

Cardiac troponin response to aerobic versus resisted exercises in obese women

Aisha A Hagag*, **Hany F Elsisy***, **Hadeel M Mohamed****, **Hend M Mohamed*****

*Department of physical therapy for cardiovascular / respiratory disorders & geriatrics, faculty of physical therapy, Cairo university.

** Department of clinical pathology, faculty of medicine, Cairo university.

***Department of physical therapy, Cairo university hospitals.

ABSTRACT

Background:

Obesity represents an important cardiovascular risk factor. Body mass index may be associated with higher resting cardiac troponinI (cTn I) levels. It is unknown whether obesity alters the exercise-induced troponinI release, this is of special importance as exercise training is routinely prescribed as an effective strategy to lose weight of individuals with obesity. The current study was carried out to determine the cardiac troponinI acute response to aerobic versus resisted exercises in obese women.

Patients and method

This study included 40 obese women, their age ranged from 30 to 40 years, their BMI ranged from 30-34.9kg/m². Subjects were randomly selected from the employees of Kasr Al Ainy teaching hospital. They divided into two groups equal in number (A&B). Group (A) performed a single bout of aerobic exercises and group(B) performed single bout of resisted exercises. Serum (cTn I) were analyzed before the exercise session and three hours from the end of exercise session. The study was done from march 2017 till January 2018 .

Results

Comparing pre and post exercise values revealed an increase in cTnI in both groups, However there was a statistical significant difference between both groups in post exercise values

Conclusion:

The magnitude of cardiac troponin I elevation in aerobic exercises is less than that of resisted exercises which suggest that aerobic exercises are more suitable and safe than resisted exercises for obese women .

Key words: Obesity, cardiac troponin, resisted exercise , aerobic exercise

Introduction

Obesity represents an important and common cardiovascular risk factor whilst the prevalence of obesity is still rising (across all ages) and affecting both the developed and developing world .Some previous studies found that measures of obesity, such as body mass index, may be associated with higher resting cardiac troponinI(cTnI) levels in the general public. However it is currently unknown whether obesity alters the exercise-induced troponin release. This is of special importance as exercise training is routinely prescribed as an effective strategy to lose weight to individuals with obesity^[1]

Troponin is a complex consisting of three single chain polypeptides: troponin –I which prevents muscle contraction in the absence of calcium, troponin T, which connects the troponin complex to tropomyosin and troponin C, which binds calcium Together with tropomyosin and under the influence of calcium, troponin regulates muscle contraction. After myocardial infarction, elevated levels of cardiac muscle specific isoform cardiac troponin I(C Tn I) levels appear within 3-6 hours. Levels peak within 14-20hours and return to normal after 5-7 days. Determinations of CTn I are necessary, which is also useful for assessing reperfusion following thrombolysis. The most important characteristic of CTn I is its apparent absolute cardiac specificity. Among patients with acute coronary syndromes, CTn I levels have been reported to provide prognostic information useful for the early identification of patients with an elevated risk of unstable angina.^[2]

Regular physical exercise is recommended for the primary prevention of cardiovascular disease(CVD). Although regular exercise training reduces cardiovascular disease risk, recent studies have documented elevations in cTn consistent with cardiac damage after bouts of exercise in apparently healthy individual.^[3]

The metabolic effects of reduced muscle mass, engendered by normal aging or decreased physical activity, lead to a high prevalence of obesity, insulin resistance, type 2 diabetes, dyslipidemia and hypertension. Skeletal muscle is the primary metabolic “sink” for glucose and triglyceride disposal and is an important determinant to resting metabolic rate. Accordingly, it has been hypothesized that resistance exercise training (RT) and subsequent increases in muscle mass may reduce multiple (CVD) risk factors.^[4]

Prescribed and supervised (RT) enhances muscular strength and endurance, functional capacity and quality of life while reducing disability in persons with and without (CVD). These benefits have made RT an accepted component of programs for health and fitness. The American Heart Association recommendations describing the rationale for participation and considerations for prescribing RT ,it provides current information regarding the health benefits of RT, impact of RT on the cardiovascular system structure and function, and role of RT in modifying CVD risk factors^[5]

The beneficial effects of moderate intensity exercise for the cardiovascular system are well documented.^[6]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.^[7]

Despite this speculation, much remains unknown with respect to the acute effect of exercise of varying intensity has on the appearance of cardiac biomarkers.^[6]

Patients and methods

Patients

A randomized controlled comparative study which was carried out on 40 obese women, their age ranged from 30 to 40 years, their BMI from 30-34.9kg/m²(class I obesity). Subjects were randomly selected from employees of Kasr Al Ainy teaching hospital.

Exclusion criteria

Obese women with any of the following characteristics excluded from the study: BMI> 35 kg/m²,History of heart disease, respiratory disorders, stroke, diabetes and cancer, Life-threatening illness or any condition that limited their ability to engage in exercise program as orthopedic or neurological abnormality.

Study has been approved by ethical committee of faculty of physical therapy , Cairo university. All subjects gave an informed consent to participate in study procedures.

Equipments

Dimension EXL 200 device made in German was used in analysis of cTnH. Electronic Treadmill made in USA with serial number FV-420 was used for aerobic exercises program. Free weights were used for resisted exercises program.

Assessment procedure

Preliminary Assessment

Careful history was taken by physical therapist, for any previous diseases hinder walking or cause pain during the exercise. Resting blood Pressure (RBP) and resting heart rate (RHR) were also measured and recorded.

The Anthropometric measures

Calculation of BMI: $BMI = \text{weight (kg)} / \text{height (m)}^2$

Blood Sampling Procedure

Blood samples were drawn from an antecubital vein before and 3 hours after finishing the exercise session. All measurements were done in clinical pathology department of Cairo university hospitals.

Training Procedures:

For group A (aerobic exercises), the program composed of warming up phase (10 minutes of walking on the treadmill at speed 1.5 km/h with zero inclination), active phase (20 minutes of high speed walking on electronic treadmill with zero inclination. Exercise intensity was determined using Karvonen formula: $\text{Target Heart Rate} = ((\text{Maximum Heart Rate} - \text{Resting Heart Rate}) \times (60-80\% \text{Intensity}) + \text{Resting Heart Rate})^{[8]}$ and Cooling down phase (10 minutes walking on the treadmill at speed 1.5 km/h with zero inclination). Pulse rate was recorded throughout the session using pulseoximeter watch.

For group B (resisted exercises), the program composed of warming up phase (10 minutes walking on the treadmill at speed 1.5 km/h with zero inclination), Active phase formed of 3 sets of exercise, each set comprised of 10-15 repetition for each exercise, with duration 20min with two minutes

rest between sets for each muscle group. Resisted exercises included the following (Knee flexion, knee extension, hip flexion, hip extension, hip abduction, hip adduction, abdominal curl , shoulder flexion, shoulder abduction and elbow flexion exercise) . Moderate resisted exercise intensity was determined by the assessment of the maximum load that the individual can lift only one time: one repetition maximum (1RM). 60%of 1 RM was taken^[9]

the session ended by cooling down (walking on the Treadmill for 10 minutes at speed 1 km/h with zero inclination and).^[10]

Statistical analysis

Descriptive statistics and t-test were conducted for comparison of subject characteristics between both groups. Unpaired t test was conducted to compare mean values of cTnI between both groups; and paired t test was conducted to compare between pre and post treatment mean values of cTnI in each group. The level of significance for all statistical tests was set at $p < 0.05$. All statistical tests were performed through the statistical package for social sciences (SPSS) version 19 for windows (IBM SPSS, Chicago, IL, USA).

- Results

- Subject characteristics:

Table (1) showed the mean \pm SD age, weight, height and BMI of group A and B. There was no significant difference between both groups in the subject characteristics ($p > 0.05$).

Table (1): Descriptive statistics and t-test for comparing the mean age, weight, height and BMI of group A and B.

	Group A	Group B	MD	t- value	p-value
	$\bar{x} \pm SD$	$\bar{x} \pm SD$			
Age (years)	36 \pm 2.47	34.95 \pm 2.25	1.05	1.4	0.16
Weight (kg)	81.65 \pm 7.06	84.67 \pm 6.88	-3.02	-1.37	0.17
Height (cm)	158 \pm 7.11	160.35 \pm 6.06	-2.35	-1.12	0.26
BMI (kg/m²)	32.61 \pm 1.23	32.7 \pm 1.11	-0.09	-0.22	0.82

\bar{x} , Mean; SD, Standard deviation; MD, Mean difference; p value, Probability value; *, Non significant.

Effect of exercise on cTnI

-Pre exercise analysis of cTnI values of both group revealed non significant difference as shown in table (2) and figure (1).

- Comparing pre and post exercise cTnI values of group (A) revealed a significant difference with 72.72% of increase; While comparing pre and post exercise cTnI values of group (B) revealed a significant difference with 138.71% of increase as shown in table (2) and figure (1).

- Comparing post exercise values revealed an increase in cTnI in both aerobic (group A) and resisted exercise (group B) while the magnitude of increase in group (B) is higher than group (A) as shown in table (2) and figure (1).

Table (2). Mean cTnI pre and post exercise of group A and B.

<i>cTnI</i> (ng/ml)	Pre exercise	Post exercise	MD	% of change	t-value	P-value
	$\bar{x} \pm SD$	$\bar{x} \pm SD$				
Group A	0.033 ± 0.01	0.057 ± 0.02	-0.02	72.72	-9.24	0.001**
Group B	0.031 ± 0.01	0.074 ± 0.01	-0.04	138.71	-14.77	0.001**
MD	0.002	-0.01				
t-value	0.41	-2.91				
P-value	0.67*	0.006**				

\bar{x} , Mean; SD, Standard deviation; MD, Mean difference; p value, Probability value; *, Non significant; **, Significant.

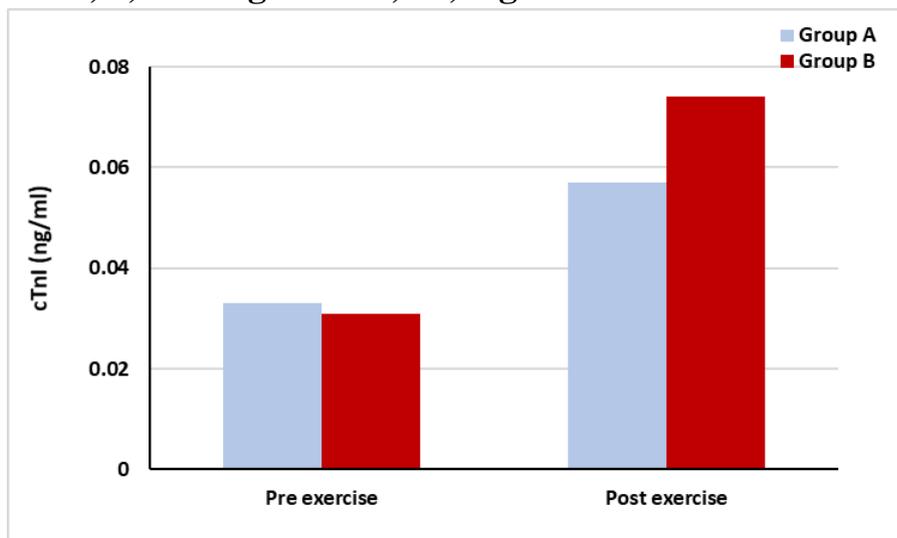


Figure (1): Mean cTnI pre and post exercise of group A and B

Discussion

A large number of obese subjects engage in high-intensity training in weight loss programs with exercise intensity and duration that may exceed the program parameters established for safe exercise. Whether the elevated exercise volume provides additional health benefits or, controversially increases health risk is currently not clear^[6]

Controversy still exists 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^[11]

Therefore, the aim of this study was to investigate the influence of a single bout of moderate resisted exercises and single bout of moderate aerobic exercise on the post-exercise release of cardiac troponinI in obese women.

This study demonstrated that resisted and aerobic exercise have a differential effect on post-exercise appearance of cTn I. Specifically, resisted exercises resulted in a substantial increase in cTnI post-exercise, compared to moderate aerobic exercise. Post-exercise cTnI was not elevated above the clinical cut-off value for myocardial infarction (0.04-0.08 ng/mL) in any participants.

The subsequent section will attempt to relate the findings of the present study to the findings of previous research. This discussion will also discuss various possible physiological mechanisms which could explain the findings of this investigation.

All through the study, this research outcome is not in agreement with some previous studies that showed three bouts of 90- minute exercise did not have any significant effect on the level of the troponin indices^[12] another showed that there were no significant changes in cardiac troponin levels over time after resisted exercise training, indicating that the results of the study do not support the idea that cardiac troponin levels might be modifiable through

exercise.^[13] Although the outcomes are similar to those of other research work including the report that showed more than half of the participants developed troponin increase after basketball training.^[14] the same increase in troponin I was reported after treadmill test^[3] marathon and ultra-marathon races leading to transitory elevation of troponin levels.^[15]

Several studies have reported non-significant cTnI increase in response to vigorous aerobic exercise. Most of these studies were field studies in sports, such as marathons, ultra marathons and cycling^[16]

There is currently limited evidence for any specific mechanism responsible for the cTn release after exercise^[17] and the clinical and/or performance implications of post-exercise cardiac troponin release are still being debated^[6]

Although cardio-vascular imaging studies were not performed alongside blood draws in the current study, recent empirical studies do not provide evidence that biomarker release and changes in left ventricular function post-exercise are causally related^[18]. Whether serum cTnI observed post-exercise reflects reversible or irreversible myocardial damage is also still to be conclusively determined^[19]

obesity-induced elevation in oxidant and free radical formation promotes the incidence of adverse obesity-related clinical responses^[20]

Elevated cTn I 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 moderate 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^[21]

The cTnI elevations found in the present study are definitely not coming from necrosis or ischemia ,if so, aerobic exercise training should be hazardous to obese subjects. The results of this study can be used as a support for the prescription of different types of exercise in the control or treatment for obesity, aiming not only to increase energy expenditure and fitness but also to improve the metabolic control in this population. Because exercise-induced myocardial injury maybe self-abating, obese subjects should not be excluded from participating in aerobic exercise programs^[22]

Conclusion

The present data revealed that (cTnI) was elevated in all subjects after exercise. However, the magnitude of (cTn I) elevation in aerobic group is less than of resisted . In consequence , moderate aerobic exercise can be regarded as safe when the allowed upper exercise heart rate is not exceeded,^[23]. So, moderate aerobic exercise can be recommended and preferred to obese patients and those with coronary artery disease for rehabilitative cardio-circulatory training.

Acknowledgment:

For all participants

REFERENCES

- 1- **Thijs M. H., Eijsvogels., Matthijs T.W.,Veltmeijer and Keith George** ·
The impact of obesity on cardiac troponin levels after prolonged exercise
in humans *Eur J ApplPhysiol* . 2012; 112:1725–1732.
- 2- **Padmaja V and Deepu P.** Cardiac Biomarkers. *HYGEIA* 2009;1 (1)
- 3- **Shave R.,Baggish A., George K., Wood M and Thompson P.** Exercise-
induced cardiac troponin elevation: evidence, mechanisms, and
implications. *J Am CollCardiol.*2010; 56(3):169-76.
- 4-**Randy W., Braith., PhD., Kerry J and Stewart.** Resistance Exercise
Training Its Role in the Prevention of Cardiovascular
Disease.*Circulation.*2006;113:2642-2650.
- 5- **Mark A.,Williams, PhD., Co-Chair; William L. et al.** A Scientific
Statement From the American Heart Association Council on Clinical
Cardiology and Council on Nutrition, Physical Activity and
metabolis,*Circulation.* 2007;116:572-584.
- 6- **Legaz-Arrese A., George K., Carranza-Garcfa L., Mungufa- Izquierdo
D., Moros-Garci'a T., Serrano-Osta'riz E.** The impact of exercise
intensity on the release of cardiac biomarkers in marathon runners. *Eur J
Appl Physiol.* 2011;111: 2961-2967.
- 7- **La Gerche A. and Prior D.** Exercise—is it possible to have too much of a
good thing? *Heart Lung. Czrc.*2007;16 (3):S 102-S104.
- 8- **Sérgi R., Antonio S., Cristmi N., FábioB., Vera L.et al.** Comparison of
Maximal Heart Rate Using the Prediction Equations Proposed by
Karvonen and Tanaka.*Arq Bras Cardiol* 2008; 91(5) : 285-288
- 9-**Dong-il S., Eonho K., Christopher A., Fahs, Lindy R,et al.** Reliability of
the one-repetition maximum test based on muscle group and gender.
Journal of Sports Science and Medicine (2012) 11, 221-225

- 10- **American College of Sports Medicine.** ACSM's Resource Manual for Guidelines for Exercise Testing and Prescription. Philadelphia, PA: LWW; 7th ed.2013; pp 289-295.
- 11-**Shave R., Ross P., Low D., George K. and Gaze D.** Cardiac troponin I is released following high-intensity short-duration exercise in healthy humans.*Int J Cardiology.* 2010. 19; 145(2):337-339.
- 12-**Rahnama N, Faramarzi M, Gaeini AA.** Effects of intermittent exercise on cardiac troponin i and creatine kinase-MB. *Intl J Prev Med.* 2011;2(1):20–23.
- 13- **Van Der Linden N, Klinkenberg LJJ, Leenders M, Tieland M, Verdijk LB, Niens M, Meex SJR.** The effect of exercise training on the course of cardiac troponin T and I levels: Results from three independent training studies. *Nederlands Tijdschrift Voor Klinische Chemie En Laborat.* 2015;40(2):100.
- 14-**Nie J, Tong TK, JShi Q, Lin H, Zhao J, Tian Y.** Serum cardiac troponin response in adolescents playing basketball. *Intl J Sports Med.* 2008;29(6):449–452.
- 15- **Regwan S, Hulten EA, Martinho S, Slim J, Villines TC, Mitchell J, Slim AM.** Marathon running as a cause of troponin elevation: A systematic review and metaanalysis. *J Intervent Cardio.* 2010; 23(5):443–450
- 16-**Passaglia D.,Emed L., Barberato S., Guerios S., Moser A., Silva M, Ishie E., Guarita-Souza L., Costantini C. and Faria-Neto J.** Acute Effects of Prolonged Physical Exercise: Evaluation After a Twenty-Four-Hour Ultramarathon. *Arq Bras Cardiol.* 2013; 100(1):21-28.
- 17-**Shave R and Oxborough D.** Exercise-Induced Cardiac Injury: Evidence From Novel Imaging Techniques and Highly Sensitive Cardiac Troponin Assays.*ProgCardiovasc Dis.* 2012;54:407-415.

- 18-Wilson M., O'Hanlon R., Prasad S., et al.** Biological markers of cardiac damage are not related to measures of cardiac systolic and diastolic function using cardiovascular magnetic resonance and echocardiography after an acute bout of prolonged endurance exercise. *Br J Sports Med.* 2011;45:780-784.
- 19-Eijsvogels T., Hoogerwerf M., Maessen M., Seeger J., George K., et al.** Predictors of cardiac troponin release after a marathon. *J Sci Med Sport.* 2014; pii: S1440-2440(13)00519-7.
- 20-Khoo N., Cantu-Medellin N., Devlin J., St Croix C., Watkins S., et al.** Obesity-induced tissue free radical generation: an in vivo immuno-spin trapping study. *Free Radic Biol Med.* 2012; 1-15;52(11 -12):2312-2319
- 21-Eijsvogels T., Veltmeijer M., George K., Hopman M Thijssen D.** The impact of obesity on cardiac troponin levels after prolonged exercise in humans. *Eur J Appl Physiol* 2011; 112(5): 1725—1732.
- 22-Koller A.** Exercise-Induced Increases in Cardiac Troponins and Prothrombotic Markers. *Med. Sci. Sports Exerc.* 2003; 35(3):444-448.
- 23-Scharhag J., Herrmann M., Urhausen A., Haschke M., Herrmann W and Kindermann W.** Independent elevations of N-terminal pro-brain natriuretic peptide and cardiac troponins in endurance athletes after prolonged strenuous exercise. *Am Heart J.* 2007;150:1128-34.

مستخلص

استجابة تروبونين القلب لتمرينات المقاومة مقابل الهوائية لدى السيدات البدنيات
* ا.م.د. عائشة عبد المنعم حجاج ، * أ.م.د. هاني فريد عيد مرسي السيبي ، * * د. هديل محمد
محمد ، * * * هند مهدي حسن محمد
* قسم العلاج الطبيعي لاضطرابات الجهاز الدوري التنفسي والمسنين، كلية العلاج الطبيعي، جامعة
القاهرة

* * قسم الباثولوجيا الكيميائية والإكلينيكية، كلية الطب، جامعة القاهرة
* * * قسم العلاج الطبيعي _ القصر العيني _ مستشفيات جامعة القاهرة.

نبذة مختصرة: السمنة عامل خطر قلبي هام ، تقليل الوزن يعتبر الهدف العام من علاج السمنة ، قد يرتبط مقاييس البدانة، مثل مؤشر كتلة الجسم بارتفاع مستويات تروبونين القلب. أهم سمة لتروبونين القلب هي خصوصيته المطلقة للقلب . **الغرض:** لتحديد استجابة تروبونين القلب أي لتمرينات المقاومة مقابل التمرينات الهوائية لدى السيدات البدنيات. **المنهجية:** شاركت 40 سيدة بدنية ذات مؤشر كتلة جسم من 30-34.9 كم² م يتراوح أعمارهم من 30-40 سنة. **تصميم الدراسة:** تم تقسيم المرضى عشوائياً إلى مجموعتين متساويتين في العدد (أ، ب). تلقت مجموعة (أ) نوبة واحدة من التمارين الهوائية وتلقت المجموعة (ب) نوبة واحدة من تمارين المقاومة. تم تحليل تروبونين القلب أي قبل التمارين وبعد ثلاث ساعات من التمارين. **النتائج:** وجد اختلاف ملحوظ بين المجموعة (أ) مقارنة بالمجموعة (ب) بعد التمارين. **الاستنتاج:** التمارين الهوائية مناسبة وآمنة أكثر للسيدات البدنيات.
الكلمات الدالة: سمنة- تروبونين أي- تمارين مقاومة - تمارين هوائية.