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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Effects of six-week cardiac rehabilitation and exercise on adiponectin in patients with acute coronary syndrome

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Abstract

Background: Increased adiponectin is a result of an anti-arteriosclerotic effect and is related to the prevention of arteriosclerosis. However, it is uncertain whether cardiac rehabilitation and exercise (CRE) increase adiponectin in patients after acute coronary syndrome (ACS).

Aim: To assess the effects of CRE intervention on adiponectin in patients after ACS.

Methods: Forty four patients participated in a cardiac rehabilitation programme after receiving percutaneous coronary intervention. The participants were divided into either an intervention (CRE) or a control (CON) group. Assessments were made at baseline and six weeks after the intervention for adiponectin, interleukin-6 (IL-6), tumour necrosis factor-α (TNF-α); high sensitivity C-reactive protein (hs-CRP), waist-to-hip ratio (WHR), and exercise duration.

Results: After six weeks of exercise training, adiponectin significantly increased in both CRE and CON ($p < 0.001$ and $p = 0.009$, respectively). Adiponectin showed a significantly greater increase in CRE than in CON ($p = 0.032$). Significant differences were not observed in IL-6, TNF-α, and hs-CRP between the groups. However, VO_{2max} and exercise duration significantly increased in CRE ($p < 0.001$). Significant increases in VO_{2max} and exercise duration were also observed in CRE but not in CON ($p < 0.001$). WHR significantly decreased in CRE, with no significant change in CON ($p < 0.05$). The difference in adiponectin between the groups showed a significantly inverse relationship with the difference in WHR ($R^2 = –0.376$, $p = 0.034$).

Conclusions: Adiponectin and cardiopulmonary fitness were significantly increased in CRE after six weeks of intervention. Although reductions in inflammatory markers were not observed, a significant inverse correlation was observed between the changes in adiponectin and WHR in CRE. Therefore, six weeks of short-term CRE intervention had a significant anti-inflammatory effect.

Key words: cardiovascular disease, percutaneous coronary intervention, maximal oxygen uptake, sub-rate pressure product

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INTRODUCTION

Coronary artery diseases (CAD) are caused by the development, growth, and rupture of plaque on the arterial wall. Such a plaque developing process is initiated by inflammation on the artery wall [1]. Adiponectin is deposited on the subendothelial layer of the damaged vessel [2], inhibiting the expression of adhesion molecules on the endothelial cells, which in turn reduces mononuclear cell adhesion [3]. Moreover, adiponectin inhibits macrophage to form cell conversions, tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) productions, and high sensitive C-reactive protein (hs-CRP) secretion in macrophage, as well as blood vessel proliferation in smooth muscles [4–6].

Therefore, adiponectin plays an important role in preventing arteriosclerosis by inhibiting such arteriosclerosis inducing inflammatory responses (anti-arteriosclerosis) [7, 8]. Adiponectin is lower in blood concentration of patients with obesity, diabetes, and CAD [3, 9]. A large-scale study

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on adiponectin and CAD found a considerably lowered risk of myocardial infarction (MI) in subjects with higher adiponectin concentrations [10]. In this case-control study, 18,255 participants free of diagnosed cardiovascular disease were followed-up for six years for the development of MI. 266 men developed nonfatal and fatal MI over the course of the six years. The results showed that high plasma adiponectin concentrations were associated with lower risk of MI. Based on such studies, researchers have recently focused on establishing the relationship between adiponectin and CAD.

As for adiponectin and exercise, although some researchers have reported increased adiponectin with exercise intervention [11], others did not find such significant changes [12, 13]. Cardiac rehabilitation is a programme designed to improve quality of life for patients with CAD by significantly improving exercise capacity, blood lipids, obesity index, psychosocial functions, fatal MI, and reducing the death rate [14, 15]. In a recent study, significant reductions in inflammatory cytokines and an improvement in cardiopulmonary fitness were observed after 14 weeks of cardiac rehabilitation and exercise (CRE) intervention [16]. Despite its importance, six weeks of a short term exercise intervention on adiponectin has not been investigated in previous studies. Furthermore, only a few studies have reported on the effects of CRE on adiponectin.

Thus, the effects of a six-week CRE intervention after a percutaneous coronary intervention (PCI) on adiponectin were investigated, along with inflammatory cytokines, and cardiopulmonary fitness [16].

METHODS

Subjects and study protocol

Patients with MI or unstable angina were referred by cardiologists to participate in a cardiac rehabilitation programme held at the cardiac rehabilitation clinic of Inje University Sanggye-Paik Hospital, Seoul, Korea. Fifty patients were recruited to participate in the study. The purpose and protocol of the study were fully explained verbally and with documented forms explaining the study procedures. Written consent was given by each subject prior to participating in the study. The purpose, procedures and protocols were approved by the Institutional Ethical Committee of Inje University Sanggye-Paik Hospital.

All the patients had successfully undergone a PCI approximately 7–10 days prior to participating in the study. Exclusion criteria were: left ventricular ejection fraction < 40%, a history of previous myocardial revascularisation (i.e. coronary artery bypass graft), PCI, severe exercise-induced myocardial ischaemia, exercise-induced malignant ventricular arrhythmia, skeletal vascular disease, smoking, and alteration of medication during the study. Patients were assessed for cardiovascular disease according to the Canadian Cardiovascular Society guidelines [17].

Six out of 50 patients were excluded to match the patient characteristics such as age and body mass index (BMI). Forty four patients were assigned to two experimental groups: the CRE (n = 27) and control (CON, n = 17) groups. Both the CRE and CON groups received individual counselling on the standard cardiac rehabilitation programme which included information on heart disease, risk factor modification, diet, stress management, and an exercise programme. The patients in the CRE group participated in the CRE programme. Patients who were referred by cardiologists to the cardiac rehabilitation clinic, but did not participate in the programme due to personal reasons such as the expense, distance, lack of need or awareness for the cardiac rehabilitation programme, or who were unwilling to change their sedentary lifestyle, were included in the CON group. Patients in the CON group participated in a survey during a six-week follow-up consultation. Seventeen patients who did not participate in physical activity during the six weeks were finally selected as the controls.

The amount of physical activity was detailed during an outpatient follow-up at the end of the programme in both groups. The state of inactivity during the study period in CON was confirmed by a medical specialist. All measurements were conducted at the baseline and immediately after six weeks of the intervention. The patients took 100 mg of aspirin and 75 mg of clopidogrel daily throughout the study duration. The medications taken by the patients are listed in Table 1.

Clinical examination

The clinical examinations were conducted before and immediately after the six week study period. The subjects were also instructed to refrain from exercise on the day before the examination. Blood pressure was measured in a seated position with a sphygmomanometer after 10 min of rest. The percentage of body fat was measured by bioelectrical impedance analysis (Inbody 3.0, Biospace, Korea). Waist-to-hip ratio (WHR) was calculated by dividing the tape measured waist circumference by the hip circumference. The waist was measured at the smallest circumference and the hip at the largest circumference in centimetres with a non-stretchable tape measure. Cardiopulmonary fitness was measured with a symptom-limited graded exercise test (GXT) using a modified Bruce protocol on a treadmill. Cardiopulmonary fitness measures VO_{2max}, exercise duration, and sub-rate pressure product (sRPP) were measured. Maximal oxygen uptake (VO_{2max}) was defined as the highest value or the plateau of directly measured oxygen consumption using a respiratory gas analyser (QMC, Quinton Instrument Co., Boston, MA, USA). Exercise duration was defined as the maximal exercising time during GXT. sRPP was calculated by multiplying the heart rate and systolic blood pressure values obtained at the second minute of the third stage modified Bruce protocol. The diagnostic ECG was continuously monitored during the test for possible clinical risk factors by an experienced physician.

Table 1. Clinical characteristics and medication of the subjects at baseline

Groups	CRE (n = 17)	CON (n = 15)	P
Age [years]	56.06 ± 7.31	54.33 ± 8.53	0.542
Height [cm]	168.47 ± 5.32	166.47 ± 4.34	0.256
Weight [kg]	75.14 ± 9.79	74.86 ± 10.36	0.939
Body fat [%]	27.95 ± 4.30	27.85 ± 4.66	0.950
WHR	0.94 ± 0.02	0.94 ± 0.03	0.927
BMI [kg/m^2]	26.41 ± 2.51	26.95 ± 2.98	0.576
Resting HR [mm Hg]	71.35 ± 11.97	73.80 ± 14.23	0.601
Resting SBP [mm Hg]	118.65 ± 18.47	106.07 ± 12.02§	0.032
Resting DBP [mm Hg]	80.18 ± 10.16	76.60 ± 7.47	0.271
Medications:			
ACEI	7 (41.2%)	8 (53.3%)	0.370
Clopidogrel	15 (88.2%)	14 (99.3%)	0.548
β-blockers	10 (58.8%)	9 (60.0%)	0.615
CCB	3 (17.6%)	2 (13.3%)	0.563
Diuretics	3 (17.6%)	2 (13.3%)	0.392
Nitrates	12 (70.6%)	10 (66.6%)	0.555
Aspirin	17 (100%)	15 (100%)	
ARB	4 (23.5%)	2 (13.3%)	0.392
Rosuvastatin	17 (100%)	15 (100%)	

Values are in mean ± SD; CRE — cardiac rehabilitation and exercise group; CON — control group; WHR — waist-to-hip ratio; BMI — body mass index; HR — heart rate; SBP — systolic blood pressure; DBP — diastolic blood pressure; ACEI — angiotensin converting enzyme inhibitor; CCB — calcium channel blocker; ARB — angiotensin II receptor blocker

Cardiac rehabilitation exercise programme

The subjects participated in a cardiac rehabilitation exercise programme within one week of their discharge. The target heart rate was calculated according to the Karvonen formula: [(maximal heart rate – resting heart rate × % exercise intensity) + resting heart rate]. The target heart rate was calculated at 60% of maximal heart rate during the first two weeks, 70% during the third and fourth weeks, and 85% during the fifth and sixth weeks. A wireless ECG monitoring system (Q-Tel ECG telemetry system, Quinton Instrument Co., Boston, MA, USA) was used to monitor possible abnormal heart rates and ECG (myocardial ischaemia or arrhythmia). The exercise programme consisted of 10 min of warm up composed of stretching, 30 min of cardiac rehabilitation exercise with treadmill and a stationary bicycle, and 10 min of cool down composed of 3 min of light walking and stretching. The cardiac rehabilitation exercise programme was composed of 15 min of treadmill exercise (MED-TRACK SR 60, Quinton Instrument Co) and 15 min of stationary cycling (Quinton CORIVAL 400, Quinton Instrument Co) with 3 min of light ground walking in between. Patients were monitored for any adverse event for an hour or less following each exercise session. Additional monitoring or proper treatment was conducted upon observation of a cardiac event [18].

Blood sampling

Blood was drawn from all the participants after 12 h of fasting during the first visits to the cardiac rehabilitation clinic as outpatients, and within one week of the discharge. Blood was drawn again six weeks after the initial intervention. Blood was collected from the antecubital vein according to the criteria presented by the guidelines of the Clinical and Laboratory Standards Institute. The collected blood samples were stored in a SST vacutainer tube (BD Vacutainer Serum Separator Tube, USA) to promote blood coagulation, centrifuged at 3,400 rpm for 10 min, separated for serum, and frozen at –70°C for later thawing for immediate analysis.

Blood analysis

The enzyme linked immunosorbent assay was used to measure TNF-α, IL-6, and adiponectin. Optical density was measured for TNF-α and IL-6 at 490 nm and adiponectin at 450 nm by a microplate ELISA reader, and then calculated for measurements by comparing with the standard curve using a R&D ELISA kit (R&D Systems Inc., Minneapolis, MN, USA). The minimum detectable doses were 0.038 pg/mL for TNF-α, 0.079 μg/mL for adiponectin, and 0.016 pg/mL for IL-6. The coefficient of variation of the intra-assay precision presented by the manufacturers was 5.3%, 3.5%, and 7.4%. All the samples were measured twice to produce mean values. Based on immunoturbidimetric assay, hs-CRP was measured by using HBI (HBI Co., Ltd., Korea) with the TBA-200FR NEO system (Toshiba, Japan). The precision during the relevant period of measurement in the laboratory was 10.51%.

Statistical analysis

Medications taken by the subjects were analysed by a χ^2 test. To determine the differences between the groups, a two-way analysis of variance test (ANOVA) was performed prior to the main effect test to confirm the presence of an interaction. When an interaction was found, the test for simple individual effects was performed with a Mann-Whitney U test. The difference between measurements before and after the cardiac rehabilitation exercise programme was tested by the Wilcoxon test. Pearson's correlation analysis was conducted to observe the correlating relationship between adiponectin and other factors. The statistical analyses were performed using the SPSS 11.0 statistical analysis program. The level of significance was $p < 0.05$.

RESULTS

The characteristics of the participants are presented in Table 1. CRE and CON were similar in all the variables such

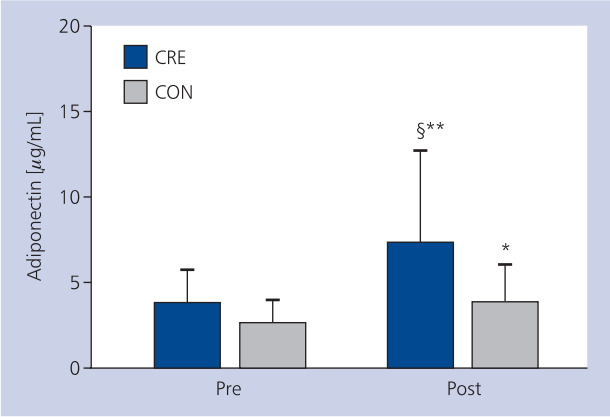

Figure 1. Changes in adiponectin [μg/mL] concentrations before (Pre) and after (Post) the cardiac rehabilitation and exercise programme; §significantly different in adiponectin concentration between cardiac rehabilitation and exercise (CRE) and control (CON) groups at $p < 0.05$; *significantly different from the baseline (Pre) at $p < 0.05$; **significantly different from the baseline (Pre) at $p < 0.01$

as age, height, weight, and BMI, except for resting blood pressure. A significant interaction was found in adiponectin between the groups. Adiponectin was significantly increased from 3.86 ± 1.95 μg/mL to 7.52 ± 5.28 μg/mL ($p < 0.001$) in CRE after six weeks of the exercise intervention. Adponectin was also significantly increased from 2.75 ± 1.25 μg/mL to 4.03 ± 2.13 μg/mL ($p = 0.009$) in CON. In addition, the increase in adiponectin was significantly greater in CRE than in CON ($p = 0.032$) (Fig. 1).

The inflammatory markers of the two groups were similar, as shown in Table 2. A significant difference was not observed in TNF-α between the groups before and after the intervention. IL-6 was significantly decreased in both CRE and CON after the intervention ($p = 0.038$, $p = 0.045$) without a difference between the two groups. As for hs-CRP, a significant reduction was shown in CRE after the intervention ($p = 0.042$) without a difference between the two groups.

Table 3 shows the changes in cardiopulmonary fitness and body composition. The variables of the exercise capacity and body composition before the intervention were similar between the two groups. After the intervention, VO_{2max} and exercise duration showed significant increases in CRE ($p < 0.001$, $p < 0.001$). VO_{2max} and exercise duration of CRE were significantly higher than in CON ($p < 0.001$, $p = 0.001$). As for sRPP, only CRE showed a significant decrease after the intervention ($p = 0.018$) without a difference between the two groups. As for body composition, BMI and body fat (%) did not show significant differences between the groups before and after the intervention. WHR showed a significant decrease in CRE after the intervention ($p = 0.002$) without a difference between the two groups. In order to examine the close relationship of adiponectin to abdominal body fat,

Table 2. Inflammation markers before (Pre) and after (Post) the cardiac rehabilitation and exercise programme

		Pre	Post
IL-6 [pg/mL]	CRE	7.13 ± 7.74	3.48 ± 2.93*
	CON	5.92 ± 4.76	3.16 ± 1.84*
TNF-α [pg/mL]	CRE	3.24 ± 1.60	3.29 ± 1.52
	CON	4.09 ± 2.20	4.22 ± 1.76
hs-CRP [mg/dL]	CRE	0.38 ± 0.40	0.21 ± 0.28*
	CON	0.38 ± 0.51	0.21 ± 0.27

Values are in mean ± SD; CRE — cardiac rehabilitation and exercise group; CON — control group; IL-6 — interleukin-6; TNF-α — tumour necrosis factor-α; hs-CRP — high sensitivity C-reactive protein; *significantly different from the baseline (Pre) at $p < 0.05$

Table 3. Cardiopulmonary capacity and body composition before (Pre) and after (Post) the cardiac rehabilitation and exercise programme

		Pre	Post
VO_2max [mL/kg/min]	CRE	27.30 ± 3.27	31.67 ± 4.30*§
	CON	27.55 ± 6.67	27.15 ± 6.46
sRPP	CRE	150.46 ± 30.03	135.18 ± 29.13*
	CON	137.71 ± 39.24	128.88 ± 26.56
Exercise duration [s]	CRE	850.41 ± 101.33	945.53 ± 75.6*§
	CON	853.87 ± 137.18	865.47 ± 122.59
BMI [kg/m^2]	CRE	26.41 ± 2.51	26.17 ± 2.16
	CON	26.95 ± 2.98	27.19 ± 3.35
Body fat [%]	CRE	27.95 ± 4.30	26.88 ± 4.38
	CON	27.85 ± 4.66	28.63 ± 4.88
WHR (ratio)	CRE	0.94 ± 0.02	0.93 ± 0.02*
	CON	0.94 ± 0.03	0.95 ± 0.03

Values are in mean ± SD, CRE — cardiac rehabilitation and exercise group; CON — control group; sRPP — sub-rate pressure product ×100; BMI — body mass index; WHR — waist-to-hip ratio; §significantly different between CRE and CON at $p < 0.05$; *significantly different from the baseline (Pre) at $p < 0.05$

Pearson's correlation was performed. A significant correlation between adiponectin and WHR was observed with the correlation coefficient of –0.376 ($p = 0.034$) (Fig. 2).

DISCUSSION

In this study, we investigated the effects of a six-week CRE programme on adiponectin, inflammatory markers, and cardiopulmonary fitness. CRE showed significant increases in adiponectin and cardiopulmonary fitness without significant reductions in inflammatory markers such as IL-6, TNF-α, and hs-CRP. In addition, the changes in adiponectin were inversely correlated with the changes in WHR.

Before the intervention, blood adiponectin was comparatively lower in both CRE and CON (≤ 4.0 μg/mL). Kumada et al. [19] reported that low adiponectin concentration is an

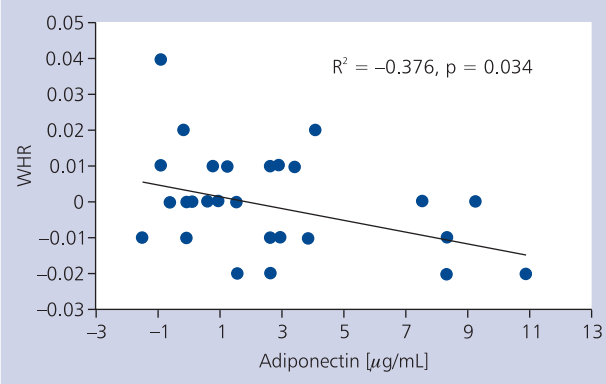

Figure 2. Correlation between waist-to-hip ratio (WHR) and adiponectin concentrations

independent risk factor for CAD and doubles the risk of CAD especially if it is less than 4.0 μg/mL. After the six-week exercise intervention, adiponectin was significantly increased in CRE (95%) and CON (47%). The changes were more significant in CRE than in CON (Fig. 1). Some researchers have reported that exercise increases adiponectin [20–22]. On the other hand, a study of obese female teenagers reported an insignificant change in blood adiponectin without a change in body weight after 12 weeks of aerobic exercise [23]. However, the association of body composition to the changes in adiponectin was not considered. Thus, in addition to body fat and WHR, body weight and BMI should be considered for investigation.

In our study, WHR was significantly decreased in CRE after the exercise intervention without a difference between the two groups. Furthermore, a significant correlation between the changes in adiponectin and abdominal fat was observed (Fig. 2). Arita et al. [24] reported that blood adiponectin was inversely correlated to BMI. Ryo et al. [5] also reported that blood adiponectin was inversely correlated to visceral fat. In this context, significantly increased adiponectin in CRE may be directly related to WRH reduction induced by increased VO_{2max} (Table 3).

A significant increase in adiponectin in CON may be related to the medication administered by the subjects. Rosuvastatin, a drug prescribed for hyperlipidaemia, was prescribed one week prior to the CRE programme. Statin reduces cardiac events in patients with cardiovascular diseases by stabilising altherom and retrogressing arteriosclerosis [25]. Atorvastatin and puvastatin reduce blood lipids and increase adiponectin in patients with ischaemic cardiovascular diseases [26]. Recently, Qu et al. [27] reported that rosuvastatin increased adiponectin concentration in patients with hypercholesterolaemia more significantly than atorvastatin. In this context, the increase in adiponectin in CON in this study may be due to rosuvastatin. However, a significantly greater increase in adiponectin was observed in CRE than in CON. This may be due to the combined effects of cardiac rehabilitation, exercise, and rosuvastatin.

Increased inflammatory cytokines such as hs-CRP, IL-6, and TNF-α have been observed to have positively correlated relationships with the risk factors of cardiovascular diseases [28]. In particular, hs-CRP, an independent risk factor for cardiovascular disease, is associated with accelerated mortality rates in patients with unstable angina and MI [29–31]. Previous studies have reported that exercise reduces hs-CRP in patients with CAD [32, 33].

In this study, IL-6, TNF-α, and hs-CRP failed to show significant differences between the groups. However, such results may be due to the duration of exercise training; in our previous study [16] the exercise intervention duration was 14 weeks while it was six weeks in this study. A longer duration of exercise training, as in our previous study, may induce more positive results in the inflammatory markers.

Cardiopulmonary fitness is the ability to supply and extract blood and oxygen to the active muscle. Major markers include VO_{2max}, exercise duration, and sRPP. VO_{2max} and exercise duration were significantly increased in CRE. Ades et al. [34] reported that CRE for three to six months increased VO_{2max} of patients by 11% to 36%. Stewart et al. [35] also reported improved fitness as well as VO_{2max} by exercise intervention. In this study, only six weeks of exercise intervention increased VO_{2max} by 16%.

Limitations of the study

There were limitations to our study. Although the patients followed the guidelines of the programme, the amount of physical activity and calorie consumption could not be closely controlled during the study.

CONCLUSIONS

Six weeks of CRE intervention was shown to increase adiponectin and cardiopulmonary fitness, although significant reductions in inflammatory markers were not observed. In addition, it was shown that adiponectin had an inverse correlation to WHR.

Such findings show that a short-term exercise intervention may have a significant effect on adiponectin, resulting from increased VO_{2max} induced WHR reduction. Improvement in adiponectin and further changes in cardiovascular fitness may require the combined effect of WHR reduction.

Conflict of interest: *none declared*

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Wpływ 6-tygodniowego programu rehabilitacji kardiologicznej i ćwiczeń na stężenie adiponektyny u pacjentów z ostrym zespołem wieńcowym

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Streszczenie

Wstęp: Zwiększone stężenie adiponektyny jest następstwem działania przeciwmiażdżycowego i wiąże się z prewencją miażdżycy. Jednak nie wiadomo, czy rehabilitacja kardiologiczna i ćwiczenia (CRE) powodują zwiększenie stężenia adiponektyny u osób po ostrym zespole wieńcowym (ACS).

Cel: Celem badania była ocena wpływu CRE na stężenie adiponektyny u pacjentów po ACS.

Metody: W programie rehabilitacji kardiologicznej brało udział 44 chorych poddanych przezskórnej interwencji wieńcowej. Uczestników przydzielano do grupy, w której stosowano badaną interwencję (CRE), lub do grupy kontrolnej (CON). Ocena przeprowadzona na początku badania i po 6 tygodniach obejmowała: stężenie adiponektyny, stężenie interleukiny-6 (IL-6), stężenie czynnika martwicy nowotworów alfa (TNF-α), stężenie białka C-reaktywnego określanego metodą wysokoczułą (hs-CRP), współczynnik talia–biodra (WHR) i czas trwania ćwiczeń.

Wyniki: Po 6 tygodniach stwierdzono istotne zwiększenie stężenia adiponektyny zarówno w grupie CRE, jak i w grupie CON (odpowiednio $p < 0{,}001$ i $p = 0{,}009$). W grupie CRE wzrost stężenia adiponektyny był istotnie większy niż w grupie CON ($p = 0{,}032$). Nie zanotowano znamiennych różnic między grupami w zakresie stężeń IL-6, TNF-α czy hs-CRP. Jednak w grupie CRE wartości VO_{2max} były istotnie większe, a czas ćwiczeń znamiennie dłuższy ($p < 0{,}001$). W grupie CRE stwierdzono również istotną redukcję WHR; współczynnik ten nie zmienił się natomiast w grupie CON ($p < 0{,}05$). Wykazano istnienie znamiennej odwrotnej zależności między różnicą stężeń adiponektyny a różnicą wartości WHR ($R^2 = -0{,}376$, $p = 0{,}034$).

Wnioski: Po 6 tygodniach stosowania badanej interwencji w grupie CRE stwierdzono istotny wzrost stężeń adiponektyny i poprawę wydolności krążeniowo-oddechowej. Mimo że nie zaobserwowano redukcji stężeń wskaźników zapalenia, w grupie CRE wykazano istotną zależność między zmianami stężenia adiponektyny a wartościami WHR. Wskazuje to, że krótki, 6-tygodniowy program CRE istotnie wpłynął na zmniejszenie stanu zapalnego.

Słowa kluczowe: choroba sercowo-naczyniowa, przezskórna interwencja wieńcowa, maksymalny pobór tlenu, wysiłek submaksymalny, produkt podwójny

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