Original article

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Troponin release following exercise test in patients with stable angina pectoris — risk factors and prognostic significance

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Abstract

Background: Increase of troponin (cTn) is a marker of myocardial injury caused by different mechanisms. Exercise testing (ExT) is a useful clinical tool in predicting the risk of myocardial ischemia, especially in patients with multivessel coronary artery disease (CAD), who are more often endangered by medical complications. The test is however limited by its low sensitivity and specificity.

Aim: To evaluate the reasons for troponin I (cTnI) release after ExT, and to determine its clinical and prognostic implications in patients with stable CAD, referred for elective coronary angiography (ANG).

Methods: 118 patients without signs of systolic heart failure, referred for planned coronary ANG were included in the analysis. After baseline measurements of NT-proBNP, hsCRP, cTnI, CK-MB levels, maximal ExT was performed, followed by the consecutive measurements of cTnI and CK-MB 12 and 24 hours after examination. All patients underwent coronary ANG and ECHO within 7 days of taking blood samples. All patients were followed up on average for 35.5 months.

Results: The cTnI elevation ≥ 0.14 ng/mL (≥ 99th percentile value of the reference group) after 24 hours of the ExT was observed in 11 (9%) patients. Predictors of cTnI release in patients after ExT were as follows: ejection fraction ≤ 50%, lack or insufficient physical activity, max systolic blood pressure > 160 mm Hg at peak of ExT (OR 6.6, 95% CI 1.2–35.4, p = 0.027; OR 5.5, 95% CI 1.1–28.8, p = 0.04; OR 6.3, 95% CI 1.3–31.6, p = 0.025, respectively). Increase of cTnI after ExT did not correlate with multivessel CAD nor with future adverse clinical events.

Conclusions: The cTnI release post ExT is more frequently observed in patients with stable CAD with ejection fraction ≤ 50%, low physical activity, and max systolic blood pressure > 160 mm Hg at peak ExT. Post ExT cTnI increase in patients with stable CAD did not correlate with the number of atherosclerotic coronary vessels, and had no prognostic implications. Increase of cTnI after ExT did not have any predictive value in respect to acute coronary syndrome and/or death during long-term follow up.

Key words: exercise test, troponin I, stable coronary artery disease

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INTRODUCTION

Cardiac ischemia, which is also present during exercise testing (ExT), has been shown to trigger cardiac troponin release (cTn) [1]. It has been also shown, that cTn might be increased in congestive heart failure (CHF) patients [2]. Thus, it confirms the theory of cTn release during the damage of myocardium, regardless of its cause. So far, all the published studies discussing the role of cTn release during ExT [3–9], have not provided definitive answers as to what conditions might be associated with this phenomenon, and whether there are any clinical implications of cTn release after ExT. The aim of our study was identify the factors predisposing to troponin I (cTnI) release after ExT, and to determine its clinical and prognostic implications in patients with stable coronary artery disease (CAD), referred for planned coronary angiography (ANG).

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METHODS

Study group

The study group consisted of all patients admitted to hospital who were referred for planned coronary ANG. The study had been approved by the Bioethics Committee of Centre for Postgraduate Medical Education.

Inclusion criteria were the following: confirmed stable CAD and ability to exercise on a treadmill. Exclusion criteria consisted of: contraindications to ExT, chronic diseases such as: cancer, hepatic cirrhosis, inflammatory diseases, autoimmunologic conditions, renal insufficiency and pulmonary embolism, NYHA class II–IV CHF, lack of written consent form and patients' cooperation. Baseline characteristics of all the study patients has been collected and included age, gander, initial diagnosis and history of myocardial infarctions (MI), time from the most recent infection(s) and frequency of infections in the past, presence of other coexisting diseases (diabetes, hypertension, heart defects, CHF), atherosclerosis risk factors (smoking, dyslipidaemia, obesity, family history of CAD, lack of physical exercise, chronic stress) and current medical treatment.

The ExT was performed using Bruce protocol or modified Bruce protocol, so that the total exercise time was 6–12 minutes and was limited by patients' symptoms. The indications for ExT, the test's layout specifications, and positive criteria of the test were in accordance with the most recent ACC/AHA coupled with current medications taken by study patients (e.g. digoxin) [10]. Duke index was calculated using the following formula: 5 × ST depression in mm — 4 × chest pain intensity (0 = no chest pain, 1 = chest pain not requiring the test to be stopped, 2 = chest pain requiring the test to be stopped) [11].

Biochemical studies

Before ExT initial measurements of cTnI, N-teminal pro B-type natriuretic peptide (NT-proBNP), high sensitive C-reactive protein (hsCRP), CK-MB levels were obtained. Furthermore, 8–12 hours after the test, cTnI was measured, followed by the assessment of cTnI, CK-MB at 24-hour time-point. To measure cTnI, MB-CPK, hsCRP we used immunochemical tests using Dimension platform (Dade Behring Diagnostics); for NT-proBNP we used electrochemiluminescent method and utilised Elecsys platform (Roche). Both cTnI and CK-MB were assessed right after collecting blood samples. In order to perform NT-proBNP i hsCRP measurements blood serum was stored in –70 degree of Celsius.

Echocardiography

All patients underwent ECHO within 7 days of taking blood samples. The transthoracic ECHO examination was performed using SONOS 5500 sonogram, and standard projections were used. The ejection fraction (EF) was measured using Simpson's method.

Coronary angiography

All patients underwent coronary ANG within 7 days of taking blood samples using Philips Integris CV coronary angiogram, utilising Judkins method. All coronary stenoses greater than 70% were considered significant/critical.

Follow up

After initial assessment, the long-term follow up was planned. During the follow up we used combined endpoint comprising: cardiac death, non-cardiac death, MI, and hospitalisation for unstable angina or CHF exacerbation, acute repeat percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) surgery. The cumulative event rate was calculated using the Kaplan-Meier method in the two study groups: patient with and without cTnI release measured after ExT. The event free survival was analysed from the time when the patient entered the study, to the time of the first event. The log-rank test was utilised to compare the survival curves.

Statistical analysis

All the study data was analysed using multifactor logistic regression model. The tests were two sided and statistical significance was considered at 5%. To analyse the cTnI levels we used a cut off point of 0.14 ng/mL, which correlated with the 99th percentile of the reference group for healthy population, and 20% coefficient of variation (CV) using Dimension platform (Dade Behring Diagnostics, USA) [12]. Standard parametric and non-parametric tests were used. The differences in categorical and continuous variables were measured using χ^2 and Student *t*-test, respectively.

RESULTS

118 patients without signs of CHF, referred for planned coronary ANG were included in the analysis. The study group's baseline characteristics are presented in Tables 1 and 2.

Eleven patients (9%) had significant icrease of cTnI ≥ ≥ 0.14 ng/mL [cTnI (+) group]. Patients in the cTnI (+) group, after 8–12 hours and at 24-hour mark after ExT, had significantly increased mean cTnI values, when compared to their baseline values. In the group of patients without increased cTnI at baseline [cTnI negative (–) group], the mean cTnI concentrations after 8–12 hours and at 24-hour mark after ExT were comparable to the baseline values (Table 3). We found no association between the cTnI release after ExT, and the multivessel CAD in the studied patients (Table 4). We performed the multivariable analysis in order to establish independent risk factors for cTnI release of ≥ 0.14 ng/mL, measured after ExT (Table 5). Independent risk factors were: EF ≤ 50%, max systolic blood pressure (SBP) > 160 mm Hg and limited physical activity. There was no increase in CK-MB in any patients after (8–12 h and 24 h) ExT.

After the mean of 35.5 months (15–49) of follow up, data have been collected to assess the combined endpoint. Due to several endpoints occurring in one patient, the number

Table 1. Baseline charecteristics

Study population	118
Age (min, max) [years]	(44, 77)
Mean ± SD	60.5 ± 8.9
Males	76 (64%)
Prior MI	54 (46%)
Diabetes	30 (25%)
Hypertension	71 (61%)
Hipercholesterolaemia	87 (74%)
Smoking	24 (20%)
History of PCI or CABG	26 (22%)
Family history of MI	29 (25%)
Obesity (BMI > 25)	54 (46%)
Physical activity:	
No	5 (4%)
Limited	29 (25%)
Average	46 (39%)
Significant	38 (32%)
Current medications:	
Statins	89 (75%)
Diuretics	5 (4%)
ACE inhibitors	72 (61%)
Beta-blockers	95 (81%)

MI — myocardial infarction; PCI — percutaneous coronary intervention; CABG — coronary artery bypass grafting, BMI — body mass index

Table 2. Study results

Exercise test	
Positive exercise test results [ExT (+)]	70 (59%)
Duke index	
(min, max)	(–19, 12)
Median (25%, 75%)	–1 (–6, 3)
Below –11	19 (16%)
Between –10 to 4	75 (64%)
Above 5	24 (20%)
Metabolic equivalents [Mets]:	
(min, max)	(3, 13.5)
Median (25%, 75%)	8 (7, 10.1)
Mets ≥ 7	90 (76%)
HR at rest in ExT	
(min, max)	(44, 114)
Mean ± SD	73 ± 13.5
Maximal HR during ExT	
(min, max)	(85, 236)
Mean ± SD	128 ± 24.5
Max SBP during ExT [mm Hg]	
(min, max)	(110, 230)
Mean ± SD	163 ± 25.3
EF [%]	
(min, max)	(45, 72)
Median (25%, 75%)	60 (50, 60)
Laboratory data	
cTnI [ng/mL]	
< 0.14	107 (91%)
≥ 0.14	11 (9%)
hsCRP prior to ExT > 3 mg/L	36 (30%)
NT-proBNP > 144 pg/mL	71 (60%)
Angiographic findings	
1-vessel disease	17 (14%)
2-vessel disease	33 (28%)
3-vessel disease	41 (35%)
2 or 3-vessel disease	74 (63%)
1–3 vessel disease	91 (77%)

HR — heart rate; SBP — systolic blood pressure; EF — ejection fraction; ExT — exercise test; hsCRP — high sensitive C-reactive protein

Table 3. Mean cTnI concentrations in group cTnI (+) and cTnI (–)

	cTnI (+)	cTnI (–)
cTnI [ng/mL] prior to ExT	0.04 ± 0.04	0.03 ± 0.04
cTnI [ng/mL] in 8–12 hours after ExT	0.1 ± 0.13	0.03 ± 0.04
cTnI [ng/mL] in 24 hours after ExT	0.39 ± 0.59	0.03 ± 0.04

of patients and clinical events were different (Table 6). The combined endpoint occurred in 3 patients (27%) in the cTnI (+) group, and in 18 patients (17%) in the cTnI (–) group (NS).

The cumulative event rates calculated for both groups using the Kaplan-Meier method and compared using the log-rank test did not differ between the study groups (Fig. 1).

DISCUSSION

Cardiac troponins exist in two forms. They are either combined with the contraction apparatus of the myocardial cells or dissolved in the cytoplasm, and account for 97% and 3% of the total troponin amount. Prolonged ischaemia and other processes of myocardial cell damage cause the release of the cTn combined with contraction apparatus. Troponin concentration change in blood serum after ischemic episode is associated with rapid cTn increase within 3–12 hours, peak concentration after 18–24 hours, and a slow decrease in concentration, which remains elevated until 4–7 days for cTnI and 10–14 days for cTnT.

It is important to establish the cause of elevated cTn in patients with stable CAD. It might be due to permanent or temporary myocardium cell damage during ischaemia or other processes such as, for example, myocytes apoptosis in CHF patients. It is also vital to determine whether the cTn elevation has an impact on long-term clinical outcomes. In our study, we found that 11 (9%) patients had cTnI concentration ≥ 0.14 ng/mL, of whom 5 (4%) patients had cTnI ≥ 0.26 ng/mL, which is a recommended cut-off point based on the

Table 4. Presence of 2- or 3-vessel disease in stable coronary artery disease confirmed angiographically and post exercise test cTnI release

	cTnI < 0.14	cTnI ≥ 0.14	Total
0–1 vessel disease	42 (95%)	2 (5%)	44
2–3 vessel disease	65 (88%)	9 (12%)	74
Total	107	11	118

Sensitivity = 12%, 95% CI: 6–22%; Specificity = 95%, 95% CI: 85–99%

Table 5. Risk factors of cTnI release (cTnI ≥ 0.14 ng/mL) after exercise test (logistic regression model)

Factor	OR (95% CI)	P
EF ≤ 50%	**6.6 (1.2–35.4)**	**0.027**
Age > 60 years		> 0.1
Males		> 0.1
Prior MI		> 0.1
Diabetes		0.07
Hypertension		> 0.1
Hypercholesterolaemia		> 0.1
Smoking		> 0.1
History of PCI or CABG		> 0.1
Family history of MI		> 0.1
Obesity (BMI > 25)		> 0.1
Physical activity		
No/limited *vs* average/significant	**5.5 (1.1–28.8)**	**0.04**
Duke index < 5		> 0.1
ExT (+)		> 0.1
HR at rest 60/min		> 0.1
Max HR > 120/min in ExT		> 0.1
Max SBP > 160 mm Hg in ExT	**6.3 (1.3–31.6)**	**0.025**
hsCRP > 3 mg/L		0.1
NT-proBNP > 144 pg/mL		> 0.1
2–3 vessel diesease		> 0.1

Abbreviations as in Tables 1 and 2

Table 6. Number of patients and total number of study endpoints during follow up

Clinical event	n = 118	No. of events
Cardiac death	1 (0.8%)	1
Non-cardiac death	4 (3%)	4
Recurrent MI	5 (4%)	8
Hospitalisation for CAD ex.	10 (8%)	16
Hospitalisation for CHF ex.	3 (3%)	4
Recurrent revascularisation	7 (6%)	8
CABG	15 (13%)	15
Emergent CABG	1 (0.8%)	1
PCI	12 (10%)	19
Emergent PCI	6 (5%)	6

MI — myocardial infarction; CAD — coronary artery disease; CHF — congestive heart failure; CABG — coronary artery bypass grafting, PCI — percutaneous coronary intervention

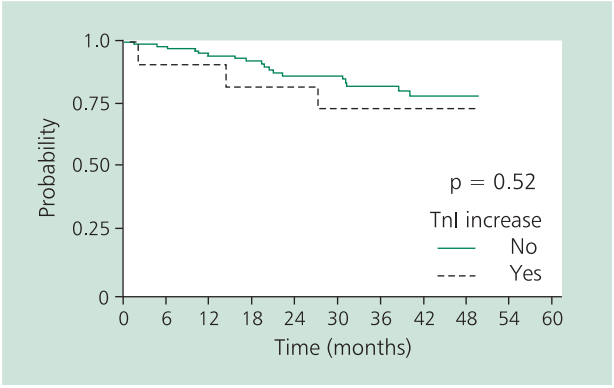

Figure 1. Incidence-free survival in cTnI (+) and cTnI (–) groups

CV ≤ 10, measured with Dimention platform (Dade Behring Diagnostics, USA).

In previously published studies, the frequency of elevated cTn following the ExT in stable CAD was 21.7%, as reported by Malyar et al. [3]. This high rate could have been associated with a low cut-off point (0.1 ng/mL) in contrast to an incidental rate of 0.7% (1 per 134 patients) reported by Choragudi et al. [4]. In the population studies, majority of patients were males, at the age of 58–60. Compared to the study by Malyar et al. [3], our patients had comparable history of prior MI, but significantly higher rates of revascularisation (22% *vs* 58%) and confirmed multivessel disease (63% *vs* 72%), although were less likely to exercise regularly (71% *vs* 40%). There was no correlation analysis done between cTnI, hsCRP and NT-proBNP, neither the authors assessed EF or presence of CHF in studied patients. Eryol et al. [5] performed a study in 100 patients with stable CAD, who underwent ExT and had troponin T (cTnT) measured before and after the test. Four (6.2%) patients with confirmed stable CAD had elevated cTnT, however in two the results of prior ExT were negative. Of patients who underwent ExT, it was showed that elevated cTnT levels were associated with 3-vessel CAD disease (p = 0.021). Eryol et al. [5] had also demonstrated that the duration of ExT was significantly shorter in patients with elevated cTnT, compared to patients with normal cTnT, (277.5 ± 81 s *vs* 428 ± 195 s, p = 0.024). Comparable to other studies [4, 6–8] we also did not find any correlation between the cTn release and the progression of atherosclerosis in patients undergoing ExT testing. Furthermore, neither we nor other authors documented any association between after ExT cTn release and the results of the ExT such as ST segment depression, required normali-

sation time, occurrence of chest pain, arrhythmias or exercise induced blood pressure drop.

So far, no study demonstrated EF ≤ 50% as an independent predictor of cTnI release after ExT. Amara et al. [2] and Gouzoumas et al. [13] described the phenomenon of cTn release in patients with advanced CHF without prior ExT. In the other study by Schultz et al. [9] the cTnI elevation above 0.1 ng/mL was described in 28 (21%) patients with EF of 31 ± 8%, who underwent ExT. Lack or insufficient physical exercise routine and the post testing cTn release has been described by Malyar et al. [3]. The demonstrated coexistence of the lack or insufficient exercise and the EF ≤ 50% may indicate asymptomatic at rest left ventricular dysfunction and cTn release process. Additionally, patients' incapability to perform strenuous exercise, and the need of high pressure generation > 160 mm Hg to overcome the exercise, in patients with average exercise performance (8 Mets), may also suggest the role of stress and exercise in the cTn release.

Middleton et al. [14] performed consecutive marker measurements study in 9 well-trained runners during a marathon (42.2 km), simulated on a treadmill. In all study patients, authors confirmed cTnT release between 60 and 120 minutes with markers normalisation within one hour after completion of the study protocol, and the following cTnT increase after 24 hours after the exercise in majority of runners.

In our study we did not find any prognostic value of the post ExT cTn release. Up to date, to our knowledge, there has been no study published describing such a relationship. Hshieh et al. [15] investigated other independent risk factors in patients with stable CAD undergoing ExT. Although authors measured only cTn prior to the ExT, they concluded that elevated cTnT was associated with a higher incidence rate of combined cardiovascular endpoint, as well as the elevated CRP, during 4.3 years of follow up. However, the relationship became insignificant after inclusion of other risk factors such as NT-proBNP and echocardiographically measured futures of the left ventricle.

Limitations of the study

The number of studied patients and events during follow-up was relatively low which made the assessment of prognostic value of cTnI release problematic.

CONCLUSIONS

1. Increase of cTnI after ExT in patients with stable CAD referred for planned coronary ANG occurs sporadically.
2. Independent risk factors associated with cTnI increase after ExT include the following: EF ≤ 50%, lack or insufficient physical activity and max SBP > 160 mm Hg during exercise.
3. Post ExT cTnT increase in patients with stable CAD did not correlate with number of atherosclerotic coronary vessels involved.
4. Increase of cTnI after ExT does not have any predictive value in respect to ACS and/or death during long-term follow up.

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Ocena warunków i znaczenia uwalniania troponiny po teście wysiłkowym u pacjentów ze stabilną chorobą wieńcową

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Streszczenie

Wstęp: Wzrost stężenia troponin sercowych (cTn) jest wyrazem uszkodzenia serca, niezależnie od przyczyny. Test wysiłkowy (TW) jest użyteczną klinicznie metodą oceny zagrożenia niedokrwieniem.

Cel: Ustalenie czynników powodujących uwalnianie troponiny I (cTnI) po TW i znaczenia klinicznego tego zjawiska, w tym również rokowniczego, u pacjentów ze stabilną chorobą wieńcową (ChW) kierowanych do koronarografii.

Metody: Do badania włączono 118 pacjentów ze stabilną ChW, bez objawów skurczowej niewydolności serca, kierowanych do planowej koronarografii. Po pobraniu krwi do oznaczeń NT-proBNP, hsCRP, cTnI, CK-MB u wszystkich badanych, wykonano maksymalny TW. Dodatkowo oznaczono cTnI po 8–12 i wraz z CK-MB po 24 godzinach. W ciągu 7 dni wykonano koronarografię i badanie ECHO serca. Po średnio 35,5 miesiąca zebrano informacje o wydarzeniach klinicznych.

Wyniki: U 11 pacjentów (9%) obserwowano wzrost cTnI ≥ 0,14 ng/ml (99. percentyl dla populacji zdrowej) w ciągu 24 godzin po TW. U chorych z frakcją wyrzutową ≤ 50%, małą aktywnością fizyczną w życiu codziennym oraz w czasie wysiłku z maksymalnymi wartościami skurczowego ciśnienia tętniczego (maks. sRR) > 160 mm Hg stwierdzono częstsze uwalnianie cTnI po TW (odpowiednio OR: 6,6; 95% CI: 1,2–35,4; p = 0,027; OR: 5,5; 95% CI: 1,1–28,8; p = 0,04; OR: 6,3; 95% CI: 1,3–31,6; p = 0,025). Wzrost cTnI po TW nie korelował z występowaniem wielonaczyniowej ChW i nie wpływał na występowanie wydarzeń klinicznych w przyszłości.

Wnioski: Chorzy z frakcją wyrzutową ≤ 50%, z małą aktywnością fizyczną w życiu codziennym oraz z maks. sRR > 160 mm Hg w trakcie wysiłku, mieli większe szanse na uwolnienie cTnI po TW. Zjawisko to nie zależało od objawów niedokrwienia w TW ani od liczby zmienionych miażdżycowo tętnic wieńcowych w koronarografii. Wzrost stężenia cTnI po TW nie miał znaczenia prognostycznego dla wystąpienia ostrych zespołów wieńcowych i/lub zgonów w okresie obserwacji odległej.

Słowa kluczowe: test wysiłkowy, troponina I, stabilna choroba wieńcowa

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