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Cardiac Troponin I Release Following A Single Aerobic Exercise Bout With Different Intensities In Adult Obese.

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Abstract

Elevated cardiac troponin I (cTnI) has been reported after exercise in healthy subjects. Currently, little is known about the impact of exercise intensity on cTnI release, but also the impact of obesity on this response. The purpose of this study was to investigate the influence of the intensity of a single bout of aerobic exercise on the post-exercise release of cTnI in obese adults. Sixty obese subjects aged 25-40 years were randomly assigned to light intensity aerobic exercise (group I, n=20), moderate intensity aerobic exercise (group II, n=20), vigorous intensity aerobic exercise (group III, n=20). All subjects performed a single bout of aerobic exercise. Serum samples were drawn before, immediately and 3 h after the exercise bout and were analyzed for cTnI. cTnI was significantly elevated after vigorous intensity aerobic exercise but not after light or moderate intensity exercise. It was concluded that exercise intensity influences the release of cTnI and that vigorous intensity is required for cTnI to be elevated significantly.

Key words: aerobic exercise, cardiac troponin I, obesity.

INTRODUCTION

The increased risk of cardiovascular and metabolic morbidity and mortality as result of obesity has been well described **[1]**, whilst the prevalence of obesity is still rising (across all ages) and affecting both the developed and developing world **[2].** Some previous studies found that measures of obesity, such as body mass index, may be associated with higher resting cardiac troponin levels in the general public **[3,4].**

Obesity is on the rise in low- and middle-income countries, particularly in the WHO African Region. Each year, 28 million individuals are dying from the consequences of overweight or obesity worldwide. The percentages of adults aged ≥20 years who are obese in Egypt are 22.5% and 46.3% for males and females respectively **[5].**

Since it is known that weight-loss treatment benefits the health of obese individuals, obese participants with risk factors for coronary heart disease should be treated by an appropriate weight-loss program **[6].** Weight reduction is the common goal in the treatment of obesity. There is sufficient evidence supporting the role of exercise training in promoting weight loss **[7].**

Regular physical exercise is recommended for the primary prevention of cardiovascular disease. Although regular exercise training reduces cardiovascular disease risk, recent studies have documented elevations in cardiac troponin (cTn) consistent with cardiac damage after bouts of exercise in apparently healthy individuals **[8].**

Cardiac troponins are highly specific markers of myocardial cell damage **[9]** and are central to the diagnosis of acute coronary syndromes **[10]**. If cTnI levels are higher at baseline, cTnI levels may rise even more under demanding conditions such as exercise. Therefore, subjects with increased cardiovascular risk may demonstrate a larger exercise-induced increase in cTnI compared to healthy controls **[11]**.

Previous studies indicated that cardiovascular risk factors are associated with higher baseline cTn-levels under resting conditions **[12].** Interestingly, recent clinical and epidemiological studies have demonstrated that these elevated resting cTn levels are predictive of all-cause mortality **[3,4,13]**and future cardiovascular disease **[4,13,14]**.

Although numerous studies have reported the release of cTn after exercise, there is no consensus regarding the prevalence, mechanisms, and clinical management of exercise-induced cTn release **[8].** Several studies have reported no significant post-exercise cTn elevations **[15,16,17],** but the majority of data documented statistically significant cTn increases after exercise **[18-23].** Possible explanations include differences in the fitness levels of participants, the type or duration of exercise, the timing of the post-exercise sample, the troponin assay used, and the detection limit used to define a "positive" cTn **[8].**

The short and long-term clinical significance of an increase in cardiac-specific biomarkers following strenuous endurance exercise is unclear. Some have suggested that there might be an optimum exercise intensity/duration with respect to the impact of physical activity on cardiovascular health **[24,25].** Despite this speculation much remains unknown with respect to the acute effect of exercise of varying intensity has on the appearance of cardiac biomarkers **[26].**

Although aerobic exercise is a common non pharmacological intervention for the management of obesity, there is no clear consensus about the optimal exercise intensity in this population **[27].** A current escalating controversy in the fitness industry is whether there are greater health-related benefits and cardiovascular safety to performing more vigorous exercise versus moderate intensity exercise **[28].** The influence of exercise duration and intensity on cardiac-specific biomarker release is poorly understood and controversial **[29].** Therefore the purpose of this study was to investigate the influence of different intensities of a single bout of aerobic exercise on the post-exercise release of cardiac troponin I in adult obese. On comparing the results, the most appropriate and safe intensity for this population can be determined.

Research Design and Methods

Subjects characteristics and general experimental design

Study subjects

Sixty obese men were selected randomly from physical therapy outpatient clinic in Kasr El Ainy hospital. with body mass index (BMI) from 30.7 to 34.2 kg/m2. The participants were diagnosed as obese (classI), non smokers and non-hypertensive, free from respiratory, kidney, liver, metabolic and neurological disorders as well as chronic inflammatory orthopedic disorders, rheumatoid arthritis or chronic cardiac problems as heart failure, ischemic heart disease, and coronary artery bypass graft. Their age ranged from 25-40 years and not regularly involved in sport activities.

Evaluated parameters

Blood Samples and Biochemical Markers

Blood samples were gathered before, immediately (post-ex1) and 3 hours after finishing the exercise bout (post-ex2) [30, 31]. The URL for cTnI, defined as the 99th percentile of healthy participants, was 0.04 µg L-1[32].

Ratings of perceived exertion (RPE) are generally believed to be valid and reliable markers of physiological intensity during exercise **[33]** and are recommended to monitor exercise intensity **[34].** Participants were randomly divided by using the Borg 6–20 RPE scale into three intensities aerobic exercise groups: Group I: light intensity group, Group II: moderate intensity group, Group III: vigorous intensity group. All sessions were supervised and participation assessed. The study procedures were carried out at Outpatient Clinic of the Faculty of Physical Therapy, Cairo University. All subjects were free to withdraw from the study at any time. All participants provided their informed consent after receiving a detailed explanation of the study. The ethics committee of research in Faculty of Physical Therapy, Cairo University approved the study. The data of all the participants were available for analysis. The detailed exercise protocol was as follows:

Exercise protocol:

For the entire exercise session all participants had to adhere to identical exercise protocol. After an initial, 5 minute warming up phase performed on the treadmill at low load, the speed was increased until the patient reached: Group I: low intensity (11 on the Borg scale)**; Group II:** moderate intensity (12-14 on the Borg scale); **Group III**: vigorous intensity (15-17 on the Borg scale) **[35]**, Then the subject walked at the obtained level of speed for 30 min, and ended with 5-minute cooling down as warming up [36].

Statistical Analysis

A comparison of baseline data for cTnI was made via repeated measures one-way ANOVA. The delta score (baseline to peak post-exercise values) was compared between exercise intensities by one-way ANOVA ($P < 0.05$).

RESULTS

The study involved sixty obese men. Their age ranged from 25 to 40 years. They were enrolled in 3 equals number groups with different exercise intensities for a single exercise session in order to compare the influence of different intensities of a single bout of aerobic exercise on the post-exercise release of cardiac troponin I in adult obese. All subjects successfully completed the exercise bouts. (Table 1) showed the all pre-exercise cTnI concentrations were below the upper reference limit (URL) for the diagnosis of acute myocardial infarction. Baseline cTnI levels were normal and not different across the three groups. No significant difference have been found between pre-ex, post-ex1 and post-ex2 cTnI values in both Group I and II as the mean values were 0.004±0.004 ng/mL, 0.004±0.004 ng/mL and 0.004±0.004 ng/mL respectively in group I and 0.002±0.003 ng/mL, 0.002±0.003 ng/mL and 0.004±0.005 ng/mL respectively in group II. While there was a significant difference between Pre-ex, Post-ex1 and Post-ex2 cTnI in Group III as the mean values were 0.004±0.006 ng/mL, 0.011±0.009 ng/mL and 0.014±0.011 ng/mL respectively shown in (Table 2).

The peak post-ex cTnI means in Group I, II and III were 0.004±0.004, 0.004±0.005 and 0.014±0.011 respectively. There was a statistically significant difference between peak Post-ex cTnI in Group III and peak Post-ex cTnI in both Group I and II (p value 0.000), while there was no significant difference between peak Post-ex cTnI in Group I and peak Post-ex cTnI in Group II.(Table 3)

Table 1: Clinical characteristics of study subjects of three groups at baseline.

Characteristic	group I (*n*=20)	group II (*n*=20)	group III (*n*=20)	*P* value
Age, yr	33.1 ± 6.3	33.6 ± 6.0	32.8 ± 5.8	P>0.05**
Height, cm	168± 4.32	170± 4.95	169± 4.89	P>0.05**
Weight, kg	93.05 ±4.6	93.50 ±4.65	94.05 ±4.26	P>0.05**
BMI, kg/m2	32.9 ±0.73	33.1 ±0.89	33.3 ±0.80	P>0.05**

*BMI, body mass index; Level of significance at P<0.05, * = significant, ** = non-significant*

Table 2: Mean value and significance of cTnI between the three groups before and after the exercise bout.

	Mean ±SD			P-value
	Pre- ex	Post-ex1	Post-ex2	
Group I	0.004±0.004	0.004±0.004	0.004±0.004	0.33**
Group II	0.002±0.003	0.002±0.003	0.004±0.005	0.31**
Group III	0.004±0.006	0.011±0.009	0.014±0.011	0.000*

*cTnI: cardiac Troponin I ; Level of significance at P<0.05, * = significant, ** = non-significant*

Table 3: Mean value and significance of peak post-exercise cTnI between the three groups.

	Mean ±SD			F-value	P-value
	Group I	Group II	Group III		
Peak Post-ex cTnI	0.004±0.004	0.004±0.005	0.014±0.011	11.08	0.000*

*cTnI: cardiac Troponin I ; Level of significance at P<0.05, * = significant, ** = non-significant*

DISCUSSION

Controversy still exists as to whether or not aerobic exercise can induce increases of cardiac troponin in obviously healthy subjects and whether obesity alters the exercise-induced cardiac troponin release in response to different intensities of aerobic exercise. ([8]; [18]). The aim of this study was to investigate the influence of the intensity of a single bout of aerobic exercise on the post-exercise release of cardiac troponin I in adult obese. The mean value of cTnI was significantly increased in group III compared to group I and group II. On the other hand, there was no significant difference in group I & group II after exercise bout. This means that vigorous aerobic exercise resulted in substantial increase in post-exercise cTnI release compared to light and moderate intensity exercise.

There is currently limited evidence for any specific mechanism responsible for the release of cTn after exercise [37] and the clinical and/or performance implications of post-exercise cardiac troponin release are still being debated [26]. It has been suggested that changes in cellular membrane permeability, subsequent to an increased rate and force of cardiac contraction during vigorous exercise, may provide a mechanism by which unbound cardiac troponins in the cystolic pool (<10%) is released into the circulation after exercise [38]. Elevated cTnI levels are suggestive for cardiac damage, but the average cTnI increase in the present study was small and was not associated with symptoms of cardiac injury. It may well be possible that the increase in cTnI did not reflect irreversible ischemic myocardial “damage”, but relates to a physiological response to high-intensity exercise. The elevated heart rate during exercise may cause an increased mechanical stress on the heart, possibly leading to an increased release of cTnI [39].

Recently, it was also suggested that cTn may be released in response to transient ischemia alone, without necrosis. It was suggested that during ischemia, blebs develop on the surface of cardiac myocytes. If the ischemia is prolonged the blebs rupture and cellular necrosis with prolonged troponin release follows. However, if the ischemia is corrected before any blebs rupture, then the blebs are either resorbed or shed into the circulation. If shed, they will release cytoplasmic contents as a “one-off” event and this will be cleared with a short half-life [40]. Vigorous exercise intensity in the present study would likely increase the relative amount of hypoxia and thus bleb formation [26].

Furthermore, stimulation of integrins through mechanical stretching of the myocardium mediates transport of cTn or its degradation products to the exterior of the cardiomyocytes, 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 ([19]). Indeed, higher pericardial fat in cases of obesity is associated with left ventricular (LV) hypertrophy [41], increased LV wall thickness and mass, increased end-systolic wall stress, and reduced LV compliance [42], that may

exaggerate the myocardial response to mechanical stretching and ensuing stimulation of integrins during vigorous exercise in the present study.

Mechanisms such as insult to the sarcolemmal membrane due to free radicals have been suggested but currently lack sufficient confirmatory data. The relatively small magnitude of cTn release and the rapid clearance of cTn post-exercise do, however, support a mechanism unrelated to frank myocardial injury or cell necrosis [37]. However, obesity-induced elevation in oxidant and free radical formation promotes the incidence of adverse obesity-related clinical responses [43] and may cause an insult to the sarcolemmal membrane and subsequent cTn release in response to vigorous exercise.

In agreement with the present study, it was suggested that exercise intensity may have a key role in the release of cardiac troponins during endurance exercise in healthy athletes [44]. Shave et al, [30] reported cTnI release after 30 min of short duration high-intensity exercise in 6 out of 8 runners and noted that the responses were markedly heterogeneous. In the same domain, Peak post-exercise values of cTnI were higher than the URL in six runners (43%), but only after the exercise at competition intensity [26]. Likewise, significant serum cTnI was observed in twenty-three healthy male professional football players approximately 24 h after a training session of intermittent high intensity exercise [45]. The concentration of cTnI measured in post-marathon samples was remarkably increased as compared with values obtained on baseline specimens in 18 trained athletes, who performed a 60-km ultra-marathon run [46]. Similarly, Eijsvogels et al, [38] found that cardiac troponin I levels significantly increased in a large heterogeneous group of athletes after completing a marathon. Further, cardiac troponin I levels were found to increase significantly in 14 runners after covering a distance of 80 km with an elevation gain of 2600 m with the race including sections of swimming, running, and cycling ([19]).

In contrast to the above mentioned positive findings, several studies have reported no significant cTn increase in response to vigorous aerobic exercise. Most of these studies were field studies in sports, such as marathons, ultra marathons, triathlons, and cycling. In a recent study, vigorous aerobic exercise caused no significant cTnT changes in 12 runners after running a mean distance of 140.3 ± 18.7 km. Although two subjects showed increased cTnT values, the values were below the reference limit for the detection of myocardial injury ([15]).

Roth et al., [47] concluded that concentrations of cTnT remained below detectable levels in 10 well-trained runners after strenuous ultra-endurance exercise (continuous 216 km). Also, no change in cTnI or cTnT was reported in 82 middle-aged runners after Boston marathon ([17]). The same finding was reached by Konig et al., [48] who observed no significant rise in cTnT after competitive, long-lasting aerobic exercise in eleven highly trained professional road cyclists. They assumed that extreme competitional exercise can be designated as unaccustomed for the whole organism, and particularly for the heart that may induced the post-exercise cTn release while subjects in their study are definitely accustomed to this type of strenuous exercise. Troponin T (TnT) was assessed before a 21-km half-marathon, at the end, and 3, 6, 24 h thereafter in 17 trained, middle-aged males. There was no significant increase in the concentration of TnT in any subject of the study participants [16].

Because the clearance rate of cTn by the kidney is likely to differ between individuals, and because the point in time relative to exercise when the post-exercise blood sample is drawn will also differ, it is overly simplistic to think that a delta change in cTn from pre-exercise to post-exercise reflects total cTn release. Therefore, the discrepancy in post-exercise cTn release between previous studies is likely due to methodological differences and the timing of blood draws post-exercise [37].

CONCLUSION

In summary, aerobic exercise intensity has a differential effect on post-exercise release of cTnI , with pronounced response in vigorous intensity aerobic exercise. In consequence, moderate exercise can be regarded as safe when the allowed upper exercise heart rate is not exceeded. However, because cardiocirculatory training effects have been shown to be superior for higher walking intensities in CAD participants, moderate-intensity exercise should be preferred in rehabilitative cardiocirculatory training.

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الملخص العربي

مدى إفراز تروبونين القلب آي بعد نوبة واحدة من التمرينات الهوائية المختلفة الشدة لدى البالغين البدناء

أثبتت الدراسات السابقة زيادة في تروبونين القلب آي بعد التمرين لدى الأفراد الأصحاء. لا يعرف حاليا إلا القليل عن تأثير شدة التمرين على إفراز تروبونين القلب آي، وكذلك تأثير السمنة على هذه الاستجابة. يهدف هذا البحث إلى دراسة تأثير شدة نوبة واحدة من التمرينات الهوائية على إفراز تروبونين القلب آي لدى البالغين البدناء.أجرى البحث على ستين شخصا من البالغين البدناء وتراوحت أعمارهم ما بين خمسة وعشرين الى أربعين عاما، تم تقسيمهم إلى ثلاث مجموعات متساوية العدد يؤدي أفراد كل مجموعة نوبة واحدة من التمرينات الهوائية خفيفة الشدة في المجموعة الأولى ، متوسطة الشدة في المجموعة الثانية و عالية الشدة في المجموعة الثالثة. سحبت عينات من الدم قبل التمرين و بعده مباشرة و ثلاث ساعات بعد ذلك وتم تحليل العينات لتحديد تروبونين القلب آي. وجدت الدراسة أن تروبونين القلب آي يرتفع بشكل ملحوظ بعد التمرينات عالية الشدة و لا يتأثر بالتمرينات خفيفة أو متوسطة الشدة. توصلت الدراسة إلى أن شدة التمرينات الهوائية تؤثر على إفراز تروبونين القلب آي و أن شدة التمرين العالية لازمة لحدوث إفراز ملحوظ.

الكلمات الدالة : (التمرينات الهوائية - إفراز تروبونين القلب - السمنة).