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The impact of physical training on endothelial function in myocardial infarction survivors: pilot study

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Abstract

Background: Endothelial dysfunction (ED) may indirectly influence the outcome of patients with coronary artery disease.

Aim: To assess the influence of cardiac rehabilitation (CR) on endothelial function in patients after ST-segment elevation myocardial infarction (STEMI).

Methods: Twenty-nine patients scheduled for CR were included in the study. CR began at least four weeks after STEMI and consisted of 12 or 24 training sessions. Endothelial function assessment was performed before and after CR, using reactive hyperaemia peripheral arterial tonometry.

Results: Before the CR, ED was diagnosed in 16 of 29 (55.2%) patients. A total of 25 patients had two assessments of endothelial function: before and after CR. In univariate analysis the factors of negative response of endothelial function to CR were: higher baseline hyperaemia index (lnRHI) (odds ratio [OR] for positive response to CR 0.01; 95% confidence interval [CI] 0.00–0.33; p = 0.01) and higher peak serum troponin I level during index hospitalisation (OR 0.97; 95% CI 0.94–1.00; p = 0.04). The independent, negative predictor of response to CR was lnRHI (OR 0.01; 95% CI 0.01–0.16; p = 0.03). Patients training for 24 sessions (n = 16) had similar lnRHI changes to those of patients training for 12 sessions (n = 9); [0.16 (–0.06)–0.30 vs. 0.10 (0.05–0.15); p = 0.44, respectively].

Conclusions: ED is a frequent abnormality in STEMI survivors. Despite the lack of statistically significant improvement of endothelial function after CR in the analysed group of patients, some factors can influence the efficacy of this type of physical activity. The best effect of CR on endothelial function was observed in patients with baseline ED.

Key words: cardiac rehabilitation, endothelial dysfunction, myocardial infarction

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INTRODUCTION

Endothelial dysfunction (ED) is strongly related to coronary artery disease (CAD); therefore, we can expect a significantly higher frequency of ED in patients after myocardial infarction (MI) [1]. ED can be reversible, so early diagnosis is especially important. In 1986 Ludmer et al. [2] showed that human coronary arteries with atherosclerotic plaque present a paradoxical reaction to infusion of acetylcholine. However, in further studies it was noticed that ED is also presented in normal vessels without morphological changes, in patients with risk factors of CAD [3]. Thus, ED is a preclinical manifestation of atherosclerosis. Most popular methods of endothelial function measurements include: infusion of acetylcholine to coronary arteries, flow-mediated dilatation of brachial artery (FMD), reactive hyperaemia flow, and skin reactive hyperaemia. The correlation between results obtained with these methods is low [4]. In practice, the invasive method is reserved for patients who undergo other invasive procedures. In our study, we chose reactive hyperaemia peripheral arterial tonometry (RH-PAT) because of its objectivity and ease of performing the measurements. The sensitivity and specificity of this method in identifying patients with CAD is estimated at 80% and 85%,

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respectively [5]. RH-PAT measures changes of the tension in peripheral vascular system related to hyperaemia [6–8]. Previous studies showed that patients with ED in coronary vessels had worse response for transitory ischaemia than patients with normal endothelial function. Therefore, RH-PAT can be a useful method for the assessment of function of coronary vessels' endothelium [5]. Suwaidi et al. [9] showed that in 28-month follow-up of patients with CAD, cardiac events were noticed only in patients with severe ED [10]. Also, ED increased the risk of cerebrovascular events by a factor of approximately four.

The beneficial influence of cardiac rehabilitation (CR) on long term follow-up of patients after MI is well known [11–14]. In this group, CR should be a life-long recommendation. Especially important is the second phase of CR. Although we know that CR reduces the mortality, there is still not enough information on its effect on endothelial function. The objective of this study was to assess the influence of ambulatory CR on endothelial function and prognostic factors of improvement of endothelial function in patients after ST-elevation MI (STEMI).

METHODS

Study population

This prospective study included 29 adult patients with diagnosis of STEMI during index hospitalisation, who were scheduled for CR and agreed to participate in two assessments of endothelial function by RH-PAT with Endo-PAT2000®, ITAMAR device. All patients were hospitalised between August 2012 and July 2013. Assessment of endothelial function was performed between October 2012 and September 2013. STEMI definition was based on the current guidelines of the European Society of Cardiology [15, 16]. The other inclusion criteria were: invasive treatment of MI with stent implantation during index hospitalisation and optimal pharmacotherapy after STEMI.

Exclusion criteria were as follows: motor disabilities that made exercise training impossible, myocarditis (current or in the past), decompensated heart failure, uncontrolled cardiac arrhythmias, severe arterial hypertension, severe aortic stenosis, head injury in the last three months, advanced neoplastic disease, persistent inflammatory state, and pregnancy.

Approval for the study was obtained from the Ethical Review Board (35/2012) and every patient signed an informed consent form to participate in the study with endothelial function assessment by RH-PAT

Clinical assessment and management during hospitalisation.

During index hospitalisation, data about clinical state and medical history were collected from all patients. Each patient underwent an urgent primary percutaneous coronary intervention (pPCI). Procedural success, defined as stent implantation with residual stenosis < 10% and thrombolysis in myocardial infarction (TIMI) grade 3 flow, was observed in 96.6% (n = 28) of patients. In one (3.4%) patient pPCI was unsuccessful, with TIMI grade 0 flow, because of the inability of widening an obstructed coronary artery. The analysed laboratory tests included: troponin I, creatine kinase-MB mass, haemoglobin, white blood cells, and N-terminal fragment of B-type natriuretic peptide. In all patients echocardiography was performed. The baseline characteristics of patients are presented in Table 1.

Table 1. Baseline characteristics of the study group

Parameter	Value
Male	26 (89.7%)
Age [years]	54 (51–62)
Systolic BP [mm Hg]	130 (110–140)
Diastolic BP [mm Hg]	80 (70–90)
Heart rate [bpm]	68 (64–73)
Weight [kg]	88 (80–92)
Body mass index [kg/m^2]	29.0 (26.1–30.1)
Haemoglobin level [g/L]	144 (134–150)
Troponin I — peak level [ng/L]	25.6 (10.4–52.8)
CK-MB — maximal level (n = 26) [μg/L]	55.2 (13.2–162.3)
NT-proBNP (n = 19) [pg/mL]	745 (447–1571)
History of previous MI	1 (3.4%)
Hypertension	17 (58.6%)
Diabetes mellitus type 2	4 (13.8%)
Atrial fibrillation	0 (0.0%)
History of smoking	21 (72.4%)
Procedural success (TIMI 3 flow)	28 (96.6%)
Current treatment:	
Beta-blockers	27 (93.1%)
ACEI	28 (96.6%)
Statin	29 (100%)
Diuretics	8 (27.6%)
Aspirin	29 (100%)
$P2Y_{12}$ receptor inhibitors	28 (96.6%)
Nitrates	1 (3.4%)
Calcium channel blockers	2 (6.9%)
Angiotensin II receptor antagonists	1 (3.4%)
Echocardiographic measurements:	
LVEF [%]	48 (45–55)
Baseline exercise test:	
Exercise capacity [METs]	8.7 (7.0–10.2)

Values are median (interquartile range) or n (%); ACEI — angiotensin converting enzyme inhibitor BP — blood pressure; CK-MB — creatine kinase-MB mass; LVEF — left ventricular ejection fraction; METs — metabolic equivalents; MI — myocardial infarction; NT-proBNP — N-terminal fragment of B-type natriuretic peptide; TIMI — thrombolysis in myocardial infarction grade flow

Cardiac rehabilitation

An electrocardiographic exercise test (ECG ExTest) was performed at least four weeks after STEMI. After determining the baseline exercise capacity, which was not associated with any signs of ongoing ischaemia in the ECG ExTest, patients were included in training cycles on bicycles (REHA ergometer 900® Ergoline®). Patients were divided into two groups according to their preferences. The first group was asked to complete 12 training sessions and the second group 24 training sessions. The load was determined on the basis of heart rate reserve (HRR). Patients in each group trained 3–4 times per week for 40–50 min per session at a target heart rate zone.

Before each training session a physician (internist or cardiologist) evaluated each patient for fitness to train. A physiotherapist supervised the group during the training, and a physician re-evaluated the patients after the session. Using Schiller software for exercise rehabilitation, the initial workload was determined to obtain the target heart rate. The software then modified the workload to maintain the set heart rate. The patient was instructed to cycle at 55–65 rpm. A single training session consisted of a 3–5-min warm-up period, the actual training lasted for 30 min, followed by a 5-min cool-down period. After the session, patients were observed and monitored for another 10 min. Upon completion of the exercise rehabilitation, patients were given instructions regarding physical activity during daily life. They were also encouraged to participate in various forms of exercise (walking, jogging, swimming, dancing) to maintain the effects of rehabilitation. After CR each patient underwent an ECG ExTest to check the influence of training on exercise capacity.

The heart rate reserve was counted according to the formula: HRR = HR max – HR rest (HR max — maximal heart rate registered during ECG exercise test, HR rest — heart rate in rest). To set the training HR zone we used the following formula: HR training zone = (HR rest + 40–80% HRR) ± 10%.

Moreover, CR consisted of psychological support and lectures about diet and lifestyle changes. The rehabilitation centre cooperated with a psychologist and dietitian, who worked with patients. They were available for the patients during the training sessions. There were also organised meetings for patients during which there was a discussion on problematic topics.

RH-PAT assessment

Two endothelial function assessments were planned for each patient. The first was done seven days before the CR and the second seven days after the last session of CR. The second test was completed in 25 of 29 patients. Two patients denied participating in the second assessment, one patient did not finish CR because of recurrent chest pain during exercise, and other one because of active urolithiasis. According to previous studies the cut-off point for ED was established as the natural logarithm of reactive hyperaemia index (lnRHI) = 0.51. Each higher value was considered as normal endothelial function. Positive response to CR was defined as an increase of lnRHI value after all planned training sessions.

The value of lnRHI was measured by Endo-PAT2000® (ITAMAR) device. The method of endothelial function assessment was based on arterial response of peripheral vascular tone to 5-min occlusion of brachial artery. Before each test, the patient rested for 15 min in neutral surroundings to stabilise all vital parameters. The sensors were placed on index fingers of both arms. All phases of the test were performed in the sitting position. During the first 5 min baseline data were obtained. Then, a blood pressure cuff was inflated to 200 mm Hg, or 40 mm Hg above systolic blood pressure, for the next 5 min. Thereafter, circulation in the arm was restored and data were collected for the next 5 min. Response to the hyperaemia was corrected for systemic changes based on the reading from the other arm's sensor. The software version 3.4.4 provided by the ITAMAR company calculated the value of lnRHI automatically.

Statistical analysis

Categorical data were presented as percentages, and if necessary the frequency was also noted. All continuous variables were presented as median value and interquartile range (IQR). Fisher's exact test and Mann-Whitney U test were performed for the comparison between groups, for categorical variables and continuous variables, respectively. The changes in lnRHI values caused by CR were calculated by Wilcoxon signed-rank test. To determine the factors of positive response to CR, univariate and multivariable logistic regression were performed. To multivariable logistic regression all factors with p-values ≤ 0.1 in univariate analyses were included. Correlations between continuous variables were assessed by Spearman correlation coefficients. Statistical significance was considered for p-values lower than 0.05. The size of the group was calculated with the following assumption: baseline mean lnRHI 0.5; change of lnRHI after CR 0.25; and standard deviation 0.2. With these values the number of patients needed to reach a power of the study of at least 0.8 was equal to 24. Statistical analyses were made using SAS software, version 9.2.

RESULTS

Endothelial dysfunction in patients after MI

In the first assessment, ED was diagnosed in 16 of 29 (55.2%) patients. Median values of lnRHI in patients with and without ED were 0.37 (0.27–0.42) and 0.68 (0.60–0.92), respectively. There were no significant differences between patients with ED and with normal endothelial function according to age, sex, clinical data, current treatment, left ventricular ejection fraction (LVEF), and exercise capacity (metabolic equivalents [METs]). However, we noticed a trend for lower values of baseline maximum exercise capacity in the ED group than in patients with normal endothelial function; 7.7 (7.0–9.5) vs. 9.4 (8.6–11.8) METs; p = 0.09. Moreover, there was

Table 2. Characteristics of patients with and without endothelial dysfunction

	Endothelial dysfunction (n = 16)	Normal endothelial function (n = 13)	P
Male	14 (87.5%)	12 (92.3%)	1.00
Age [years]	55 (52–64)	54 (44–60)	0.39
Systolic BP [mm Hg]	138 (115–150)	125 (110–130)	0.13
Diastolic BP [mm Hg]	90 (75–100)	80 (70–85)	0.11
Heart rate [bpm]	66 (64–79)	68 (62–70)	0.90
Weight [kg]	87 (79–93)	88 (84–92)	0.66
Body mass index [kg/m^2]	29.2 (26.6–30.1)	28.0 (26.0–31.0)	0.95
Haemoglobin level [g/L]	144 (131–152)	144 (134–149)	0.82
White blood cells count [$\times 10^9$/L]	9.5 (6.6–10.7)	9.4 (7.2–11.0)	0.73
Troponin I — peak level [ng/L]	20.0 (3.7–36.0)	35.0 (16.5–58.8)	0.17
CK-MB — maximal level (n = 26; 14/12) [μg/L]	42.3 (5.24–197.0)	70.6 (42.5–134.0)	0.70
NT-proBNP (n = 19; 10/9) [pg/mL]	1169 (479–1591)	586 (290–1199)	0.14
History of previous MI	0 (0.0%)	1 (7.7%)	0.45
Hypertension	11 (68.8%)	6 (46.2%)	0.27
Diabetes mellitus type 2	3 (18.8%)	1 (7.7%)	0.61
History of smoking	13 (81.3%)	9 (69.2%)	0.68
Procedural success (TIMI 3 flow)	15 (93.8%)	13 (100%)	1.0
LVEF [%]	48 (42–55)	50 (46–55)	0.36
Exercise test:			
Baseline maximum exercise capacity [METs] (n = 25; 14/11)	7.7 (7.0–9.5)	9.4 (8.6–11.8)	0.09
Exercise capacity after CR [METs] (n = 25; 14/11)	10.3 (10.0–11.7)	12.4 (10.2–15.3)	0.12
Change in exercise capacity after CR [METs] (n = 25; 14/11)	2.6 (1.6–3.2)	1.8 (1.0–2.7)	0.27
Endothelial function assessment:			
Baseline lnRHI (n = 25; 14/11)	0.37 (0.27–0.42)	0.68 (0.60–0.92)	< 0.0001
lnRHI after CR (n = 25; 14/11)	0.53 (0.43–0.66)	0.67 (0.40–0.87)	0.40
Change of lnRHI after CR (n = 25; 14/11)	0.16 (0.09–0.17)	–0.04 [(–0.40)–0.17]	0.08
Endothelial dysfunction after CR (n = 25; 14/11)	38.5%	33.3%	1.0

Values are median (interquartile range) or n (%); CR — cardiac rehabilitation; lnRHI — natural logarithm of reactive hyperaemia index; rest abbreviations as in Table 1

a trend for better response of endothelial function on CR in patients with baseline ED than in patients with normal function of endothelium; change of lnRHI: 0.16 (0.09–0.17) vs. –0.04 [(–0.40)–0.17], respectively; p = 0.08. The detailed characteristics and differences between two groups of patients, based on endothelial function, are summarised in Table 2.

Impact of CR on endothelial function

A total of 25 patients had two assessments of endothelial function: before and after CR programme. We noticed no statistically significant differences between lnRHI values before and after CR; 0.49 (0.36–0.67) vs. 0.58 (0.43–0.74); p = 0.14. Baseline and final values of lnRHI for particular patients are presented in Figure 1. In univariate analysis, factors of negative response of endothelial function to CR were: higher baseline lnRHI (odds ratio [OR] for positive response to CR 0.01; 95% confidence interval [CI] 0.00–0.33; p = 0.01) and higher maximum serum troponin I level during index hospitalisation (OR 0.97; 95% CI 0.94–1.00; p = 0.04). There was also a trend for worse efficacy of CR on endothelial function in patients with higher baseline exercise capacity (OR 0.72; 95% CI 0.48–1.07; p = 0.10). On the other hand, we noticed a trend for positive response of endothelial function to CR in patients with higher LVEF (OR 1.13; 95% CI 0.99–1.29; p = 0.07). After backward elimination multivariate regression analysis only higher baseline lnRHI reached statistical significance, as a negative predictor of response to CR (OR 0.01; 95% CI 0.01–0.16; p = 0.03). The influence of other factors on CR efficacy is presented in Table 3.

We performed also the analysis of response to CR in subgroups of patients with and without baseline ED. Although there was no difference in the values of lnRHI before and

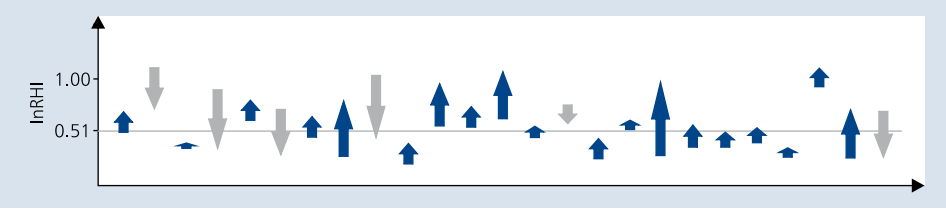

Figure 1. Impact of cardiac rehabilitation on endothelial function for particular patients. Back of the arrow presents natural logarithm of reactive hyperaemia index (lnRHI) value before rehabilitation, and head of the arrow points lnRHI value after cardiac rehabilitation. lnRHI = 0.51 is cut-point value for endothelial dysfunction

Table 3. Influence of factors on positive response to cardiac rehabilitation

	Univariate analysis (odds ratio; 95% CI)	P	Multivariate analysis (odds ratio; 95% CI)	P
Male	0.95 (0.08–11.9)	0.97		
Age [years]	1.00 (0.92–1.10)	0.96		
Systolic BP [mm Hg]	1.00 (0.96–1.05)	0.86		
Diastolic BP [mm Hg]	1.01 (0.95–1.08)	0.67		
Heart rate [bpm]	1.03 (0.94–1.12)	0.53		
Body mass index [kg/m^2]	0.92 (0.75–1.16)	0.46		
Haemoglobin level [×10 g/L]	1.20 (0.81–1.77)	0.35		
White blood cells count [×10^9/L]	1.20 (0.82–1.77)	0.36		
Troponin I — peak level [ng/L]	0.97 (0.94–1.00)	0.04	0.99 (0.89–1.09)	0.76
CK-MB — maximal level (n = 26) [μg/L]	1.00 (0.99–1.01)	0.95		
NT-proBNP (n = 19) [pg/mL]	1.00 (1.00–1.00)	0.78		
History of previous MI	–	1.0		
Hypertension	3.25 (0.66–15.9)	0.15		
Diabetes mellitus type 2	0.47 (0.06–3.97)	0.49		
Atrial fibrillation	–	1.0		
History of smoking	0.60 (0.09–3.89)	0.59		
Procedural success (TIMI 3)	–	1.0		
LVEF [%]	1.13 (0.99–1.29)	0.07	1.86 (0.95–3.66)	0.07
Baseline exercise capacity [METs]	0.72 (0.48–1.07)	0.10	1.60 (0.39–6.53)	0.51
Baseline lnRHI	0.01 (0.00–0.33)	0.01	0.01 (0.00–0.16)	0.03

In case of questionable validity of the model fit, "–" statement was used. Values are odds ratio (95% confidence interval [CI]); abbreviation as in Tables 1 and 2

after rehabilitation in the entire study population, we noticed statistically significant improvement of endothelial function in patients with baseline ED — before: 0.37 (0.27–0.42) vs. after: 0.53 (0.43–0.66); p = 0.0002. In patients without ED before CR, there was no significant change of lnRHI value — before: 0.68 (0.60–0.92) vs. after: 0.67 (0.40–0.87); p = 0.48 (Table 4).

Finally, the comparison of two subgroups of patients according to the number of training sessions was made (24 vs. 12 sessions). There were no significant differences in endothelial function between these patients before and also after CR. The patients training for 24 sessions (n = 16) had similar lnRHI changes as patients training for 12 sessions (n = 9); 0.16 [(–0.06)–0.30] vs. 0.10 (0.05–0.15); p = 0.44, respectively (Table 5).

DISCUSSION

The present study shows that the frequency of ED is relatively high in patients after MI. The improvement of endothelial function after CR in the entire group of patients was not statistically significant. However, better baseline endothelial function, higher peak serum troponin I level during STEMI-related hospitalisation, and higher baseline exercise capacity seem to be negative predictors of response to CR in the aspect of ED. The opposite effect is observed in patients with higher LVEF.

Table 4. Changes of natural logarithm of reactive hyperaemia index (lnRHI) values after cardiac rehabilitation (CR) according to baseline endothelial function

	Total (n = 25)	Endothelial dysfunction (n = 13)	Normal endothelial function (n = 12)
Baseline lnRHI	0.49 (0.36–0.67)	0.37 (0.27–0.42)	0.68 (0.60–0.92)
lnRHI after CR	0.58 (0.43–0.74)	0.53 (0.43–0.66)	0.67 (0.40–0.87)
Change of lnRHI after CR	0.13 (0.02–0.17)	0.16 (0.09–0.17)	–0.04 [(–0.40)–0.17]
P-value for change of lnRHI	0.14	0.0002	0.48

Values are median (interquartile range)

Table 5. Changes of natural logarithm of reactive hyperaemia index (lnRHI) values after cardiac rehabilitation (CR) according to number of training sessions

	12 training sessions (n = 9)	24 training sessions (n = 16)	P-value for difference between subgroups
Baseline lnRHI	0.47 (0.37–0.67)	0.51 (0.28–0.69)	0.77
lnRHI after CR	0.53 (0.32–0.62)	0.63 (0.44–0.76)	0.37
Change of lnRHI after CR	0.10 (0.05–0.15)	0.16 [(–0.06)–0.30]	0.44
P-value for change of lnRHI	0.57	0.21	

Values are median (interquartile range)

Interestingly, the number of training sessions does not seem to have an influence on endothelial function after CR.

Correlation between coronary artery disease and endothelial dysfunction

Because of the strong association between atherosclerosis and ED, impaired endothelial function is frequent in patients with CAD. Among our patients, more than the half had ED. In a previous study, in which RH-PAT was used, Toggweiler et al. [17] showed that 46% of patients with chronic CAD and 58% with acute CAD had ED. Interestingly, in this study, ED was also present in 67% of patients without CAD but with three or more risk factors of CAD. According to previous studies, there is a well-established connection between ED and risk factors of CAD such as: male gender, age, high serum level of cholesterol, family history of CAD, hypertension, diabetes mellitus (DM), and total number of risk factors [18–22]. Conversely, we did not notice any particular factor in our study, which might be related to the incidence of ED. We detected a trend for higher values of systolic and diastolic arterial pressure in patients with ED. The incidence of hypertension in this group was also higher. However, these differences did not reach statistical significance.

Efficacy of cardiac rehabilitation on endothelial function improvement

Based on previous studies, it is hard to clearly determine the influence of CR on endothelial function. Similarly to our study, Cornelissen et al. [23] did not find any substantial changes of lnRHI values in patients with stable CAD after CR. However, they observed an improvement of endothelial function assessed by FMD. It confirms how difficult it is to objectively evaluate endothelial function. Partially, this difference may result from the fact that each method measures endothelial function in different vessel beds. FMD focuses on macrovascular, while RH-PAT is mainly based on microvascular system assessment [24].

Low baseline lnRHI seems to be the most interesting prognostic factor that is associated with positive response to CR. In patients with baseline ED, efficacy of CR was considerably higher. Similar results were presented in two studies, which determined the influence of aerobic training on patients with DM type 1 and type 2 [25, 26]. In both of these studies, a positive effect of rehabilitation was noticed. What is important, almost all these patients had baseline ED, which is a common finding in patients with DM. This observation confirms the very important role of aerobic training in patients with current ED, because the benefits for these patients may be more significant. We could also consider endothelial function assessment after MI, and, in particular, encourage patients with ED to participate in CR programmes.

Limitations of the study

The main limitation of this study was the number of patients. The sample size was calculated for 50% improvement of lnRHI, which could be hard to achieve. Nowadays we cannot determine how large an increase in lnRHI will influence patients' outcomes. Moreover, it was difficult to select a proper control group. The favourable effect of CR on the outcome of patients after MI is well established. Hence, excluding

patients from the CR programme should be considered as unethical. Finally, it is not possible to exclude the influence of the period of time after STEMI on changes in endothelial function, even without aerobic training. This effect might be assessed by comparison of effects after 12 and 24 training sessions. However, it was not possible to perform proper randomisation, because of personal preferences of patients on the number of training sessions. Some patients wanted to reduce the number of training sessions, while others, after 12 training sessions, wanted to prolong their CR programmes.

CONCLUSIONS

Endothelial dysfunction is a relatively frequent abnormality in STEMI survivors. Despite the fact that no improvement of endothelial function after CR was shown in the analysed group of patients, some factors can influence the efficacy of this type of physical activity. The largest effect of CR on endothelial function was observed in patients with baseline ED.

Conflict of interest: *none declared*

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Wpływ wysiłku fizycznego na funkcję śródbłonka u pacjentów po zawale serca: badanie pilotażowe

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Streszczenie

Wstęp: Dysfunkcja śródbłonka (ED) może pośrednio wpływać na rokowanie pacjentów z chorobą wieńcową. Jednym ze sposobów poprawy funkcji śródbłonka jest aktywność fizyczna.

Cel: Celem niniejszego badania była ocena wpływu ambulatoryjnej rehabilitacji kardiologicznej (CR) na funkcję śródbłonka oraz identyfikacja predyktorów poprawy funkcji śródbłonka u chorych po zawale serca z uniesieniem odcinka ST (STEMI).

Metody: Do badania włączono 29 dorosłych pacjentów z rozpoznanym STEMI podczas hospitalizacji, u których zaplanowano CR. Rehabilitację kardiologiczną rozpoczynano po ≥ 4 tygodniach po zawale serca i składała się ona z 12 lub 24 sesji treningowych. Ocenę funkcji śródbłonka przeprowadzono dwa razy — przed i po CR. W tym celu stosowano pomiar zmian napięcia ścian naczyń w przebiegu reaktywnego przekrwienia (*reactive hyperemia peripheral arterial tonometry*).

Wyniki: Przed CR ED stwierdzono u 16 z 29 (55,2%) chorych. Nie zaobserwowano istotnych różnic między pacjentami z ED i bez ED w zakresie wieku, płci, danych klinicznych, aktualnego leczenia, pomiarów echokardiograficznych i danych uzyskanych podczas testu wysiłkowego. Łącznie u 25 pacjentów wykonano dwie oceny funkcji śródbłonka — przed i po CR. W analizie jednoczynnikowej czynnikami negatywnej odpowiedzi funkcji śródbłonka na CR były: wyższy bazowy logarytm naturalny indeksu przekrwienia reaktywnego (lnRHI) (iloraz szans [OR] dla pozytywnej odpowiedzi na CR 0,01; 95% przedział ufności [CI] 0,00–0,33; p = 0,01) oraz wyższe, maksymalne stężenie troponiny I w surowicy w trakcie hospitalizacji (OR 0,97; 95% CI 0,94–1,00; p = 0,04). W analizie wieloczynnikowej jedynie lnRHI pozostał niezależnym, negatywnym predyktorem odpowiedzi funkcji śródbłonka na CR (OR 0,01; 95% CI 0,01–0,16; p = 0,03). Zmiany lnRHI były zbliżone w przypadku treningów składających się z 24 sesji (n = 16) oraz 12 sesji (n = 9); odpowiednio: 0,16 [(–0.06)–0.30] *vs*. 0,10 (0,05–0,15); p = 0,44.

Wnioski: Dysfunkcja śródbłonka występuje stosunkowo często wśród pacjentów po STEMI. Pomimo braku istotnej poprawy czynności śródbłonka po CR dla całej badanej grupy, niektóre czynniki mogą wpływać na skuteczność tego typu aktywności fizycznej. Największy, pozytywny wpływ CR na czynność śródbłonka zaobserwowano u pacjentów z wyjściową ED.

Słowa kluczowe: dysfunkcja śródbłonka, zawał serca, rehabilitacja kardiologiczna

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