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ORIGINAL ARTICLE

Cardiac performance after an endurance open water swimming race

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Abstract

Purpose Endurance exercise competitions have shown a transient negative effect on global right ventricular (RV) performance. Most published studies are based on terrestrial sports. The aim of our study was to evaluate the cardiac effects after an open water swimming race.

Methods We evaluated 33 healthy swimmers (mean age 40.9 ± 7.2) participating in a 9.5 km open water swimming race. All subjects underwent a standard transthoracic echocardiography including an evaluation of dimensions and myocardial ventricular deformation. Echocardiography was performed 24 h before and within the first hour of arrival at the finish line. Cardiac troponin I (cTn I), NT-ProBNP and leukocytes were also evaluated.

Results No changes in left ventricle (LV) ejection fraction or LV global longitudinal strain were observed. A significant increase in RV end-diastolic area (RVEDA) was noted after the race (RVEDA at baseline 15.12 ± 1.86; RVEDA after race 16.06 ± 2.27, $p < 0.05$), but no changes were seen in RV fractional area change or RV global longitudinal strain. Cardiac biomarkers and leukocytes significantly increased. No association was detected between the increase in cTn I or NT-proBNP and the RV acute dilatation or LV performance. A significant association was observed between cTn I and leukocytes ($r = 0.375$, $p < 0.05$).

Conclusions An acute RV dilatation but without an impairment in RV deformation was observed after participating in an endurance swimming race. The correlation between the increase in cTn I and leukocytes, but not with ventricular performance, may support the hypothesis of an exercise-induced increase in myocardial sarcolemmal permeability due to an inflammatory response rather than myocardial injury.

Keywords Athletes · Endurance swimming · Right ventricle remodelling · Cardiac performance

Abbreviations

BSA	Body surface area
cTn I	Cardiac troponin I
CO	Cardiac output
CRP	C-reactive protein
DBP	Diastolic blood pressure
ET	Ejection time
FWRV	Free wall right ventricle
GFR	Glomerular filtration rate
HR	Heart rate
IVS	Inter-ventricular septal thickness
LV	Left ventricle
LVEF	Left ventricle ejection fraction
LVEDV	Left ventricle end-diastolic volume
LVGLS	Global left ventricle peak systolic strain
PW	Pulsed Doppler
PWT	Posterior wall thickness
RV	Right ventricle
RVEDA	Right ventricle end-diastolic area
RVFAC	Right ventricle fractional area change
RVGLS	Global right ventricle peak systolic strain
SBP	Systolic blood pressure
SD	Standard deviation

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SV Stroke volume
TDI Tissue Doppler imaging
TP Time to peak

Introduction

Endurance training induces cardiac functional and structural changes as a result of the cardiovascular adaptation to overload induced by exercise (Pluim et al. 2000; Maron and Pelliccia 2006). The right ventricle (RV) seems to present the most pronounced remodelling (La Gerche et al. 2011, 2012), since it has to support a disproportionate wall stress increase as compared to the left ventricle (LV) and thus might be more susceptible to fatigue after strenuous exercise (Teske et al. 2009). A different adaptation to exercise of different RV segments has also been observed (Teske et al. 2009; Sanz de la Garza et al. 2016a). In addition, an acute release in cardiac biomarkers has been demonstrated after acute bouts of endurance exercise (Shave et al. 2007b; Knebel et al. 2009; Mousavi et al. 2009; Karlstedt et al. 2012), which has been related to potential transient myocardial damage and associated to at least transient RV dysfunction (Neilan et al. 2006a, b; La Gerche et al. 2012). Furthermore, it has been hypothesized that training and competing in extreme endurance events over years may lead to myocardial fibrosis (La Gerche et al. 2012) in some predisposed individuals, potentially acting as a substrate for ventricular arrhythmias (Ector et al. 2007; Benito et al. 2011; La Gerche et al. 2015).

However, most of these studies have been performed on subjects practising terrestrial sports, particularly running and cycling, and there is little information about the effects of swimming on cardiac performance (Alexiou et al. 2005). In 2008, open water swimming became an Olympic sport. Since then, the number of national and international competitions (competitive distances between 5 and 25 km) has grown significantly and includes athletes of a wide range of ages. The specific features of this sports discipline might differentially influence cardiac performance (Tipton and Bradford 2014).

Therefore, the purposes of this study was: (1) to analyze the cardiac structural and functional response to an open water swimming race with special focus on the right ventricle; (2) to determine, through biochemical analysis if participation in a competitive open water swimming event promotes the release of cardiac biomarkers; and (3) to examine if this release of cardiac biomarkers is related to the cardiac structural and functional responses to exercise.

Materials and methods

Study population

All registered swimmers in the open water swimming race "Oceanman Palamós 9.5 km" (Catalonia, Spain) aged between 18 and 55 years, were invited to participate in our prospective study by e-mail. Finally, 33 healthy volunteer swimmers gave their informed written consent to participate in the study. Between 8 and 4 weeks before the race, a cardiovascular evaluation including a comprehensive personal and family history, resting 12-lead electrocardiogram, treadmill stress test and transthoracic echocardiograpy at rest was performed to exclude cardiovascular disease. All participants filled out a questionnaire providing details of their training history. Swimmers were asked to refrain from vigorous training during the 24 h before the race to avoid basal elevations of cardiac biomarkers. There was no fluid restriction during the competition and the use of a wetsuit was optional.

The study protocol was approved by the ethics committee of our institution and complies with the Declaration of Helsinki.

Baseline and post-race clinical and analytical parameters

Basal heart rate (HR) and systolic and diastolic blood pressure were determined at 24 h before race and immediately after arriving at the finish line. Blood samples were collected at baseline (24 h before race) and immediately after arriving at the finish line. Blood was taken via venipuncture into an EDTA tube and serum was obtained by centrifugation. All samples were immediately frozen and stored in a refrigerated box until analysis was performed in a central laboratory. Specimens for analysis were transported to the laboratory within 8 h after the blood was drawn. To evaluate hydration status, baseline and post-race urea, creatinine, glomerular filtration rate (GFR), sodium, and haematocrit values were measured.

Quantitative determination of cardiac troponin I (cTn I) was performed by a chemiluminescent method using a multiparametric analyzer Dxl-800 (ACCESS AccuTnI + 3, Beckman Coulter, Nyon, Switzerland). The level above the lower limit of detection, which is equal to the 99th percentile of the reference population, was 30 ng/l. NT-proBNP levels were measured with an electrochemiluminescent method using a COBAS 411 analyzer (proBNP II, Roche Diagostics, Mainnheim, Germany). The upper limit of normality was considered 125 pg/ml.

Echocardiographic studies

Standard transthoracic echocardiography was performed at baseline (24 h hours before the race) and within the first hour of arriving at the finish line by three experienced sonographers, with the subject at rest lying in the left lateral decubitus position. Each participant's baseline and post-race echocardiography was made by the same sonographer. Echocardiographic studies were carried out using a commercially available ultrasound system (VividQ; GE Medical; Milwaukee, USA). All studies were performed according to the guidelines of the American Society of Echocardiography (Lang et al. 2015). Parasternal (long and short axes) and apical (four, three, and two chambers) LV views, parasternal short axis at pulmonary artery level and RV apical views were obtained. For a better quantification of RV size, a dedicated four-chamber apical view focused on the RV was used.

2-dimensional pulsed-Doppler (PW) and tissue Doppler imaging (TDI) were also performed. Three cardiac cycles of each view were collected, digitally stored and later offline analyzed using an EchoPAC software (GE, Vingmed, Horten, Norway). All values were indexed for the body surface area of each patient.

The following parameters were measured in each individual at baseline and after the race: LV end-diastolic and end-systolic volumes, LV ejection fraction (using the biplane Simpson method), inter-ventricular septal thickness (IVS), posterior wall thickness (PWT), left ventricle mass, RV end-diastolic and end-systolic areas, relative apical and mid-basal RV areas and RV systolic function [assessed by fractional area change (RVFAC)]. Relative apical and mid-basal RV areas were measured by dividing the RV global area into two parts using the moderator band as a reference.

PW Doppler interrogation was performed to assess the mitral and tricuspid peak early (*E*) and late (*A*) inflow velocities. TDI was also performed to measure the mitral and tricuspid annular velocities [early (*E′*) and late (*A′*) peak lateral annular velocities]. The ratio between the time to peak ejection and the ejection time (TP/ET) of the RV outflow measured from the PW Doppler was used as a surrogate of the pulmonary artery pressure as most participants did not have an adequate tricuspid regurgitation signal [with higher ratios implying lower pulmonary artery pressures and lower ratios, higher pressures (López-Candales and Edelman 2012)].

Myocardial deformation was also assessed using speckle tracking (2D strain, EchoPac, General Electric Healthcare, Milwaukee, WI, USA). For the RV analysis, images were acquired from the apical four-chamber view; global RV peak systolic strain (RVGLS) was measured as an average of all six RV segments (three RV-free wall and three inter-ventricular septum segments). In addition, RV peak systolic segmental strains were measured in the basal (inlet), mid and apical segments of the RV-free wall. For the LV analysis, global LV peak systolic strain (LVGLS) was obtained as an average of the peak systolic strain assessed in the two-, three- and four-chamber view. Special care was taken to acquire images with an adequate frame rate (60–70 fps) and endocardial border delineation, including all the LV and RV segments in separate focused images. Those segments without a suitable tracking were excluded for the analysis.

The LV systolic volume (SV) was calculated using quantitative Doppler as the product of the LV outflow tract area and the velocity–time integral of flow at that level (Baumgartner et al. 2009).

Intra- and inter-observer intra-class correlations for strain measurements were performed in 10 athletes and were, respectively: 0.97 and 0.91 for LVGLS, 0.98 and 0.90 for RVGLS, 0.99 and 0.94 for RV basal segmental strain, 0.99 and 0.87 for RV mid segmental strain and 0.99 and 0.98 for RV apical segmental strain.

Statistical analysis

Statistical analysis was carried out using the SPSS Software version 22.0 (SPSS Inc., Chicago, Illinois, USA). The calculation of the sample size was based on a mean reduction of 5% between baseline and post-race right ventricular ejection fraction (4% typical deviation of the difference), assuming a statistical power of 90% and a statistical significance level of 5%. Values were expressed as mean + standard deviation (SD). All continuous variables were analyzed for normality of distribution using a Kolmogorov–Smirnov test. Subsequently, a paired Student *t* test was used to compare pre- and post-race data. If normality was not confirmed, Wilcoxon-signed rank test was applied. Bivariate correlational analysis was used for all variables that were significantly altered after the race to determine any relationship. A *p* value of less than 0.05 was considered indicative of statistical significance. Reproducibility was expressed by an intra-class correlation coefficient.

Results

Race, haemodynamic, and clinical characteristics

The Oceanman Palamós 9.5 Km open water swimming race took place on 24 May 2015. The air temperature was 21 °C and the water temperature was 16 °C. All 33 swimmers (26 men and 7 women, mean age 40.9 ± 7.2 years) finished the race without any incident. Table 1 shows the baseline characteristics, training level, and the mean time to complete the race, which was 174.0 ± 27.2 min. Baseline and post-race haemodynamic parameters are shown in Table 2.

Table 1 Baseline characteristics, training level, and time to complete the race

Parameter	*n*	*p* value
Age (years)	33	40.9 ± 7.2
BSA (kg/m^2)	33	1.9 ± 0.2
Indexed LV mass (g/m^2)	33	87.9 ± 32.4
Time mark of 1 km race (min)	31	15.2 ± 2.3
Training (days/week)	31	5.8 ± 3.1
Training (min/session)	31	82.4 ± 30.2
Training (km/week)	31	23.6 ± 30.4
Race completion time (min)	33	174.0 ± 27.2

BSA body surface area, *LV* left ventricle

Table 2 Hemodynamic characteristics and analytical data at baseline and post-race

Parameter	*n*	Baseline	Post-race	*p* value
HR (bpm)	33	63.2 ± 11.7	86.3±12.0	< 0.05
SBP (mmHg)	33	129.2 ± 15.4	110.9 ± 14.0	< 0.05
DBP (mmHg)	33	78.5 ± 9.8	69.7 ± 9.6	< 0.05
Urea (mg/dl)	33	37.8 ± 8.4	41.7 ± 8.3	< 0.05
Creatinine (mg/dl)	32	1.0 ± 0.1	1.1 ± 0.2	< 0.05
GFR (ml/min)	32	88.8 ± 13.9	81.9 ± 13.9	< 0.05
Sodium (mmol/l)	33	137.3 ± 1.2	140.9 ± 1.8	< 0.05
Leukocytes (x 10e9/l)	32	7.4 ± 1.8	13.8 ± 2.9	< 0.05
NT-ProBNP (0–125 pg/ml)	33	29.6 ± 18.2	79.6 ± 56.7	< 0.05
cTn I (0–30 ng/l)	33	5.6 ± 3.5	33.2 ± 32.1	< 0.05
CRP (mg/l)	33	1.0 ± 0.7	0.9±0.6	0.37

HR heart rate, *SBP* systolic blood pressure; *DBP* diastolic blood pressure; *GFR* glomerular filtration rate, *CRP* C-reactive protein

Baseline and post-race echocardiographic: parameters

Echocardiographic parameters are depicted in Table 3. LV wall dimensions were within normal limits. At baseline, indexed left ventricular end-diastolic volume was at the upper limit of normality and decreased significantly after the race.

No changes in any of the parameters of LV systolic function were observed after the race. A significant decrease in early peak mitral velocities and a significant increase in late peak mitral velocities were noted, resulting in lower *E/A* and *E′/A′* as well as an increased LV isovolumic relaxation time after the race.

The ratio TP/ET (a surrogate measure of the pulmonary artery systolic pressure) was unchanged after the race ($p = 0.395$). RV dimensions were within the normal limits at baseline; however, a significant increase was observed mainly at the expense of an increase in the area of the mid-basal part of the RV after the competition (Fig. 1). No significant changes in any of the parameters of RV systolic function were documented after the race, while a significant increase in *A′* of the tricuspid annulus was shown, resulting in lower *E′/A′* ratios.

Globally, RV systolic deformation (global and segmental) was unchanged after the race. However, when the individual response was analyzed, 8 swimmers (26.7%) showed impairment in RVGLS. We observed that at baseline, these athletes only differed from the rest in that they had larger areas of the apical segment of the RV and, after race, presented a larger dilatation of the basal segment.

The percentage change in RVGLS showed no correlation with age, gender, duration of the race or the amount of training hours.

Cardiac output (CO) is a result of a combination of HR and SV. In addition, SV is determined by myocardial deformation and cavity size (Bijnens et al. 2012). In consequence, the required increase in CO during exercise can be achieved by an increase in HR, an increase in cavity size or an increase in global deformation. Taking these considerations into account and to evaluate the acute RV remodelling, we analyzed the possible responses of participants according to the change in SV (ΔSV) after the race. Swimmers were divided into two groups according to the ΔSV after the race. Those participants who showed a decrease in SV after the race were classified in the first group ($\Delta SV \leq 0$) and those who showed an increase in this parameter were classified in the other group ($\Delta SV > 0$).

Table 4 shows the contribution of the changes in HR, RV size, and deformation to the required increase in CO during the race. Swimmers in the first group ($\Delta SV \leq 0$, $n = 16$) showed a decreased SV after the race (− 15.3 ± 10.4 ml) as a result of an insufficient cavity dilatation and a trend to lower RV deformation (a combination usually considered adverse remodelling), having to increase their CO by increasing HR. Swimmers in the other group ($\Delta SV > 0$, $n = 17$) increased SV after the race (16.9 ± 11.5 ml) through a larger increase in RV size. The change in SV did not correlate with dehydration parameters such as haematocrit ($r = -0.341$, $p = 0.065$) or serum sodium ($r = -0.078$, $p = 0.667$). Likewise, the change in SV did not correlate with prior training [evaluating frequency (days training session/week) $r = -0.068$, $p = 0.717$; intensity (minutes mark to swim 1 km) $r = 0.063$, $p = 0.734$; time (training minutes/week) $r = -0.117$, $p = 0.546$) or duration of race ($r = -0.061$, $p = 0.737$)].

In addition, Fig. 2 shows the individual response in HR (Fig. 2a), RV deformation (Fig. 2b), and size (Fig. 2c) before and after the race, with a high inter-individual variability.

Table 3 Baseline and post-race echocardiographic parameters

Parameter	*n*	Baseline	After race	*p* value
IVS (mm)	33	10.48 ± 1.50	10.50 ± 1.40	0.79
PWT (mm)	33	9.33 ± 1.49	9.3 ± 0.19	0.92
LVEDV (ml/m^2)	33	74.13 ± 14.76	67.74 ± 15.27	< 0.05
LVEF (%)	33	59.33 ± 4.14	59.81 ± 4.39	0.64
E peak velocity (m/s)	33	0.77 ± 0.15	0.65 ± 0.15	< 0.05
A peak velocity (m/s)	33	0.53 ± 0.16	0.62 ± 0.18	< 0.05
E/A ratio	33	1.49 ± 0.41	1,13 ± 0.38	< 0.05
Lateral mitral annulus E (cm/s)	30	11.57 ± 2.37	10.84 ± 2.71	< 0.05
Lateral mitral annulus A (cm/s)	30	6.67 ± 2.07	8.22 ± 2.79	< 0.05
E/A lateral mitral annulus	30	1.94 ± 0.85	1.63 ± 1.16	< 0.05
IRVT	20	74.26 ± 5.90	94.48 ± 16.36	< 0.05
LVGLS	32	20.68 ± 1.87	21.18 ± 2.20	0.08
RVEDA (cm^2/m^2)	33	15.12 ± 1.86	16.06 ± 2.27	< 0.05
RVEDA apex (cm^2/m^2)	33	3.36 ± 1.11	3.64 ± 1.23	0.23
RVEDA base (cm^2/m^2)	33	11.87 ± 1.87	12.54 ± 2.27	< 0.05
RVFAC (%)	33	47.13 ± 6.97	46.79 ± 5.26	0.76
E tricuspid flow (m/s)	31	0.45 ± 0.10	0.51 ± 0.09	0.21
A tricuspid flow (m/s)	31	0.32 ± 0.06	0.35 ± 0.09	0.07
E/A tricuspid flow (cm/s)	31	1.52 ± 0.25	1.52 ± 0.35	0.94
Lateral tricuspid annulus E (cm/s)	33	9.17 ± 2.30	9.09 ± 2.74	0.85
Lateral tricuspid annulus A (cm/s)	33	9.40 ± 2.76	12.55 ± 3.49	< 0.05
E/A tricuspid annulus (cm/s)	33	1.05 ± 0.38	0.76 ± 0.30	< 0.05
Lateral S tricuspid annulus (cm/s)	33	10.76 ± 1.55	11.23 ± 1.64	0.13
RVGLS (%)	29	22.52 ± 2.46	23.62 ± 3.25	0.09
Inlet RVLS (%)	29	24.52 ± 4.60	24.28 ± 5.61	0.71
Mid RVLS (%)	29	28.01 ± 3.52	28.07 ± 5.18	0.68
Apex RVLS (%)	29	30.06 ± 3.99	30.57 ± 4.36	0.77
FWRV strain (%)	29	27.55 ± 3.47	27.64 ± 4.45	0.67
Ratio TP/ET	31	0.40 ± 0.05	0.39 ± 0.07	0.40

IVS inter-ventricular septal thickness, *PWT* posterior wall thickness, *LVEDV* left ventricle end-diastolic volume, *LVEF* left ventricle ejection fraction, *IRVT* LV isovolumic relaxation time, *LVGLS* global left ventricle peak systolic strain, *RVEDA* right ventricle end-diastolic area, *RVFAC* right ventricle fractional area change, *RVGLS* global right ventricle peak systolic strain, *RVLS* right ventricle longitudinal strain, *FWRV* free wall right ventricle, *TP* time to peak velocity in the RV outflow, *ET* ejection time of the RV outflow

Baseline and post-race analytical results

Analytical parameters are given in Table 2. Leukocyte blood levels showed a significant increase after the race, suggesting a potential inflammatory response induced by exercise.

A significant increase in cTn I levels was confirmed after the race, with 13 (39.7%) swimmers increasing cTn I over the normal limits. In addition, a significant increase in NT-ProBNP concentrations was documented after the race, with 6 swimmers (18.2%) had NT-ProBNP levels above normality after the race.

There was no association between the increase in cTn I or NT-ProBNP and any parameter regarding training level, the time to complete the race, age or gender. A moderate correlation was found between the increase in leukocytes and the increase in cTn I (Fig. 3).

The release in cardiac biomarkers (NT-ProBNP and cTn I) did not correlate with any of the changes in structural and functional echocardiographic parameters of both ventricles.

Discussion

The current exploratory study provides a comprehensive evaluation of cardiac performance and cardiac biomarkers after a long-distance swimming race. To our knowledge, the effects of acute bouts of endurance swimming have never been examined before.

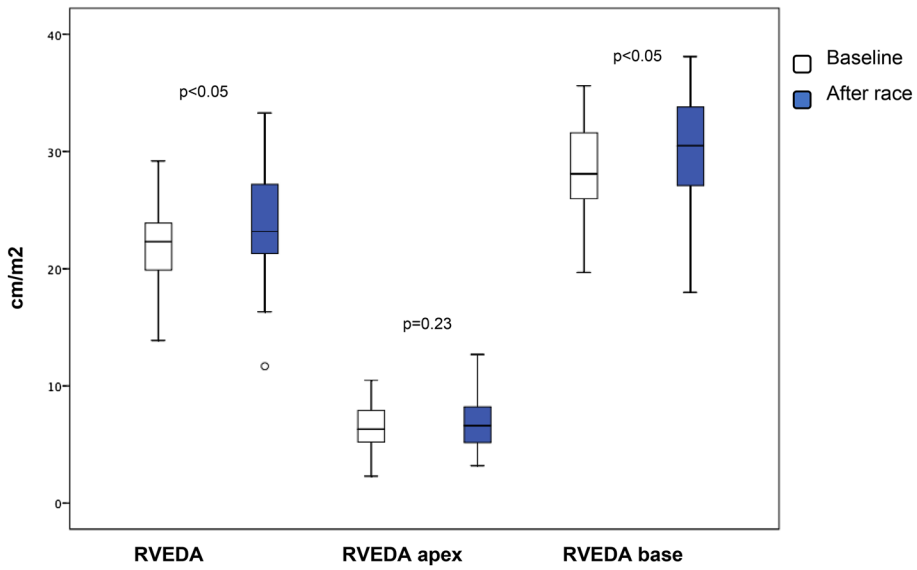

Fig. 1 Basal and post-race RV dimensions. RVEDA: RV end-diastolic area

Table 4 Contribution of heart rate, RV strain, and RV end-diastolic area according to the change in stroke volume after the race

Parameter	ΔSV ≤ 0 ($n = 16$)	ΔSV > 0 ($n = 17$)	p value
ΔHR (bpm)	36.97 ± 17.18	40.54 ± 23.75	0.63
ΔRV Strain	4.81 ± 16.31	7.03 ± 17.39	0.73
ΔRVEDA (cm/m^2)	3.14 ± 7.13	9.28 ± 9.25	< 0.05

Δ percentage change after the race, *SV* systolic volume, *HR* heart rate, *RV* right ventricle, *RVEDA* RV end-diastolic area

The study has three key findings: (a) globally, an acute RV dilatation was observed after swimming a long-distance race, while no significant changes in RV systolic function and deformation were documented; no changes were observed in LV performance after the race; (b) in a few athletes, an impairment in RV systolic deformation was observed after the race, suggesting a potentially poorer RV adaptation to exercise; this decrease in RV contractility was associated with larger baseline RV apical areas; and (c) an acute release in cardiac biomarkers (NT-ProBNP and cTn I) was demonstrated after the race, which did not correlate with changes in ventricular function, suggesting no functional myocardial damage.

After the race, only dilatation of the RV and not of the LV was observed, pointing to a disproportionate wall stress imposed by endurance swimming race, in accordance with prior evidence based in other sport disciplines. However, we could not confirm the previously described impairment in RV global systolic function (assessed by RVFAC) (Neilan et al. 2006a, b; Mousavi et al. 2009; Karlstedt et al. 2012) or in RV deformation after exercise (Knebel et al. 2009; Trivax et al. 2010; La Gerche et al. 2012; Sanz de la Garza 2016a, b). In addition, changes in diastolic filling of both ventricles that could be explained from the observed dehydration were seen after the swimming race.

Our data could not support the exercise-induced increase in pulmonary arterial pressure observed in the previous studies (Neilan et al. 2006a, b; Mousavi et al. 2009; Trivax et al. 2010). This increase in pulmonary arterial pressure has been established as a possible explanation for the increased RV wall stress and potentially for the acute RV impairment (La Gerche et al. 2011). It has been observed that swimmers, as compared to other athletes, have larger lung volumes as well as a better pulmonary function (with higher values of vital capacity, forced-vital capacity (FVC), forced expiratory volume for one second (FEV1) and FEV1/FVC), unrelated to anthropometric features or to training history (Sable et al. 2012; Lazovic-Popovic et al. 2016). This is anticipated, taking into account the specific characteristics of this sport discipline as compared to land-based sports: (1) swimming is performed in a horizontal position; (2) breath is held for prolonged periods, leading to intermittent hypoxia and alveolar hyperplasia; (3) ventilation is restricted underwater; (4) respiratory muscles have to work under greater pressure; and (5) swimmers use predominantly upper body muscles.

In addition, the previous authors have hypothesized that subjects with large lungs may have larger capacitance of the pulmonary vascular bed and thereby a lower increase in pulmonary arterial pressure with exercise (Thoresen et al. 2006). Considering all these aspects, circulatory response in swimmers is thought to be potentially different from land-based sports.

Nonetheless, an impairment in RV strain after the race was observed in 8 swimmers. These athletes had larger RV apical areas at baseline and presented larger dilatation of the basal segment after the race, which suggests a different pattern of adaptation to endurance chronic training in

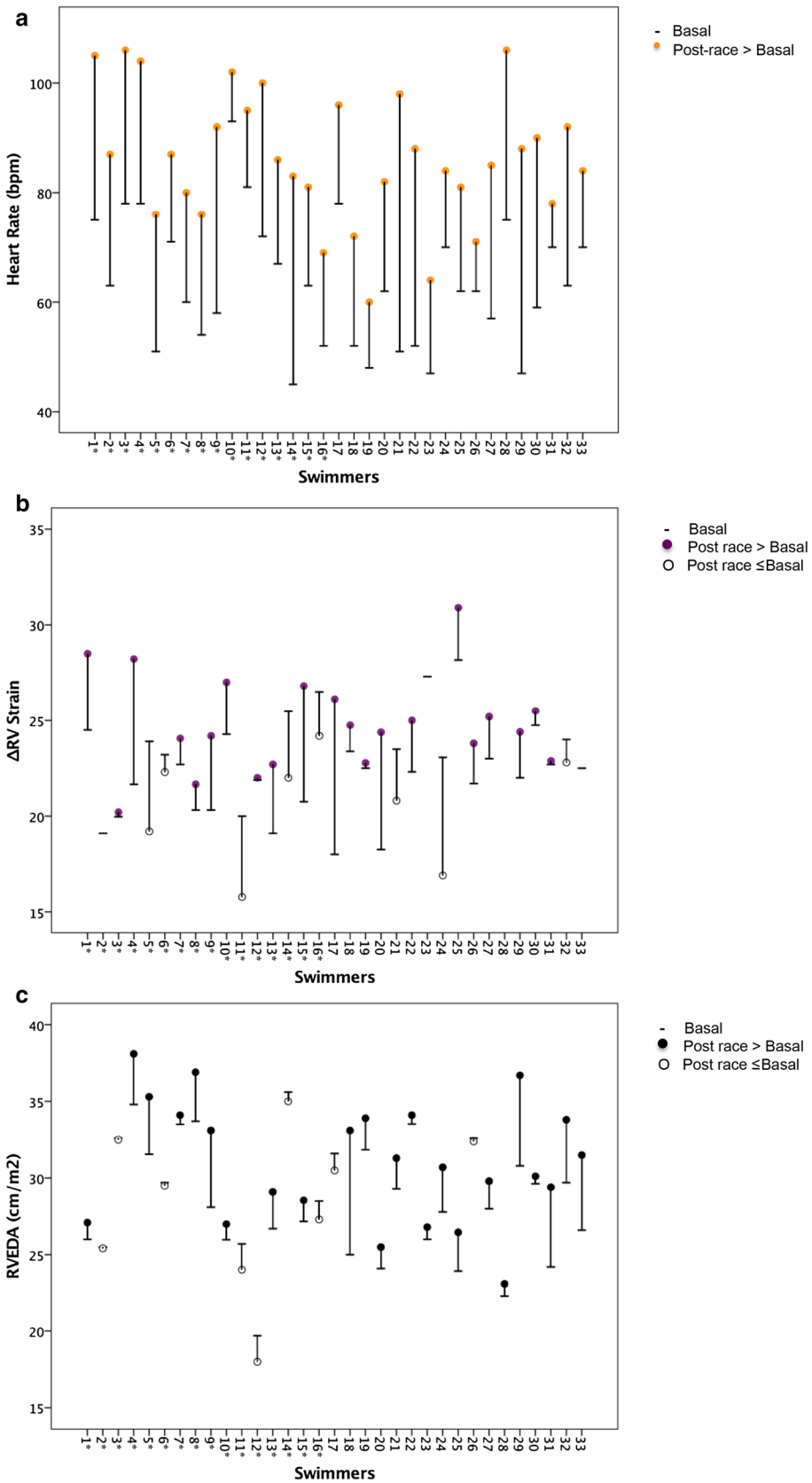

Fig. 2 **a** Individual response in heart rate before and after the race. Swimmers 1–16 (*) belong to group in which ΔSV is ≤ 0; swimmers 17–33 belong to group in which ΔSV is > 0. **b** Individual respons in RV strain before and after the race. Swimmers 1–16 (*) belong to group in which ΔSV is ≤ 0; swimmers 17–33 belong to group in which ΔSV is > 0. **c** Individual respons in RV end-diastolic area (RVEDA) before and after the race. Swimmers 1–16 (*) belong to group in which ΔSV is ≤ 0; swimmers 17–33 belong to group in which ΔSV is > 0

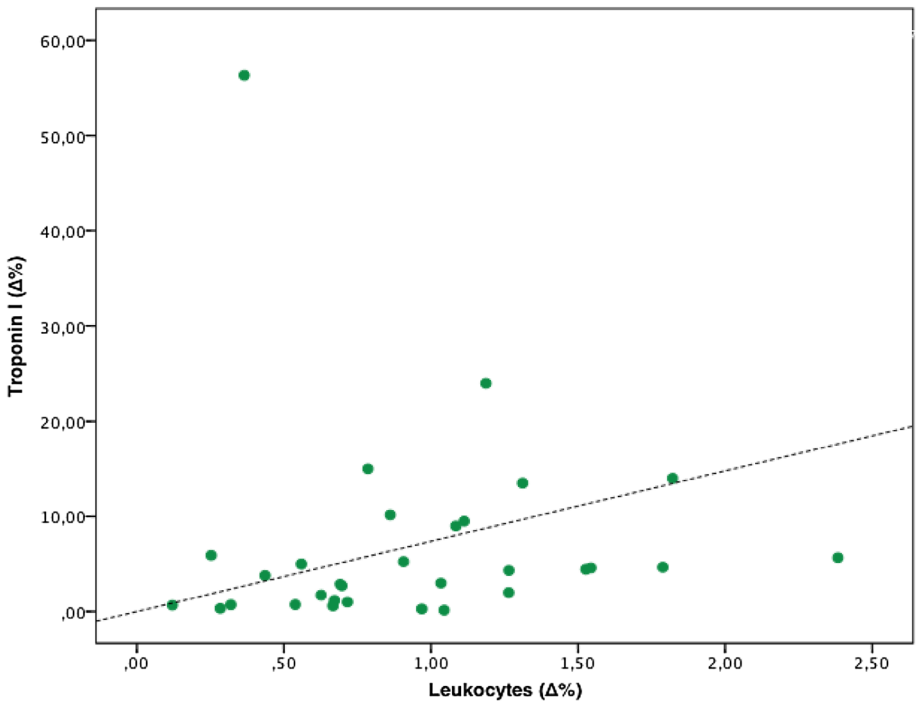

Fig. 3 Relationship between the increase in cardiac troponin I and leukocytes before and after the swimming race (Δ%) ($r = 0.375$, $p = 0.037$)

swimmers. This acute RV response showed a high inter-individual variability.

The required increase in cardiac output during exercise can be achieved by a combination of an increase in HR, ventricle dilatation or an increase in global deformation. In our study, two groups of swimmers were identified according to the change in SV after the race: those who were able to increase CO by increasing SV mainly at the expense of larger RV dilatation, and those who could not increase SV due to lower RV dilatation and had a trend to lower RV deformation, and need to increase HR. This different pattern of response to exercise also exhibits a high inter-individual variability.

In contrast, prior evidence based on terrestrial sports has also established a relationship between the RV impairment observed after acute bouts of endurance exercise with a release in cardiac biomarkers suggesting potential myocardial damage (Neilan et al. 2006a, b; La Gerche et al. 2011). Nevertheless, the true significance and the pathophysiological mechanisms underlying this increase of cardiac biomarkers are controversial (Shave et al. 2010). Middleton et al. (2008) showed that cardiac troponin T is released with a biphasic pattern in all subjects after completing a marathon on a treadmill, possibly representing a physiologic response and not actual myocardial damage. In addition, the kinetics and the level of cardiac troponin released after endurance exercise are not the same as those observed with ischemic injury (Neumayr et al. 2001). Accordingly, the previous studies using contrast-enhanced magnetic resonance have failed to correlate the increase in cardiac troponin with persistent myocardial dysfunction or fibrosis (Mousavi et al. 2009; Trivax et al. 2010; Karlstedt et al. 2012).

The previous studies have also shown that prolonged exercise induces an inflammatory process and oxidative stress which leads to leukocytes mobilization, release of cytokines and production of free radicals (Wilkerson et al. 1977; Natale et al. 2003; Zaldivar et al. 2006). No significant change in C-reactive protein (CRP) levels after swimming was observed, but the levels of this inflammatory marker have been proved not to have a consistent response to endurance exercise and multiple physiologic factors (such as carbohydrate ingestion or shifts in blood volume) or the time point of CRP assessment may influence the results (Shave et al. 2007a). Although there was no fluid restriction during the competition, a dehydration status was observed after the race, given the increase in sodium, creatinine, and urea as well as the decrease in GFR.

In our study, no association between the increase in cTn I and cardiac performance was found, but an association was observed with the increase in white blood cells count after the race. This relationship was weak but raises the previously described alternative hypothesis related to an increased membrane permeability due to the inflammatory status induced by strenuous exercise as the cause of the release in cTn I rather than true myocardial damage (Shave et al. 2010). Regarding changes in NT-ProBNP, we did not find any association with cardiac performance or with diastolic function. La Gerche et al. (2010) observed that BNP increases post-exercise correlated with pulmonary artery systolic pressure, which suggests that this biomarker may

be secreted from the RV during exercise due to the acute increases in afterload. Indeed, it was suggested that LV loading is not the main determinant for the exercise-induced rise in BNP. Our data showed a trend to the association between the change in NT-ProBNP levels and estimates of pulmonary artery pressure after the swimming race.

Limitations

This exploratory study was carried out in a small sample size limiting the statistical power and generalizability of the results. We cannot exclude some possible gender differences in cardiac performance during endurance swimming as only 21% of the swimmers were women. Our data were obtained after a 9.5 km swimming competition and might not be extended to other distances races. Therefore, further studies with a larger athlete population and different distances races are needed to better understand the cardiac effects of endurance swimming.

Heart rate could not be determined during exercise due to the characteristics of the competition, which is performed in the open sea. Measurements were, therefore, taken within the first 15 min of reaching the arrival point and do not correspond to maximum heart rate. Similarly, echocardiographic parameters refer to the ventricular performance immediately after exercise.

Biomarkers were only acquired immediately after race, limiting the assessment of the exercise-induced changes in CRP, cTn I and NT-proBNP, that typically reach the maximum peak between 3 and 24 h after exercise. Recent evidence has observed that gravitational gradients could modify certain echocardiographic parameters, including a significant reduction in global longitudinal strain with increasing gravitational stress. Given that echocardiographic studies were performed outside the sea, without the hydrostatic pressure, we do not think that it can account for the results obtained in our population. Finally, the pulmonary artery pressure was indirectly assessed using a surrogate variable, because most of the participants had inadequate Doppler signals of tricuspid regurgitation. Additional research using a more accurate system to evaluate the response of pulmonary vascular circulation to exercise is also needed to better understand the RV arterio-ventricular coupling in swimmers.

Conclusions

To our knowledge, this is the first study that assesses RV performance after an endurance swimming race. The previous evidence, based mainly on terrestrial sports, has observed an acute release in cardiac biomarkers and a transient RV dysfunction after endurance exercise. The present study demonstrates that a long-distance swimming race promotes an acute RV dilatation but without impairment in global RV systolic function. An acute release in cardiac biomarkers after the race was also demonstrated which did not correlate with ventricular function response to exercise suggesting no real myocardial damage. Indeed, the observed relationship between the release in leukocytes and cTn I after the race supports the theory of an exercise-induced inflammatory state that would lead to an increase in membrane permeability and may ultimately explain the increase in cardiac biomarkers rather than a real myocardial damage. Our findings could help to understand cardiac adaptation to exercise and to identify those athletes with potentially better or worse RV remodelling.

Author contributions VM conceived and designed the study; VM, MSG, and GG performed the echocardiographic studies; GG, PC, BG, and AC conducted the blood sample extractions; JT processed the blood samples; VM analyzed the data and interpreted the results; MSG, BB, and MS contributed to the interpretation of the results; VM wrote the manuscript; and MS supervised the study. All authors read and approved the final manuscript.

Data Availability The data sets generated or analyzed during this study are available from the corresponding authors on reasonable request.

Compliance with ethical standards

Conflict of interest The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Human right and animal participants All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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