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CHARACTERISTICS OF THE POST-INFARCTION PERIOD IN OBESE PATIENTS AFTER PERCUTANEOUS CORONARY INTERVENTION

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Annotation. *The study on the metabolic profile in the long-term period after myocardial infarction with comorbidity is relevant. The aim of the work was to examine metabolic profile and echocardiographic parameters in patients with ST-elevation myocardial infarction (STEMI) and obesity following percutaneous coronary intervention (PCI) after a 1-year follow-up. A total of 60 patients with STEMI and obesity were examined. The first subgroup consisted of 20 patients with medicamentous therapy, and the second group - 38 patients with PCI. Adropin, irisin, fatty acid-binding protein 4 (FABP4), C1q/tumor necrosis factor-related protein-3 (CTRP3) were measured by the enzyme-linked immunosorbent assay. The statistical processing of the study results obtained was carried out using the software package "IBM SPPS Statistics 27.0". The following parameters were increased in patients who received combined medicamentous and PCI therapy before and after the treatment (p<0.05): end-diastolic size (EDS) (by 16.83% and 10.89%, respectively), end-diastolic volume (EDV) (by 45.95% and 18.92% respectively), end-systolic volume (ESV) (by 40.0% and 27.69%, respectively), stroke volume (SV) (by 33.85% and 18.46%, respectively), left ventricular myocardial mass index (LVMMI) (by 18.93% and 10.06%, respectively), adropin (by 27.13% and 47.21%, respectively), irisin (by 2.07 times and 2.75 times, respectively) and CTRP3 (by 15.98 % and 31.96%, respectively), while the following parameters were decreased: systolic blood pressure (by 16.0% and 16.67%, respectively), diastolic blood pressure (by 15.56% and 14.44%, respectively), insulin (by 40 .38% and 48.59%, respectively), glucose (by 10.97% and 15.74%, respectively), atherogenic index (by 6.03% and 12.33%, respectively). Thus, patients with post-infarction cardiosclerosis and obesity have been revealed with increased echocardiographic parameters and imbalanced energy and adipokine metabolism.*

Keywords: *myocardial infarction, markers, obesity, percutaneous coronary intervention.*

Introduction

Obesity is a global health problem worldwide and a risk factor for cardiovascular disease (CVD). It is known that obesity plays an important role in the development of atherosclerosis and coronary heart disease (CHD), as well as it is involved in the structural and functional alterations of the heart, the progression of heart failure (HF) and responsible for the risk of atrial fibrillation and sudden cardiac death [2]. A study has demonstrated that stratification by a change in body mass index (BMI) after percutaneous coronary intervention (PCI) can help predict adverse events in patients with CHD [16]. Scientists deal with the issues of obesity paradox that indicates reduced mortality rates among high BMI patients following PCI [10]. PCI has the same effect on STEMI patients with or without obesity, and the low risk of side effects in obese patients can not be explained by a lower severity of myocardial infarction [6, 11]. Meanwhile, metabolic changes in obese patients with STEMI after PCI remain insufficiently studied to this day.

The purpose of this study was to examine the metabolic profile and echocardiographic parameters in obese STEMI patients following PCI after a 1-year follow-up.

Materials and methods

In total, 60 patients with STEMI and obesity were enrolled in the study that continued from September 1, 2018 to December 31, 2021. The first subgroup consisted of 20 patients who received standard drug therapy alone, and the second subgroup was composed of 38 patients after PCI. All the patients were diagnosed with STEMI, diagnosis and treatment were carried out according to the European recommendations of cardiologists [5].

The study design was approved by the Ethics Commission of Kharkiv National Medical University (Protocol No. 2 dated April 2, 2018). All the patients included in the study were notified and signed a voluntary informed consent to participate in the study.

The patients received medicamentous treatment including anticoagulants, acetylsalicylic acid, ticagrelor or clopidogrel, high-dose statin therapy, nitrates, beta-blockers (depending on heart rate), angiotensin-converting enzyme inhibitor (for blood pressure correction), and spironolactone or eplerenone (based on ejection fraction (EF)).

Myocardial revascularization was not performed owing to anatomical difficulties in performing coronary artery stenting, hospitalization of patients in the period of time window incompatible with reperfusion (more than 24 hours after the onset of myocardial infarction) or without pain syndrome manifestations upon admission and in patients who refused to implant a stent.

Serum levels of all biochemical indicators were measured prior to the treatment. Adropin, irisin, fatty acid binding protein 4 (FABP4) and C1q/TNF-related protein

(CTRP3) were detected by the enzyme-linked immunosorbent assay using commercially available reagents "Human adropin", "Human Fibronectin type III domain-containing protein 5", "Human FABP4" (Elabscience, Houston, USA) and "Human CTRP3" (Aviscera Bioscience Inc, Santa Clara, USA), respectively. Serum total cholesterol (TC) and high-density lipoprotein (HDL) cholesterol were quantified by peroxidase enzymatic method with "Cholesterol liquicolor" test (Human GmbH, Germany) and "HDL Cholesterol liquicolor" test (Human GmbH, Germany), respectively. Serum triglyceride (TG) levels were determined by enzymatic colorimetric method using a "Triglycerides liquicolor" reagent (Human GmbH, Germany). Atherogenic index (AI) was calculated in accordance with the standard formula proposed by A. M. Klimov. The levels of very low-density lipoprotein (VLDL) and low-density lipoprotein (LDL) were estimated based upon the Friedewald equation by. Fasting blood glucose levels were tested by glucoseoxidase method with a commercial test system "Human Glucose" (LLC NPP "Filisit-Diagnostics", Ukraine).

Doppler echocardiographic examination was performed according to the conventional technique on a Radmir ULTIMA Pro30 ultrasound scanner. End-diastolic size (EDS), end-systolic size (ESS), end-diastolic volume (EDV), end-systolic volume (ESV), left ventricle ejection fraction (LV EF), stroke volume (SV), interventricular septal thickness (IVST), aorta diameter, left atrial size (LA), and posterior wall thickness of the left ventricle (LV PWT) in diastole were evaluated. LV myocardial mass (LVMM) and LVMM index (LVMMI = LVMM / body surface area (m2)) were calculated. LV hypertrophy (LVH) was considered at an LVMMI value of more than 110 g/m2 for women and more than 125 g/m2 for men. The study also entailed calculation of the LV relative wall thickness (RWT) (LV RWT = (LV PWT + IVST) / LV EDS)) as well as determination of the LV remodeling type. LV RWT?0.45 and normal LVMMI was classified as concentric LV remodeling. Obesity was diagnosed by BMI following the established formula: weight (kg)/height (m^2).

Statistical processing of the data obtained was carried out using the computer program IBM SPPS version 27.0 (2020) (IBM Inc., USA, license No. L-CZAA-BKKMKE). The analysis of the examined parameters regarding the normality of the distribution was carried out according to the Shapiro-Wilk test. Quantitative variables were used in the statistical analysis. Quantitative data were presented as percentage, median, and interquartile range (25th and 75th percentiles). The non-parametric Mann-Whitney rank test was used to compare quantitative indicators between two independent groups, and the Wilcoxon T test was used for two dependent groups. The limit value of significance for testing statistical hypotheses in the study was set at a level of $p<0.05$.

This paper is a part of the scientific-research works "Ischemic heart disease in polymorbidity: pathogenetic aspects of development, course, diagnostic and improvement of treatment" No. 0118U000929, valid term 2017 - 2019 and "Prediction of the course, improvement of diagnosis and treatment of ischemic heart disease and arterial hypertension in patients with metabolic disorders" № 0120U102025, valid term 2020 - 2022.

Results

Table 1 shows the dynamics of anthropometric indicators and structural and functional parameters of the LV in patients with obesity after myocardial infarction before treatment and 1 year after myocardial revascularization. The following indicators demonstrated significant differences before treatment and 1 year after standard medicamentous therapy and PCI: systolic blood pressure (SBP) and diastolic blood pressure (DBP), EDS, EDV, ESV, SV, LVMMI ($p<0.05$). After combined medicamentous and

Table 1. Dynamic evaluation of the echocardiographic and anthropometric indicators in patients with obesity and myocardial infarction after the 1-year follow-up.

Indicator, unit of measurement	Before treatment, (n=60)	After treatment		Significance (p)
		Patients without PCI, (n=20)	Patients with PCI, (n=38)	
	1	2	3	
SBP, mmHg	150.0 (137.0; 163.75)	126.0 (120.0; 130.0)	125.0 (120.0; 128.0)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
DBP, mmHg	90.0 (80.0; 100.0)	76.0 (67.0; 80.0)	77.0 (72.0; 80.0)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
HR, bpm	75.0 (66.5; 85.75)	68.0 (67.0; 72.0)	65.0 (64.0; 70.0)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
Pulse, bpm	72.0 (64.5; 83.65)	66.0 (62.0; 68.0)	64.0 (62.0; 68.0)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
Weight, kg	92.0 (88.25; 100.01)	101.0 (86.25; 105.35)	95.5 (91.0; 97.0)	$p_{1-2}<0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$

Continuation of table 3.

Indicator, unit of measurement	Before treatment, (n=60)	After treatment		Significance (p)
		Patients without PCI, (n=20)	Patients with PCI, (n=38)	
	1	2	3	
BMI, kg/m^2	32.10 (30.85; 33.55)	35.2 (31.25; 37.35)	33.1 (31.8; 36.32)	$p_{1-2}<0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
Waist circumference, cm	118.0 (108.25; 130.75)	121.0 (111.0; 132.0)	120.1 (109.45; 132.15)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
Hip circumference, cm	109.0 (102.0; 115.0)	112.0 (105.0; 118.0)	110.0 (103.0; 118.0)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
EDS, cm	5.05 (4.73; 5.5)	5.90 (5.50; 6.00)	5.60 (5.40; 5.80)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
ESS, cm	3.80 (3.40; 4.30)	4.30 (3.95; 4.63)	4.10 (3.70; 4.40)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
EDV, mL	129.50 (108.75; 154.0)	189.0 (154.0; 213.0)	154.0(141.0; 173.0)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}<0.05$
ESV, mL	65.0 (50.0; 82.5)	91.0 (70.0; 102.5)	83.0 (74.0; 91.0)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
SV, mL	65.0 (58.25; 80.0)	87.0 (74.0; 94.0)	77.0 (63.0; 86.0)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}<0.05$
EF, %	52.0 (45.0; 55.0)	49.0 (46.0; 54.0)	55.0 (53.0; 57.0)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
IVST, cm	1.2 (1.0; 1.3)	1.3 (1.2; 1.4)	1.25 (1.1; 1.2)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
LV PWT, cm	1.25 (1.1; 1.3)	1.28 (1.2; 1.4)	1.22 (1.1; 1.3)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
LA, cm	3.7(3.3; 4.18)	4.2 (3.85; 4.65)	3.8 (3.4; 4.1)	$p_{1-2}<0.05$ $p_{1-3}>0.05$ $p_{2-3}<0.05$
Aorta diameter, cm	3.2 (3.0; 3.48)	3.4 (3.25; 3.65)	3.3 (3.1; 3.5)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
LVMM, g	306.25 (235.33; 352.5)	323.50 (274.25; 304.20)	317.90 (267.70; 366.50)	$p_{1-2}<0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
LVMMI, g/m^2	138.65 (110.03; 170.90)	164.90 (117.1; 181.5)	152.60 (145.0; 174.0)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
LV RWT	0.48 (0.43; 0.52)	0.47 (0.43; 0.49)	0.45 (0.43; 0.51)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$

Note: LV RWT - left ventricle relative wall thickness, SBP - systolic blood pressure, DBP - diastolic blood pressure, LVMMI - left ventricular myocardial mass index, BMI - body mass index, ESV - end-systolic volume, ESS - end-systolic size, EDV - end-diastolic volume, EDS - end-diastolic size, LA - left atrium, LVMM - left ventricular myocardial mass, LV PWT - left ventricular posterior wall thickness, IVST - interventricular septal thickness, SV - stroke volume, EF - ejection fraction, HR - heart rate.

Table 2. Metabolic profile of patients with obesity and myocardial infarction after the 1-year follow-up.

Indicator, unit of measurement	Before treatment, (n=60)	After treatment		Significance (p)
		Patients without PCI, (n=20)	Patients with PCI, (n=38)	
	1	2	3	
Glucose, mmol/L	6.29 (5.08; 7.22)	5.60 (5.20; 5.85)	5.3 (5.2; 5.6)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}<0.05$
Insulin, mcU/mL	26.57 (21.77; 38.09)	15.84 (13.39; 18.79)	13.66 (11.76; 15.42)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}<0.05$
Adropin, pg/mL	15.04 (8.77; 18.06)	19.12 (16.09; 21.59)	22.14 (20.65; 22.69)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}<0.05$
Irisin, ng/mL	1.62 (1.28; 2.12)	3.35 (2.89; 4.37)	4.46 (3.78; 4.97)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}<0.05$
FABP4, ng/mL	10.96 (9.25; 12.65)	8.35 (6.69; 9.57)	6.90 (6.38; 9.82)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
CTRP3, ng/mL	235.50 (204.47; 268.63)	273.13 (225.94; 291.79)	310.76 (290.60; 319.51)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}<0.05$
TC, mmol/L	5.32 (4.28; 6.04)	5.40 (5.10; 6.15)	5.22 (5.01; 5.89)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
VLDL, mmol/L	0.90 (0.66; 1.25)	1.20 (0.41; 1.33)	1.09 (0.40; 1.24)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
LDL, mmol/L	3.14 (2.22; 4.06)	3.36 (2.98; 3.91)	3.22 (2.88; 3.81)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
TG, mmol/L	1.91 (1.40; 2.73)	1.92 (1.21; 2.06)	1.81 (1.34; 1.98)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
HDL, mmol/L	1.15 (0.96; 1.32)	1.11 (1.10; 1.32)	1.15 (1.10; 1.23)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
AI	3.65 (2.54; 4.90)	3.43 (2.63; 3.87)	3.20 (2.23; 3.88)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$

Note: TC - total cholesterol, VLDL - very low-density lipoprotein, LDL - low-density lipoprotein, TG - triglyceride, HDL - high-density lipoprotein, AI - atherogenic index, FABP4 - fatty acid binding protein 4, CTRP3 - C1q/TNF-related protein - 3.

PCI treatment, obese patients with post-infarction cardiosclerosis were revealed with significantly lower values of indicators such as SBP (by 16.0% and 16.67%, respectively), DBP (by 15.56% and 14.44%, respectively) and significantly higher values of such indicators as EDS (by 16.83% and 10.89%, respectively), EDV (by 45.95% and 18.92%, respectively), ESV (by 40.0% and 27.69%, respectively), SV (by 33.85% and 18.46%, respectively), LVMMI (by 18.93% and 10.06%, respectively), as compared to the values of those indicators before treatment, respectively, ($p<0.05$). After the treatment with medicamentous therapy alone, the patients with post-infarction cardiosclerosis and obesity showed significantly higher values of such indicators as weight by 9.78%, BMI by 9.65%, LA by 13.51%, LVMM by 5.63%, while after PCI alone, significantly lower values of heart rate (HR) by 13.33%, pulse rate by 11.11% were defined as compared to the values of those indicators before treatment, respectively, ($p<0.05$).

Table 2 shows the dynamics of indicators related to carbohydrate, energy, lipid, and adipokine metabolism in obese patients after myocardial infarction before treatment and a year after myocardial revascularization. Significant differences were found regarding such indicators as glucose, insulin, AI, adropin, irisin, FABP4, CTRP3, ($p<0.05$) before treatment and after the one-year standard medicamentous therapy and PCI. After treatment, the patients with post-infarction cardiosclerosis and obesity had

significantly lower values of such indicators as insulin (by 40.38% and 48.59%, respectively), glucose (by 10.97% and 15.74%, respectively), AI (by 6.03% and 12.33%, respectively) and higher values of adropin (by 27.13% and 47.21%, respectively), irisin (by 2.07 times and 2.75 times, respectively) and CTRP3 (by 15.98% and 31.96%, respectively) in comparison with the values of those indicators before treatment, respectively ($p<0.05$). The patients with post-infarction cardiosclerosis and obesity demonstrated significantly 5.24% lower TG levels after the treatment with PCI alone as compared to those before the treatment, ($p<0.05$). No differences in those indicators were found in patients who received medicamentous therapy alone, ($p>0.05$).

Comparing the studied indicators between the subgroups after medicamentous treatment and PCI, a significant decrease in EDV by 18.52%, SV by 11.49%, LA by 9.53%, glucose levels by 5.36%, insulin levels by 13.76% was detected along with an increase in the serum levels of adropin by 15.79%, irisin by 33.13%, and CTRP3 by 13.78%, respectively ($p<0.05$).

Discussion

The study on the clinical course characteristics in patients after myocardial infarction who underwent an invasive intervention should be detailed, because the restoration of coronary blood circulation does not exclude the further progression of atherosclerotic lesions and the recurrence of major cardiovascular events. Adropin is principally involved in cardiac energy metabolism and may be a presumed contestant for the treatment of heart diseases associated with insulin resistance [1]. Serum adropin levels were significantly lower in overweight/obese individuals as compared to those in normal-weight ones, suggesting a probable role of this hormone in the pathogenesis of obesity [14]. Serum adropin levels were decreased to a greater extent in patients with STEMI than those in patients without CHD. In addition, the levels of serum adropin were decreased with worsening severity of coronary artery damage, indicating the severity of CHD [9].

Irisin regulates mitochondrial energy, glucose metabolism and fatty acid oxidation. Cardiomyocytes produce irisin that influences various functions of the cardiovascular system. At different stages of heart failure, the impact of irisin varies widely on mitochondrial dysfunction, oxidative stress, metabolic imbalance, energy expenditure, and the prognosis of heart failure [4]. Patients with CHD and a high degree of coronary artery lesion severity had lower concentrations of serum irisin as compared to patients with less severe lesions of coronary arteries [3]. Serum FABP4 concentrations have been shown to be associated with prognosis for stable angina patients following PCI, suggesting that serum FABP4 levels may be useful indicators for secondary prevention assessment [15]. According to M. Obokata et al. (2018), FABP4 attained a maximum level at hospital admission or immediately after PCI in patients with AMI [8].

Kyung Mook Choi et al. (2014) have reported a correlation between lower serum CTRP3 levels and higher both weight and waist circumference [7]. According to M. Sawicka et al. (2016) [12], serum levels of CTRP3 were decreased in patients with AMI. Authors M. Shanaki et al. (2020) have noted a post-infarction cardiac fibrosis attenuation and myofibroblast differentiation inhibition through AMP-activated protein kinase and Akt signaling pathways mediated by CTRP3 [13].

We have found increased values of EDS, EDV, ESV, SV, adropin, irisin, CTRP3 and decreased values of SBP and DBP, parameters of carbohydrate metabolism, adipokine FABP4, AI in patients who received both medicamentous therapy and PCI. Following PCI, the patients had a slow-moving tendency to echocardiographic changes, a significant decrease in HR and TG levels as compared to those before treatment. In the study process, changes in energy and adipokine metabolism have been determined.

Conclusions and prospects for further development

1. An imbalance of energy and adipokine metabolism indicators has been revealed as evidenced by the low serum levels of adropin, irisin, CTRP3 and increased concentrations of FABP4 in obese patients after myocardial infarction.

2. Slowing structural and functional changes in the LV myocardium have been found in patients following PCI compared to patients receiving medicamentous therapy alone.

3. Higher values of energy metabolism markers have been demonstrated by patients after PCI. The profile of adipokine homeostasis markers has been also found to be improved, namely the increased serum levels of CTRP3 and decreased serum levels of FABP4.

This study had some limitations. First, the sample size was relatively small (n = 58), therefore it should be larger in the future to confirm the conclusions. Second, since only patients with STEMI and obesity were included in the study, it would be interesting to examine patients with non-ST-segment elevation myocardial infarction and obesity following PCI.

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ОСОБЛИВОСТІ ПЕРЕБІГУ ПІСЛЯІНФАРКТНОГО ПЕРІОДУ У ПАЦІЄНТІВ ІЗ ОЖИРІННЯМ, ЯКІ ПЕРЕНЕСЛИ ПЕРКУТАННЕ КОРОНАРНЕ ВТРУЧАННЯ

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Анотація. *Актуальним є дослідження впливу метаболічного профілю на перебіг віддаленого періоду інфаркту міокарда із коморбідністю. Метою роботи було дослідити метаболічний профіль та ехокардіографічні параметри у хворих із гострим інфарктом міокарда із ST елевацією (STEMI) та ожирінням після перкутанного коронарного втручання (ПКВ) через 1 рік спостереження. Обстежено 60 пацієнтів із STEMI та ожирінням. Перша група склала 20 хворих із медикаментозною терапією, а друга група - 38 пацієнтів із ПКВ. Адропін, ірисин, білок, що зв'язує жирні кислоти 4 (FABP 4), C1q/фактор некрозу пухлини асоційований білок 3 (CTRP 3) визначали імуноферментним методом. Статистичне опрацювання отриманих результатів дослідження проведено за допомогою програмного пакета "IBM SPPS Statistics 27,0". У пацієнтів за умов медикаментозної терапії та ПКВ були збільшені наступні параметри: кінцево-діастолічний розмір (на 16,83% та 10,89% відповідно), кінцево-діастолічний об'єм (КДО) (на 45,95% та 18,92% відповідно), кінцево-систолічний об'єм (на 40,0% та 27,69% відповідно), ударний об'єм (УО) (на 33,85% та 18,46% відповідно), індекс маси міокарда лівого шлуночка (на 18,93% та 10,06% відповідно), адропін (на 27,13% та 47,21% відповідно), ірисин (у 2,07 рази та 2,75 рази відповідно) та CTRP 3 (на 15,98% та 31,96% відповідно) та зменшилися показники: систолічний артеріальний тиск (на 16,0% та 16,67% відповідно), діастолічний артеріальний тиск (на 15,56% та 14,44% відповідно), інсулін (на 40,38% та 48,59% відповідно), глюкоза (на 10,97% та 15,74% відповідно), індекс атерогенності (на 6,03% та 12,33% відповідно) у порівнянні із хворими до лікування відповідно ($p<0,05$). Отже, у пацієнтів із постінфарктним кардіосклерозом та ожирінням спостерігається зростання ехокардіографічних параметрів, дисбаланс в енергетичному й адипокіновому обміні.*

Ключові слова: *інфаркт міокарда, маркери, ожиріння, перкутанне коронарне втручання.*