Letter to the Editor

Hs-cTnT levels in professional soccer players throughout a season: No evidence of sustained cardiac damage

Fabian Sanchis-Gomar a,⁎, Rafael Alis b,c,1, Ermanno Rampinini d, Andrea Bosio d, Marco Romagnoli a,e, Giovanni Lombardi f, Giuseppe Lippi g

a Research Institute of the Hospital 12 de Octubre (‘i + 12’), Madrid, Spain
b Research Institute ‘Dr. Vílchez Gimeno’, Molecular and Mitochondrial Medicine, Catholic University of Valencia San Vicente Mártir, Valencia, Spain
c School of Medicine, Catholic University of Valencia San Vicente Mártir, Valencia, Spain
d Human Performance Laboratory, MAPED Sport Research Center, Italy
e Laboratory of Experimental Biochemistry & Molecular Biology, I.R.C.C.S. Istituto Ortopedico Galeazzi, Milano, Italy
f Department of Physical Education and Sports, University of Valencia, Valencia, Spain
g Laboratory of Clinical Chemistry and Hematology, Academic Hospital of Parma, Parma, Italy

A R T I C L E   I N F O

Article history:
Received 15 June 2015
Accepted 26 June 2015
Available online 30 June 2015

Keywords:
Troponins
Exercise
Football
Training

Olah et al. have recently shown adverse effects of exhaustive exercise on the heart in a murine model [1]. The authors showed elevated plasma levels of cardiac troponin T and creatinine kinase after exhaustive exercise, compared to controls, along with elevated nitro-oxidative stress, myocardium enhanced apoptotic signaling and dysregulation of the matrix metalloproteinase system [1]. In this line, Legaz-Arrese et al. have investigated the influence of a mild endurance training program (14 weeks running, 3–4 days/week, 120–240 min/week, 65–85% of maximum heart rate) on exercise-induced increase of high-sensitivity cardiac troponin T (hs-cTnT) in humans, showing that both baseline and post-exercise hs-cTnT values after training were higher compared with pre-training and the response of the control group (P = 0.008) [2].

Cardiac troponins (cTn) are released into the circulation after myocardial ischemia and are currently considered the preferred biomarkers for diagnosing acute coronary syndrome and cardiac stress [3]. In the past years several studies also showed that prolonged exercise induces elevation of circulating cTn, probably reflecting increased myocardial membrane permeability and mild-transient cellular damage [4]. Detectable levels of cTn are more frequent in running than in cycling or triathlon [5]. Moreover, the exercise-induced release of cTn in a running trial depends on exercise intensity [6]. Because of these findings, controversy remains as to whether exhaustive exercise (i.e., half-marathon, marathon or extensive endurance training) may be a suitable exercise setting to prevent cardiovascular risk and counteract sedentary lifestyle in the general population [7]. The recent findings by Olah et al. seem to further reinforce the idea that, from the heart point of view, exhaustive exercise should be avoided.

We previously demonstrated that professional soccer players exhibit increased values of cardiac damage biomarkers after playing a competitive match [8]. This outcome is probably attributable to oxidative stress occurring during exercise, since allopurinol treatment was effective to lower the impact of competitive match on cTn [8]. The accumulation of successive efforts in professional soccer players could lead to the deleterious effects on myocardium.

To further clarify this issue, we recruited 15 male professional soccer players from an Italian major league (i.e., Serie A) team (age (mean ± SD) 27 ± 5 years, weight 76.9 ± 4.1 kg, height 1.82 ± 0.05 m), whose blood was collected 3 times during the last part of a competitive season (January, March and May) as well as at the beginning of preseason training (i.e., July). The full description of the study protocol is available elsewhere [9]. All participants were informed of the purpose, protocol, and procedures of the study before agreeing to participate. The study complies with the World Medical Association Declaration of Helsinki regarding ethical conduct of research involving human subjects and/or animals, and was approved by the ethics committee of University of Valencia, and by the soccer clubs involved.

Blood samples were drawn from an antecubital vein in fasting conditions, between 8–9 AM, into evacuated blood tubes spray coated with clot activator (SSTII Advance Vacutainer, BD, Franklin Lakes, NJ, USA). The samples were allowed to clot for 60 min, and then centrifuged at 3000 g for 10 min at room temperature. After centrifugation, serum was immediately aliquoted and frozen at − 80 °C. In all samples, serum high-sensitivity (hs) cardiac troponin T (cTnT) concentration was measured using Roche Elecsys 2010 (Roche Diagnosis GmbH, Penzberg, Germany). The detection limit for this test is 0.01 ng/mL.
Undetectable hs-TnT values were observed in all players except 4 in January, 2 in March and 2 in May (Fig. 1). After the detraining period, no player displayed detectable levels (Fig. 1). Moreover, 2 players exhibited detectable levels during the competitive season (player 4: 0.04 ng/mL, 0.06 ng/mL and 0.03 ng/mL; player 5: 0.01 ng/mL, 0.01 ng/mL and 0.01 ng/mL, in January, in March and in May, respectively).

The efforts of professional players in soccer matches are characterized by a 45 + 45 min continuous running at low/mild intensity, with short burst of high activity, all combined with frequent changes of direction. Therefore, it is not surprising that the values of cTn may increase after a soccer match [8], since this type of physical engagement is similar to other types of exercise that were found to be associated with cardiac biomarkers elevation [6]. However, despite the fact that professional soccer players are highly trained subjects, we failed to find a baseline elevation of hs-cTnT throughout the end of a competitive season. Therefore, accumulating efforts in professional soccer players do not seem to elicit a persistent cardiac damage, as would be reflected by the presence of cTn values continuously exceeding the detectable limit of the hs cTnT assay.

These findings support the conclusion that elevations of cTn induced by exercise do not probably originate from irreversible myocardial injury in healthy subjects, and that efforts are not per se the cause of sustained cardiac damage. Also in this line, in a previous study, we failed to find differences in both right ventricular function indicators after comparing former elite endurance athletes to age and gender-matched non-athletic controls [10].

Altogether, the data of the present study further reinforce the notion that the transient mild increase of cTn after prolonged or exhaustive exercise would reflect an adaptive response of cardiomyocytes to the physiological milieu induced by such exercise rather to persistent cardiac damage.

**Competing financial interests**

The authors declare no competing financial interests.

**Acknowledgments**

This research has been supported by grant DEP2012-37494 from the Spanish Government and by grants 2013-168-002 and 2012-011-001 from Catholic University of Valencia. RA is a predoctoral fellow of Catholic University of Valencia.

**References**