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Antonio J. Romero-Puche,[a,*] Natalia Trigueros-Ruiz,[b] M. Carmen Cerdán-Sánchez,[a] Fernando Pérez-Lorente,[a] Diego Roldán,[a] and Tomás Vicente-Vera[a]

[a]*Servicio de Cardiología, Hospital General Universitario Reina Sofía, Murcia, Spain*
[b]*Servicio de Urgencias, Hospital General Universitario Reina Sofía, Murcia, Spain*

* Corresponding author:
E-mail address: antoniojoserp@hotmail.com (A.J. Romero-Puche).

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Cardiac Troponin I Increases in Female Adventure Racers

Elevación de la troponina cardiaca I en corredoras de raids de aventura

To the Editor,

Although exercise can reduce the incidence of cardiovascular disease by between 33% and 50%,[1–3] intense or prolonged exercise can increase cardiac troponin (cTn) levels in individuals with no coronary obstruction.[4] Understanding the significance of elevated cTn could prevent unnecessary or invasive procedures in athletes. The objective of this study was to determine the behavior of cardiac troponin I (*c*TnI) in women participating in adventure races.

In the Women International Adventure Raid, participants cover a distance of 80 km with an elevation gain of 2600 m. The race includes sections of swimming, running, and cycling. Before the race, study participants completed an interview to collect data on age, weight, height, body mass index (BMI), toxic habits, medical history, medications, weekly training time, and nutrition. Blood tests were also performed.

Data collected after the event included the contestants' race time and any symptoms appearing during it. A second blood test was performed and glucose, cholesterol, triglyceride, high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), creatine kinase (CK), and serum cTnI levels before and after the race were measured. An increase in cTnI was defined as ≥0.04 ng/mL.

Blood test results were compared using non-parametric tests. A logistic regression analysis was used to analyze the influence of different variables on elevated troponin levels.

Of the 50 riders who entered the race, 34 (68%) participated in the study. Median age was 32.5 [30-35.25] years; BMI was 21.44 [20.28-22.34]. Median weekly training and race times were 8.5 [5.37-12] h and 618 [610-629.25] min, respectively.

None of the racers reported symptoms of heart disease during the race.

At the end of the race, troponin I levels had increased significantly, by 0.03 [0.01-0.08] ($P<.001$); a moderate increase (≥0.04 ng/mL and ≤0.5 ng/mL) was observed in 14 runners (41.18%), and a >0.5 ng/mL (0.76 ng/mL) increase was observed in one case.

Significant increases were recorded in HDL-c (8.6 [5.15-11.53] mg/dL; $P<.001$), glucose (12 [7-31] mg/dL; $P=.013$), and CK (402 [227-668] mg/dL; $P<.001$). No increase was observed in any of the other variables (Table 1).

There was a statistically significant correlation between increased CK and race time (r=0.408; $P=.017$), but not between cTnI and CK, hours of training, or race time (Table 2). Finally, there was a negative correlation between LDL-C and increased cTn1.

The release of cTn secondary to myocardial injury can be due to ischemia from rupture of arterial plaque and coronary occlusion, ischemia without atherosclerosis, increased myocardial oxygen demand, and non-ischemic injury or direct damage (trauma, myocarditis or cardiotoxicity from drugs).[5] These do not explain the release of cTn in healthy subjects after exercise.

Several studies have shown cTn elevations in sports in which cardiac output, heart rate, and blood pressure remain high for hours, such as marathons, ultra marathons, triathlons, and cycling.[4,5]

This elevation could be due to damage to cardiomyocytes from the sustained increase in cardiac work rate combined with the physiological environment existing in situations of prolonged exercise (altered pH, increased core temperature, etc.).

Table 1
Pre- and Post-Race Troponin, Creatine, Glucose, and Lipid Values

	Pre-race	Post-race	*P*
Troponin I	0 [0-0.01]	0.04 [0.02-0.08]	<.0005
CK	101 [72-122]	474 [345-845]	<.0005
Blood sugar	88 [82-100]	103 [85-117]	.0130
Total cholesterol	176 [152-200]	182 [162-203]	.1200
Triglycerides	69 [53-89]	59 [48-74]	.1460
HDL-C	59 [49-71]	69 [60-78]	<.0005
LDL-C	100 [84-121]	99 [88-114]	.1020

CK, creatine kinase; HDL-C, high density lipoproteins cholesterol; LDL-C, low density lipoproteins cholesterol.
Data are medians [interquartile range].

Table 2
Spearman Correlations (*P*) Between Increased Cardiac Troponin I and Creatine Kinase, and Other Variables

	Elevated cardiac troponin I	Elevated CK
Age	−0.123 (0.488)	−0.164 (0.354)
BMI	0.136 (0.443)	−0.025 (0.889)
Weekly training time (h)	0.159 (0.369)	−0.144 (0.415)
Race time (min)	0.176 (0.391)	0.408 (0.017)
Elevated glucose	0.180 (0.309)	−0.256 (0.144)
Elevated LDL-C	−0.532 (0.001)	−0.140 (0.429)
Elevated HDL-C	−0.298 (0.087)	0.001 (0.997)
Elevated triglycerides	0.207 (0.240)	−0.234 (0.182)
Elevated cardiac troponin I	—	0.206 (0.241)

BMI, body mass index; CK, creatine kinase; HDL-C, high density lipoproteins cholesterol; LDL-C, low density lipoproteins cholesterol.

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A meta-analysis of 26 studies showed an increase in cTn in about half of the participants, a figure which was consistent with our study, in which increased troponin levels after racing was relatively common. Elevated cTn was related to the intensity and duration of exercise and the presence of cardiovascular disease,[6] which is more frequent among sedentary people doing long walks, and marathon runners, than in ultramarathon runners.[5]

Except for myocardial fibrosis in veteran athletes or alterations in cardiovascular magnetic resonance imaging in marathon runners over 50 years of age,[5] most non-invasive explorations find no association between increased cTn after exercise and the presence of permanent myocardial damage.[4,5] This difference in results may be due to the fact that cTn was determined.

Myocardial sarcolemmal hyperpermeability facilitates the release of cytosolic cTn into the extracelular space.[5]

Stimulation of integrins through stretching of the myocardium mediates transport of cTn or its degradation products to the exterior of the cardiomyocytes,[4,5] a process which differs from the release of cTn from necrotic myocardial tissue. Integrins are involved in cardiac remodeling after myocardial infarction or pressure overload.[5]

In rats, it has been shown that increased preload, without ischemia, leads to an increase in the degradation of cTnI, a finding which would indicate that myocardial stretching itself can degrade cTnI.[5] Although periods of prolonged exercise produce persistent myocardial stretching, we have not found any studies into the products of cTnI degradation after exercise.

In conclusion, there is no evidence that elevation of cTn after exercise is due to myocardial necrosis, and endurance sports can cause mild elevations of cTn in the absence of cardiac ischemia.

The study of cTnI degradation products could help to determine whether the release of cTnI is due to myocardial stretching or ischemia and thereby help to clarify the mechanism underlying cTn elevation.

Enric Subirats,[a,b,*] Gemma Subirats-Vila,[b] Iñigo Soteras-Martinez,[a] Emili Corbella,[c] Antoni Martinez,[d] and Xavier Pintó[c]

[a]*Servicio de Medicina Interna, Urgencias y Unidad de Medicina de Urgencias de Montaña, Hospital Transfronterer de Puigcerdà, Girona, Spain*
[b]*Facultat de Medicina, Universitat de Girona, Girona, Spain*
[c]*Unitat Funcional de Risc Vascular, Servei de Medicina Interna, Hospital de Bellvitge-Idibell, L'Hospitalet de Llobregat, Barcelona, Spain*
[d]*Servei de Cardiologia, Hospital Parc Taulí, Sabadell, Barcelona, Spain*

* Corresponding author:
E-mail address: esubirats@telefonica.net (E. Subirats).

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Congenital Hereditary Anomalous Coronary Artery Origin

Anomalía coronaria congénita familiar

To the Editor,

The incidence of anomalous origin of a coronary artery from the contralateral coronary cusp is estimated at 0.28% to 1.74%.[1] In most cases, these aberrant arteries do not lead to ischemia and are detected as incidental findings on diagnostic coronary angiography. However, they are a recognized potential cause of ischemic symptoms and sudden death, particularly in young athletes with an interarterial course of the anomalous vessel, in which it has been hypothesized that the coronary can become compressed between the aortic and pulmonary arteries. It is also believed that the presence of a coronary anomaly is associated with a greater degree of atherosclerosis and that it occurs more commonly in patients with degenerative aortic valve disease.[2]

There are few reports of this condition and most are isolated cases; thus, the clinical relevance and management of these anomalies remains to be defined.[3]

We present the case of two first-degree relatives (father and son) with this condition referred to our center for diagnostic coronary angiography.

The first patient was an 81-year-old man, who had undergone mechanical mitral and aortic valve implantation to treat rheumatic valvular disease, had a dual-chamber pacemaker, and was hospitalized for progressive dyspnea. Perfusion scintigraphy with dipyridamole technetium showed inferolateral ischemia, and the patient was referred for coronary angiography. The coronary study showed a dominant left circumflex artery (LCx) with anomalous origin in the right coronary cusp and sharing a common ostium with the right coronary artery (RC) (Fig. 1A).

Multislice computed tomography coronary angiography (64 channels) was then performed and the LCx was seen to have a retroaortic course that ran between the left atrium and ascending aorta (Figs. 1B and figs. 2A and B).

The second patient (son of the first) is a 50-year-old man, smoker, with dyslipidemia, who underwent coronary angiography to investigate chest pain on exertion. The son's coronary anatomy was very similar and could virtually be superimposed on that of his father (Figs. 1C and D, and figs. 2C and D).

Anomalous origin of the LCx from the right coronary cusp is the second most common coronary artery anomaly (some authors consider a high origin of the RC to be the most common anomaly), and accounts for one-third of all such conditions.[4] These vessels are not usually associated with symptoms and are considered the paradigm of "benignity" among these conditions.

A few studies have suggested that these anomalies can show family aggregation. Horan et al. described the case of a father and daughter, both with a single coronary artery[5]: in the father, the RC originated in the middle LCx, whereas in the daughter, the left