

THE EFFECTS OF A SOCCER MATCH ON CARDIAC TROPONIN I LEVELS IN MALE AMATEUR SOCCER PLAYERS

Marco Alfonso Perrone^{1,2}, Massimo Pieri³, Bruno Donatucci², Federica Spolaore⁴, Francesco Romeo¹, Sergio Bernardini^{2,3}, Ferdinando Iellamo^{2,5}

1. Department of Cardiology, University of Rome Tor Vergata, Rome, Italy

2. University Sports Centre, University of Rome Tor Vergata, Rome, Italy

3. Department of Clinical Biochemistry and Clinical Molecular Biology, University of Rome Tor Vergata, Rome, Italy

4. Department of Laboratory Medicine, Santa Chiara Hospital, Trento, Italy

5. Istituto di Ricovero e Cura a Carattere Scientifico San Raffaele Pisana, Rome, Italy

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ABSTRACT

Cardiac troponins are the reference markers for the diagnosis of acute coronary syndromes. However, recent studies have shown that healthy professional athletes have a significant increase in cardiac troponins after intense physical activity. Conversely, there are few studies on the evaluation of cardiac troponins in amateur athletes. The aim of this study was to evaluate cardiac troponin I in amateur soccer players after a soccer game. For the study, 22 amateur soccer players from the University Sports Centre of the University of Rome Tor Vergata were enrolled. The blood samples were collected before the start of the soccer game, immediately at the finish, 3 hours and 24 hours from the time the game ended. The data showed a significant increase of serum troponin concentrations immediately after the game ended and after 3 hours ($p < 0.001$) and then a return to baseline levels after 24 hours ($p < 0.001$). Our study demonstrated a significant increase in cardiac troponin I in amateur soccer players, without any evidence of cardiac damage. Further studies with more athletes will be needed to confirm these data.

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1. Introduction

Since their discovery, cardiac markers have gradually gained greater clinical relevance in the diagnosis, treatment and prognosis of patients with cardiovascular diseases. In particular, the most recent international guidelines recommend the use of cardiac troponin I (cTnI) and T (cTnT) in the differential diagnosis of acute coronary syndromes (1,2). To confirm this, in 2018 the Fourth Universal Definition of Myocardial Infarction document established that cTnI and cTnT are the biomarkers of choice for the assessment of myocardial damage and the diagnosis of myocardial infarction (3). Cardiac troponins are regulatory proteins that control the calcium-mediated interaction of actin and myosin, which causes contraction and relaxation in striated muscles.

The troponin complex comprises three subunits: troponin C, which binds calcium; troponin I, which inhibits actin-myosin interactions; and troponin T, which binds the troponin complex by binding to tropomyosin and facilitates contraction (4, 5). While troponin C is expressed by cardiac and skeletal muscle cells, troponins I and T are unique to the heart muscle. This difference allowed the development of diagnostic kits to detect cardiospecific troponins T and I in the blood (6, 7). However, while on the one hand the dosages of cardiospecific biomarkers such as troponins I and T have provided answers to fundamental pathophysiological and clinical questions, they have also opened up new avenues for experimental and clinical research. In fact, in recent years, several authors have evaluated cardiac troponins in healthy athletes during sport and exercise (8 -10).

* Corresponding author: Marco Alfonso Perrone, marco.perrone@uniroma2.it

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Physical activity is globally recognized as one of the fundamental factors of a healthy lifestyle and most exercise and sport is practiced by amateur or non-professional athletes (11,12). However, in the literature, most studies are based on professional athletes. Indeed several authors have shown that in professional athletes there is a significant increase in cardiac troponins after intense exercise, sometimes with values above the upper reference limit (URL) (13,14). Conversely, there are few studies on the assessment of cardiac troponins in amateur athletes in different sports. Nie et al. for example, investigated cardiac troponin I and T in adolescent basketball players after a game and found that cardiac biomarkers increased during physical activity and returned to baseline after 24 hours (15). Soccer is certainly among the various sports most practiced in the world. It is estimated that around 4% of the world population plays soccer (16). Despite this there are few data in the literature on the assessment of cardiac troponins in amateur soccer players and consequently the effects of amateur soccer on troponin levels in these athletes are not yet fully understood. The aim of this study was to evaluate cardiac troponin I levels in adult amateur soccer players of the soccer team of the University Sports Centre of the University of Rome Tor Vergata after a soccer match.

2. Material and methods

For the study, 22 male amateur soccer players from the University Sports Centre of the University of Rome Tor Vergata were enrolled. To be eligible, athletes had to avoid any form of exercise during the 48 hours prior to the game. Each player was in possession of the medical-sports certification, issued by the Sports Medicine and Cardiology Centre of the University of Rome Tor Vergata. Medical history, routine biochemical tests, clinical visits and electrocardiograms were normal for every soccer player. All the athletes followed the same training loads. The characteristics of the soccer players are shown in Table 1. The blood samples were collected before the start of the soccer game, immediately at the finish, 3 hours and 24 hours from the time the game ended. Blood samples were quickly centrifuged at 3000 g for 15 min and stored at -80 °C for further analysis in the Department of Laboratory Medicine, University of Rome Tor Vergata. Cardiac Troponin I was determined using a troponin I immunoassay in a Dimension Vista Siemens platform (Siemens Healthcare GmbH, Marburg, Germany). The study was approved by the local ethics committee and all athletes signed an informed consent. The study was conducted in accordance with the Declaration of Helsinki. The non-parametric Kruskal-Wallis test was used to assess the difference on cardiovascular biomarker levels at each time analyzed, supplemented by Dunn's multiple comparison test. Statistical analyses were performed by using SPSS statistics (SPSS 21.0: IBM Corporation, Armonk, NY, USA). All p-values <0.05 were considered statistically significant.

N= 22	Mean	Standard Dev.
Age (y)	22.09	± 1.56
Height (cm)	170.45	± 11.07
Weight (kg)	67.81	± 11.35
BMI (kg/m ²)	23.28	± 2.43

Table 1. Physical characteristics of the soccer players enrolled.

3. Results

All athletes completed the game in the regular 90 minutes, without substitutions. No athlete had health problems during or after the game. Figure 1 shows the troponin I levels pre, post, 3 hours and 24 hours after the game. The data showed a significant increment of serumtroponin concentrations immediately after the end of the game and after 3 hours ($p<0.001$) and then a return to baseline levels after 24 hours ($p<0.001$). There was no significant difference in blood values between the various roles on the pitch (attacker, midfielder and defender). Goalkeepers were excluded from the analysis. The results showed a significant increase in troponin I after physical exertion, but no athlete had values above the URL (0.045 ng/ml).

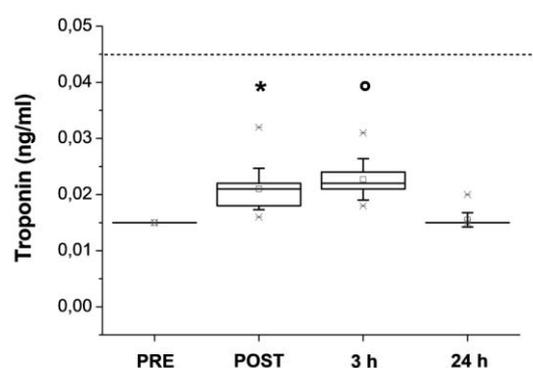


Figure 1. Serum troponin I levels 22 amateur soccer players before, immediately after, 3 and 24 hours from the time the soccer game ended. The upper reference value is indicated by the horizontal line (0.045 ng/ml). The box-plot indicates 25 and 75 percentile, whisker indicates standard deviation, the square indicates mean and the line in the box the median. 'x' indicates individual values for 99 and 1 percentile. * indicates significantly different from pre, 3h and 24h ($p<0.001$). ° indicates significantly different from pre, post and 24h ($p<0.001$).

3. Discussion and conclusions

The aim of this study was to evaluate whether, even during a soccer game, there was a significant increase in cardiac troponin I levels in amateur soccer players. Our data showed that immediately after the end of the game and 3 h later there is a significant increase in cardiac troponin I levels from baseline. However, cardiac troponin I levels are not above the URL in any soccer player and returned to baseline after 24 h. In the literature many authors have shown that after intense physical effort in professional athletes there is a significant increase in cardiac troponin levels, sometimes even above the URL (13,14,17). The finding of these "pathological" values of troponin and the effects of intense physical effort on the heart of professional athletes is currently the subject of scientific debate.

Some authors have described this phenomenon as a transient myocardial ischemia that occurs constantly after intense acute aerobic exercise and this explains high concentrations of cTnI, also combined with reduced renal clearance (18-20). However, Hanssen et al. showed that marathon runners after the race had a significant increase in cTnT and echocardiograms indicated alterations of diastolic filling (21). However, authors found no evidence of left ventricle late gadolinium enhancement (LGE) during cardiac magnetic resonance imaging (CMR). In addition, magnetic resonance tagging demonstrated unaltered radial shortening, circumferential and longitudinal strain. Thus, this evidence suggests that although marathon running leads to a transient increase of cTnT, no detectable myocardial necrosis was observed as evidenced by LGE-CMR. Also Trivax et al. (22) showed that healthy, middle-aged marathon runners, after the race, had a significant increase in cTnI, which does not appear to be caused by ischemic injury to any cardiac chamber. In fact, the authors demonstrated that marathon running caused dilation of the right atrium and right ventricle, a reduction of right ventricular ejection fraction, but there were no morphological changes observed in the left atrium or left ventricle or evidence of ischemic injury to any chamber by LGE-CMR. Furthermore, there were no significant arrhythmias.

If, on the one hand, there are several studies on the role of cardiac troponins in professional runners, or in general in endurance athletes, surely there is much less evidence in soccer players. Franchis-Gomar et al. (23) have shown that even professional soccer players after a match have a significant increase in the cTnT, with values above the URL in some athletes. Instead, our study has shown that even in adult amateur soccer players, there is a significant increase in cTnI levels after a match, but no value is above the URL. Furthermore, the cTnI levels returned to baseline after 24h. Our data are in agreement with those of Hosseini et al. who reported a return to baseline cTnI levels 24 h after a soccer game in adolescents (24).

Several hypotheses were put forward to explain the pathophysiology of troponin increase after physical exercise. Since the current knowledge of cell biology of cardiomyocytes has led to definitely ruling out that measurable amount of troponins in the blood after physical exercise would reflect a necrotic and clinically harmful myocardial injury, it is now assumed that they would instead reflect an increased cell membrane permeability and early troponin release ("leakage") from the unbound, cytosolic pool, which represents nearly 3-8% of the total cardiac troponin content (9,25,26). This hypothesis is supported by the fact that no evidence of ischemic injury by LGE-CMR could be detected (21,22). Furthermore, in trained athletes and healthy subjects free of cardiovascular alterations, the cardiac troponin concentrations usually return within the normal range within the 24-48 hours after physical endurance exercises and the increase in the biomarker is only moderate (27,28).

On the contrary, the complete kinetic curve of cardiac troponin concentrations in patients with acute coronary syndromes is longer (from 4 to 10 days) and the increase in biomarker is significantly higher (1,3,29).

Our study has therefore shown that the increase of cTnI in adult amateur soccer players is a physiological response to physical effort, without any evidence of myocardial damage. To our knowledge, there are no similar studies in the literature of adult amateur soccer players using our cTnI dosage methods.

Our data showed that after a 90 min physical effort during a regular soccer match, amateur soccer players have a significant increase in cTnI, as a physiological response to physical activity, without any evidence of cardiac damage. Further studies with more athletes will be needed to confirm these data.

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