml/m^2.

LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle.

Discussion

In this study, performed in male master athletes, mainly involved in lifelong endurance and competitive sports, structural cardiac adaptations were frequent. These changes affected all cardiac chambers and were more pronounced in athletes involved in endurance sporting disciplines, with high intensity and high volume of exercise. Importantly, this data reinforces the concept that the characteristics of exercise are major determinants of cardiac remodeling, also in master athletes.

Physiological cardiac adaptations induced by regular exercise training encompasses morphological, functional and electrical changes, routinely designed as 'athletes' heart'. This cardiac remodeling is influenced by individual and anthropometric characteristics, such as gender, ethnicity or BSA, and several exercise-related characteristics.[1] Thus, during athlete's evaluation it is important to consider these factors. Though in young athletes several studies describing with detail the cardiac adaptations to exercise have been published in the last years, data regarding older and master athletes are scarce and controversial.[16]

Structural changes

The long-term practice of competitive endurance sports produces an overall pattern of cardiac remodeling characterized by a balanced and homogeneous increase in the dimensions of the cardiac chambers.[17] In 1975, Morganroth et al[18] described by TTE the relationship between type of exercise and LV remodeling, showing a greater LVM due to greater LVEDV in athletes involved in dynamic exercise. In the current study, although the majority (more than two thirds) of the athletes had LV volumes above the reference, its number decreased substantially when adjusted to BSA, reinforcing the advantage of the use of indexed values. LV geometry it was changed in approximately half of the athletes, mainly due to eccentric hypertrophy. Similar findings were showed by Utomi at al[19] in a cohort of athletes engaged in dynamic sports, with predominance of normal LV geometry and eccentric hypertrophy. In a study performed by Ryffel et al[20] on a population of male adult competitive athletes, the adaptive responses to endurance exercise differed according to the age of onset of training. Athletes that started at a younger age had more frequently an eccentric pattern, while athletes that started exercise at an older age showed more frequently a concentric hypertrophy. Elite and recreational athletes have a similar geometric pattern, but the last showed a lower degree of LV hypertrophy, in consideration of the lower intensity of exercise. Conversely, athletes that started regular exercise at middle age had lower increase in LV cavity size and higher increase in LVWT, which may result in a relative concentric pattern of LV hypertrophy. The sports starting age and the number of years of continuous endurance training should also be considered and may lead to a better understanding of structural cardiac adaptations. Finocchiaro et al[21] reported that athlete's gender also affects the LV remodeling phenotype in endurance athletes, with a more common concentric pattern among male and eccentric pattern among female athletes. However, as the study was performed in young athletes and in the current study only male athletes were included, this association was not possible to prove.

RV enlargement was also common in most of the athletes. Physiological adaptations in the RV are frequently observed in athletes, especially in those involved in lifelong high-level endurance exercise. The RV chamber size seems to increase during the competitive season in top-level athletes, without association with a reduction in RV function or myocardial deformation and occurs in close association with changes on LV, suggesting physiological remodeling.[22] In this context, the potential for erroneous diagnosis of arrhythmogenic cardiomyopathy is considerably greater.[9]

LA dilation was evident in more than half of the athletes included. This adaptation was described in several previous studies, but most of them used linear dimensions and not indexed volumes, as recommended and performed in our study. LA remodeling in competitive athletes may be regarded as a physiologic adaptation to exercise conditioning, largely without adverse clinical consequences.[23] In a recent meta-analysis, Cuspidi et al[24] suggests that the adaptation of LA to intensive exercise is characterized by a marked increase in LAV, being more pronounced in the athletes involved in high-dynamic/high-static exercise. Elite athletes have larger LA dimensions compared with controls when evaluated by either LA diameter or volume corrected for BSA, with the largest average diameters reported in endurance athletes.[25]

Only one athlete had aorta segments dimensions above the reference values. Although it has been reported an association between athletic training small increase in aorta dimensions, this difference is clinically nonsignificant.[26] Dilation of aorta in athletes is uncommon, unlikely to represent a feature of athlete's heart, and most probably an expression of a pathological condition, requiring close clinical surveillance.[27]

The structural adaptations observed were significantly associated with several characteristics of exercise. LV remodelling was more common in high intensity sports and athletes exercising more hours/week, while RV dilation in endurance sports and athletes with higher volume of exercise. Specifically, regarding the volume of exercise, there was a significant linear correlation with structural adaptations in the four cavities. The pattern and magnitude of physiologically adaptations also vary with the nature of sports training. Data from large athlete populations assessed with multivariate analysis show that 75% of variability in LV cavity size is attributable to factors including type of sport, gender and age, but BSA was the largest of these components.[28]

Functional changes

Only a minority of athletes showed a reduced LVEF at rest. Although uncommon, this finding has been previously described in different populations of endurance athletes.[29] On the other hand, a significant percentage of athletes (22%) had an abnormal GLS. The impact of exercise training on LV systolic mechanics remains unclear, but this finding was also previously published, in a study in which the GLS was reduced in 31% of the athletes studied, more frequently in those involved in high level exercise. The reduction of GLS was associated with a normal/enhanced diastolic performance and larger LV volumes, which can justify the lower needs of systolic deformation to eject the same stroke volume than athletes

with smaller volumes.[30] In this seting, the values of GLS in our study also seem to belong to the spectrum of healthy physiology cardiac remodeling. In line with this assumption is the presence of normal diastolic function in all athletes, which may represent a beneficial effect of regular exercise, enhancing LV diastolic function and counteracting its reduction associated with normal ageing.[2]

Physiological versus pathological adaptations

Despite the fact that cardiac adaptations have been described as physiological and reversible in the majority of the athletes, the eventual development of pathological events after lifelong exercise training is an actual issue on debate.[31] In a study performed by Pelliccia et al,[32] among 114 young Olympic endurance athletes, no cardiac diseases were diagnosed after up to 17 years of intense and uninterrupted endurance training. However, in a different study of elite male athletes with large cardiac dimensions, followed over 5.6 years of deconditioning period, the resolution of cavity enlargement was incomplete in some athletes, which could not rule out future clinical implications.[33] In the current study, during a mean continuous exercise training of 17±10 years, no pathological adaptations were identified, but no deconditioning was performed.

The ‘athlete’s heart’ is easily differentiated from pathological conditions in the majority of the cases, but some athletes present changes in the ‘grey zone’, in which a comprehensive evaluation of several clinical and complementary date should be valorized.[34] TTE assumes a main role in evaluation and characterization of the cardiac adaptations in athletes and the differential diagnosis from several pathological conditions. Although not recommended for pre-participation screening of athletes, some authors propose TTE in specific ages, especially a first exam during adolescence to rule out structural heart conditions associated with sudden cardiac death not detected by electrocardiogram, and a second exam from the age of 30–35 years, when athletes become master, to evaluate pathological cardiac remodeling to exercise, late onset cardiomyopathies and wall motion abnormalities due to myocarditis or coronary artery disease.[35]

Limitations

Some study limitations should be highlighted. The data do not apply to women and may not be representative of all sports modalities, as recruitment bias cannot be excluded. The sample size may be underpowered to test the methodology used. As the study was cross-sectional, some factors with potential influence in cardiac adaptations, such as past performance enhancing substances abuse, were difficult to collect. The absence of a control group limits the clarification of the influence of the exercise-related characteristics in cardiac remodeling.

Conclusions

Male master athletes, predominantly involved in endurance and competitive sports frequently show structural cardiac adaptations. These changes affected all the cardiac chambers, being more pronounced in endurance sports and in athletes with higher intensity and volume of exercise. These data reinforce the

concept that the characteristics of exercise are major influencers of cardiac remodelling and should be considered during athletes' evaluation. Further research is required to determine the potential for detrimental cardiac effects of lifelong exercise training.

Declarations

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Figures

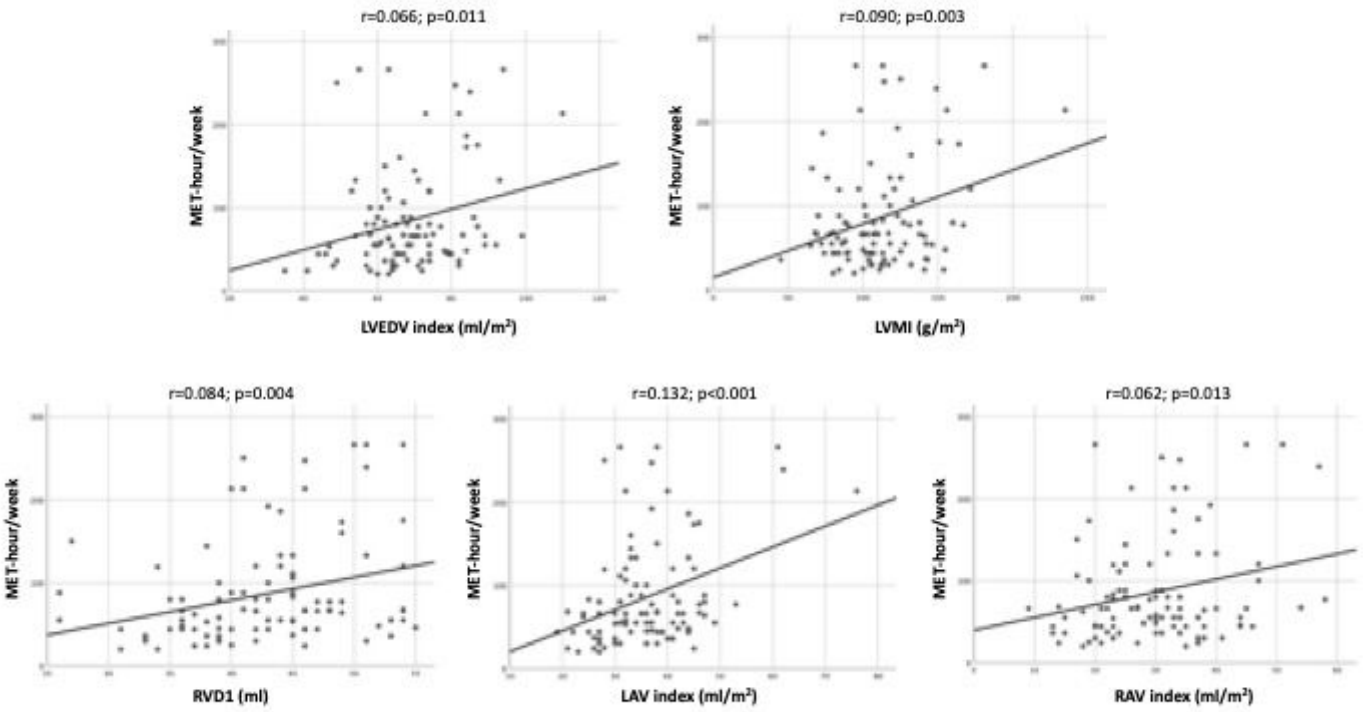

Figure 1

Left ventricular geometry.

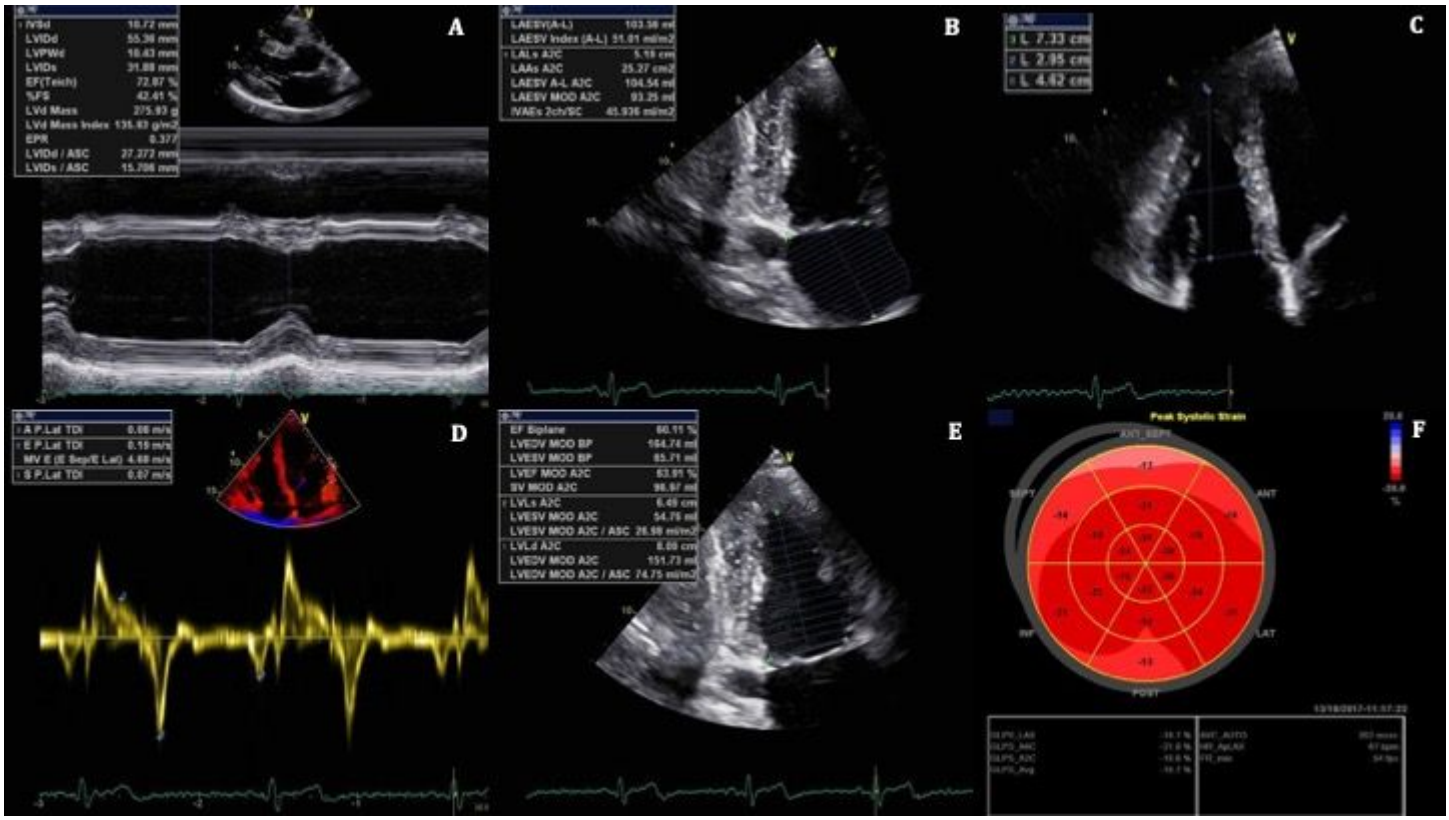

Figure 2

Echocardiographic findings of a 47 years-old male ultra-distance triathlete: A - M mode of LV showing eccentric hypertrophy; B - Dilated LA (indexed biplane volume); C - RV dimensions (apical 4-chamber view: RVD1, RVD2 and RVD3); D - Diastolic function (tissue Doppler velocities); E - LV volumes and LVEF (biplane); F - 2D LV GLS.

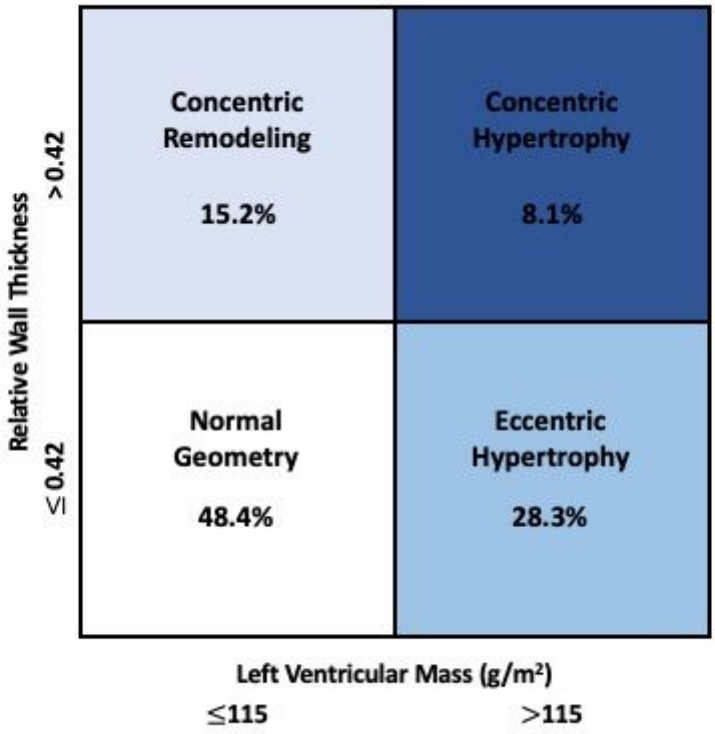

Figure 3

Statistically significant correlations between the volume of exercise (MET-hour/week) and echocardiographic parameters.