

# Elevated serum high-sensitive cardiac troponin T in adolescent runner: exercise or something else?

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#### Abstract

It has been reported that prolonged strenuous exercise increases cardiac biomarkers probably due to transient strain, ischemia, inflammation, or metabolic causes. However, in emergency situation the possible increase of troponin serum concentrations in a young runner may lead to a suspicion of an acute myocardial damage. We report a case of elevated high-sensitive troponin T (hs-cTnT) in a half-marathon runner who had a transient loss of consciousness after a race associated with an increased serum levels of hscTnT without cardiac damage. Physicians must be able to interpret hs-cTnT serum concentrations using clinical expertise especially in an emergency situation.

### Introduction

It has been reported that prolonged strenuous exercise increases cardiac biomarkers probably due to transient strain, ischemia, inflammation, or metabolic causes.<sup>14</sup> However, in an emergency situation the possible increase of troponin serum concentrations in a young runner may lead to a suspicion of an acute myocardial damage. Thus, we report a case of an elevated high-sensitive troponin T (hs-cTnT) in an half-marathon runner who had a transient loss of consciousness after a race associated with an increased serum levels of hs-cTnT.

#### **Case Report**

A 19-year-old African male, half marathon runner that was always in good health, had a transient, self-limited loss of consciousness with an inability to maintain postural tone followed by spontaneous recovery at the end of a footrace of 21 km.

His past medical history was unremarkable



and he was not a smoker or a drug addict; he never used medications and he had no cardiovascular risk factor. Regarding the family history, the father was a half marathon runner as welland had several episodes of loss of consciousness that were not clinically studied, one of the episodes happened at the end of a race; the brother suffered fromepilepsy.

On admission to the Emergency Room the patient was asymptomatic and the vital signs were normal; he did not exactly remembered what happened. The physical examination was normal apart of sticky mouth and dry skin.

Electrocardiography showed a sinus rhythm with a heart rate of 86 beats per minute, left ventricular hypertrophy, T wave inversion in inferior leads and biphasic-T wave from V3 to V5 (Figure 1). Chest X-ray was normal as well as the CT of the head. Blood examination revealed increased serum concentrations of cTnT that were 137 ng/L (upper reference limit: 14) as well as an increased serum levels of AST 61 U/L (upper reference limit: 38) and ALT 50 U/L (upper reference limit: 41) and serum creatinine of 1.6 mg/dL (reference range 0.5-1.2). After 3 h, the second blood examination of hs-cTnT was 290 ng/L and after 16th h, it had decreasesd to 61 ng/L. Microbiological analyses were all negative.

Echocardiogram carried out in an emergency showed a marked concentric hypertrophy of the left ventricle (tele-diastolic/telesistolic ventricular diameter 4.8/2.7 cm; telediastolic interventricular septum 1.6 cm; left ventricular ejection fraction 68%), left ventricular myocardial non-compaction [ratio of non-compacted to compacted (NC/C) myocardium>2] Correspondence: Bahjat Barakat, Department of Emergency Medicine, S. Orsola-Malpighi Hospital, via Massarenti 9, 40138 Bologna, Italy. Tel/Fax: +39.051.636 3687. E-mail: bahjat.barakat@aosp.bo.it

Key words: troponin T, exercise, adolescent runner.

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and no district kinetic deficits, no outflow obstruction at rest, no significant valvulopathy.

The patient was hospitalized, and during the hospital stay, he was hydrated with an intravenous fluid administration and remained asymptomatic. There were no arrhythmic events monitored by the electrocardiography (ECG). The exercise test was also negative for myocardial ischemia by stress, in the absence of significant arrhythmias. A second echocardiogram, after 3 days of hospitalization showed a framework compatible with the athlete's heart. On ECG the T-wave inversion, previously present, disappeared (Figure 2). Finally, the genetic analysis for myocardial hypertrophy major genes was negative.

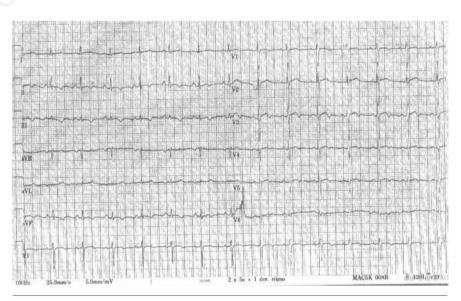


Figure 1. Electrocardiography carried out on admission showing a sinus rhythm with heart rate of 86 beats per minute, left ventricular hypertrophy, T wave inversion in inferior leads and biphasic-T wave from V3 to V5.



The patient was discharged without any specific therapy and a magnetic resonance imaging (MRI) performed 2 months later showed a mild concentric left ventricular hypertrophy with a diastolic maximum thickness of 11 mm (interventricular septum); left ventricular size was of a normal competitive sports activities upper limit; spongy aspect of the left ventricular myocardium that did not reach the MRI criteria for a myocardial noncompaction (Figure 3).

## Discussion

Serum hs-cTnT is considered to be a marker of a myocardial cell damage during or after exercise.<sup>5-7</sup> From the first report of Mair *et al.*<sup>8</sup> other studies did not confirm an increase of troponin T after exercise;<sup>9-12</sup> others confirmed different percentages in the relationship between elevated cardiac troponine T and physical exercise without myocardial dam-

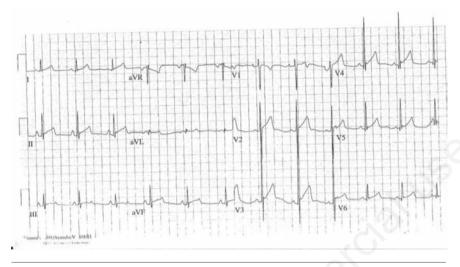


Figure 2. Electrocardiography carried out after three days from admission shows the disappearance of the T-wave inversion previously present.

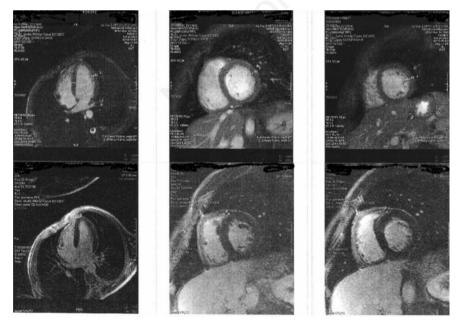


Figure 3. Magnetic resonance examination performed 2 months later after deconditioning showing a mild concentric left ventricular hypertrophy with diastolic maximum thickness of 11 mm (interventricular septum).

age,<sup>2,6,13-22</sup> whereas others<sup>9,18,23-27</sup> reported that an increase of troponin T concentration may be partially attributed to a myocardial damage during the race.

The mechanisms that could explain this situation vary: transient mechanical strain of cardiomyocytes, myocardial cell necrosis, even if this explanation is supported by convincing data, other causes such as inflammation or the presence of prolonged dehydratation as evidenced by the value of creatinine and hematocrit of the patient performed in First Aid.<sup>28-31</sup>

In our case, we hypothesized that there was an episode of tachyarrhythmia in patients with hypertrophic cardiomyopathy. In fact, high serum concentrations of troponin T have been reported in subjects having hypertrophic cardiomyopathy characterized by inappropriate hypertrophy, small-vessel coronary artery disease, myocyte alteration, and increased interstitial fibrosis; elevated concentration of troponin T could be a reliable indicator of myocardial remodeling, a proposed prognostic marker in hypertrophic cardiomyopathy.<sup>32</sup> This assumption was not confirmed by the various examinations carried-out and, in addition, echocardiography showed the regression of hypertrophy after two months of detraining. Thus, we believe that increased serum levels of hs-cTnT may be due to dehydration.29-31

#### Conclusions

In conclusion, physicians must be able to interpret hs-cTnT serum concentrations using clinical expertise especially in an emergency situation.<sup>33</sup>

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