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PECULIARITIES OF THE POST-INFARCTION PERIOD CLINICAL COURSE IN PATIENTS WITH DIABETES MELLITUS TYPE II AFTER PERCUTANEOUS CORONARY INTERVENTION

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Aim *– to define the parameters' dynamics of carbohydrate, lipid, energy and adipokine metabolism and the structural and functional state of the left ventricle (LV) myocardium in patients with post-infarction cardiosclerosis and type 2 diabetes mellitus (DM).*

Materials and methods. *74 type 2 DM patients, who underwent myocardial infarction, were examined. The first subgroup comprised 20 patients, who received medicinal therapy, and the second subgroup was composed of 50 patients who underwent percutaneous coronary intervention (PCI). Serum levels of insulin, adropin, irisin, fatty acid binding protein 4 (FABP4) and C1q/tumor necrosis factor-related protein (CTRP3) were determined by enzyme-linked immunosorbent assay. Doppler echocardiographic examination was performed using a Radmir ULTIMA Pro30 ultrasound scanner. The results were statistically analyzed using IBM SPPS software (version 27.0). A level of p<0.05 was set for determining statistical significance.*

Results. *Following parameters: end-diastolic volume (17,19% and 7,42% respectively), end-systolic volume (30,71% and 10,24% respectively), stroke volume (32,58% and 10,61% respectively), iricin (37,57% and 79,89% respectively) increased and such indices as insulin (46,70% and 58,03% respectively), glucose (35,24 and 39,81% respectively) decreased in patients with post-infarction cardiosclerosis and type 2 DM under conditions of medicinal therapy in comparison with values of these indices in patients before the treatment respectively (p<0,05). Following the treatment only with medicinal therapy, in patients with post-infarction cardiosclerosis and type 2 DM higher values of such indicators as EDS 4.90%, LV PWT 16.67%, LA 18.42%, LVMM 7.77%, LVMMI 19.24%, have been determined, reliably lower value of diastolic arterial pressure – 16,19%, lipoproteins of low density – 5,90%, triglycerides – 13,30%, atherogenic coefficient – 16,09%, adropin – 41.43%, FABP4-27.71%, CTRP3-32.91% were established under conditions of only PCI treatment in comparison with the values of those biochemical parameters before the treatment, respectively, (p<0.05).*

Conclusions. *1. An increase of echocardiographic indicators, imbalance in energetic and adipokine metabolism have been found in patients with post-infarction cardiosclerosis and type 2 DM. 2. In patients under conditions of PCI structural-functional changes of LV myocardium delay in comparison with those receiving only medicinal therapy.*

Key words: *echocardiography, diabetes, myocardial infarction, markers, course.*

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ОСОБЛИВОСТІ ПЕРЕБІГУ ПІСЛЯІНФАРКТНОГО ПЕРІОДУ У ПАЦІЄНТІВ ІЗ ЦУКРОВИМ ДІАБЕТОМ 2 ТИПУ, ЯКІ ПЕРЕНЕСЛИ ПЕРКУТАННЕ КОРОНАРНЕ ВТРУЧАННЯ

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Мета *– визначити динаміку параметрів вуглеводного, ліпідного, енергетичного, адипокінового обміну та структурно-функціонального стану міокарда лівого шлуночка (ЛШ) у пацієнтів із постінфарктним кардіосклерозом та цукровим діабетом (ЦД) 2 типу.*

Матеріали і методи. *Обстежено 74 пацієнтів, хворих на ЦД 2 типу, які перенесли інфаркт міокарда. Першу підгрупу сформували з 20 пацієнтів, які отримували медикаментозну терапію та другу підгрупу – з 50 пацієнтів, яким виконано перкутанне коронарне втручання (ПКВ). Вміст адропіну, ірисину, білка, що зв'язує жирні кислоти 4 (FABP 4) і C1q/фактор некрозу пухлини асоційованого білка (CTRP 3) визначено імуноферментним методом. На ультразвуковому сканері Radmir ULTIMA Pro30 проведено доплер-ехокардіографічне дослідження. Статистичну обробку результатів дослідження здійснено за допомогою програми «IBM SPPS Statistics 27,0». При значенні p<0,05 різниця вважалася достовірною.*

Результати. *У пацієнтів із постінфарктним кардіосклерозом та ЦД 2 типу за умов медикаментозної терапії та ПКВ збільшилися наступні параметри: кінцево-діастолічний об'єм (на 17,19% та 7,42% відповідно), кінцево-систолічний об'єм (на 30,71% та 10,24% відповідно), ударний об'єм (на 32,58% та 10,61% відповідно),*

Ключові слова: *ехокардіографія, діабет, інфаркт міокарда, маркери, перебіг.*

Клінічна та експериментальна патологія 2022. Т.21, №3 (81). С. 39-45.

ірисин (на 37,57% та 79,89% відповідно) та зменшилися такі показники: інсулін (на 46,70% та 58,03% відповідно), глюкоза (на 35,24% та 39,81% відповідно) порівняно зі значенням цих показників у хворих до лікування відповідно (p<0,05). Після лікування у хворих із постінфарктним кардіосклерозом і ЦД 2 типу за умов проведення лише медикаментозної терапії встановлено вірогідно більш високі значення таких показників, як кінцево-діастолічний розмір на 4,90%, товщина задньої стінки ЛШ на 16,67%, лівого передсердя на 18,42%, маса міокарда ЛШ на 7,77%, індекс маси міокарда ЛШ на 19,24%, тоді як за умов проведення лише ПКВ визначено вірогідно більш низьке значення діастолічного артеріального тиску на 16,19%, ліпопротеїдів низької щільності – на 5,90%, тригліцеридів –на 13,30%, коефіцієнту атерогенності – на 16,09%, адропіну – на 41,43%, FABP 4 –на 27,71%, CTRP 3 – на 32,91% порівняно зі значенням цих показників до лікування відповідно (p<0,05).

Висновки. *1. У пацієнтів із постінфарктним кардіосклерозом та ЦД 2 типу відбувається зростання ехокардіографічних показників, дисбаланс в енергетичному та адипокіновому обміні. 2. За умов ПКВ у пацієнтів уповільнюються структурно-функціональні зміни міокарда ЛШ порівняно із хворими, котрі отримують лише медикаментозну терапію.*

Introduction

Treatment of acute myocardial infarction (AMI) in diabetic patients does not differ from that in non-diabetic individuals. Mortality rates among diabetic patients are increasing, reperfusion measures such as direct percutaneous coronary intervention (PCI) should therefore be performed rapidly. The authors believe that the development of an integrated strategy for the coronary heart disease (CHD) treatment should be finalized after stabilizing the condition of a patient and further differentiated metabolic monitoring needs to be carried out surely in patients after AMI [7]. Restoration of coronary blood circulation does not exclude the further progression of atherosclerotic lesions and the complicated course of myocardial infarction; it is therefore necessary to study the characteristics of the disease clinical course after invasive interventions.

Adropin is associated with CHD through lipid metabolism regulation, insulin resistance improvement, protection of vascular endothelial cell function and anti-inflammatory effects. Serum adropin levels were significantly lower in patients with CHD, suggesting that adropin levels may be related to the pathogenesis of CHD [15].

Scientists have determined that irisin is a new approach by means of which cardioprotection can be provided via improving the mitochondrial function by inhibiting the mitochondrial permeability transition pore opening and suppressing mitochondrial swelling. Irisin treatment has been found to be significantly mitigate hypoxic or post-reoxygenation injury of cardiomyoblasts as evidenced by diminishing lactate dehydrogenase leakage and the number of apoptotic cardiomyocytes [11].

In the modern lifestyle with excessive caloric intake and reduced energy expenditure, the presence and induction of fatty acid binding protein 4 (FABP4) or increased secretion of conformationally altered FABP4, which can bind palmitic acid with relatively high affinity, might have a quite adverse effect on regulation of inflammatory processes or metabolic homeostasis. In such conditions, FABP4 inhibition, neutralization or elimination of secreted FABP4, as well as the use of possible unidentified FABP4 receptor antagonists may be an effective therapeutic strategy for metabolic disorders and cardiovascular diseases (CVD) [3].

C1q/tumor necrosis factor-related protein 3 (CTRP3) is a pro-inflammatory adipokine responsible for the regulation of glucose and lipid metabolism, involved in pathogenesis of systemic inflammation in obesity, insulin resistance, modulation of mitochondrial biogenesis, and it serves as an independent predictor of atherosclerosis [12]. Low CTRP3 plasma concentrations have been found in patients with CHD which was considered as a predictor of coronary artery disease [2].

The aim of the study

To define the dynamics of parameters related to carbohydrate, lipid, energy and adipokine metabolism and the structural and functional state of the left ventricle (LV) myocardium in patients with post-infarction cardiosclerosis and type 2 diabetes mellitus (DM).

Materials and methods of the research

A total of 74 patients with ST-segment elevation AMI (STEMI) and type 2 DM were enrolled in the study. Examinations lasted from 01 September 2018 to 31 December 2021. Clinical, instrumental and laboratory findings served as the basis for diagnosing STEMI and type 2 DM according to the criteria proposed by the European Society of Cardiology, American Diabetes Association and the European Association for the Study of Diabetes joint consensus. The patients were divided into 2 subgroups. The first subgroup comprised 20 patients who received medicinal therapy alone and did not undergo myocardial revascularization, while the second subgroup was composed of 50 patients who underwent PCI. The medicinal therapy consisted of anticoagulants, acetylsalicylic acid, ticagrelor or clopidogrel, high-dose statin treatment, nitrates, beta-blockers (depending on heart rate (HR)), angiotensin-converting enzyme inhibitor (for blood pressure correction), and spironolactone or eplerenone (in accordance with ejection fraction (EF)).

Patients diagnosed with STEMI and type 2 DM were included in the study.

Exclusion criteria were type 1 DM, non-ST-segment elevation myocardial infarction (NSTEMI), COVID-19, autoimmune diseases, pituitary and hypothalamic diseases, thyroid disease, symptomatic hypertension, valvular heart disease, IV FC chronic heart failure to

myocardial infarction, chronic obstructive pulmonary disease, impaired liver and kidney functions, severe anemia, malignancy.

The Ethics Committee of Kharkiv National Medical University approved the study design (Protocol No. 2 dated 02 April 2018). All the participating patients agreed and voluntary signed informed consent prior to the study.

Blood samples were taken, and echocardiographic examinations were performed before the treatment and after one-year follow-up of patients. All studies were carried out at the Biochemical Department of the Central Research Laboratory of Kharkiv National Medical University of the Ministry of Health of Ukraine. Serum levels of insulin, adropin, irisin, FABP4 and CTRP3 were detected by enzyme-linked immunosorbent assay using corresponding kits: Human Insulin reagent (Monobind Inc, USA), Human adropin, irisisn, FABP4 reagents (Elabscience Biotechnology, USA) and Human CTRP3 (Aviscera Bioscience Inc, USA) in accordance with the manufacturer's instructions. Serum total cholesterol (TC) and high-density lipoprotein (HDL) cholesterol were analyzed by peroxidase enzymatic method with commercial kits «Cholesterol liquicolor» (Human GmbH, Germany) and «HDL Cholesterol liquicolor» (Human GmbH, Germany), respectively. Triglyceride (TG) levels were quantified by enzymatic colorimetric method using a commercial kit «Triglycerides liquicolor» (Human GmbH, Germany). The standard A. M. Klimov formula was used to assess atherogenic index (AI). The levels of very low-density lipoprotein (VLDL) and low-density lipoprotein (LDL) were calculated by the Friedewald formula. Fasting blood glucose levels were measured by glucoseoxidase method with the help of commercial test system «Human Glucose» (LLC NPP «Filisit-Diagnostics», Ukraine).

Doppler echocardiographic examination was performed according to the conventional technique using 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 analyzed. LV myocardial mass (LVMM) and LVMM index (LVMMI = LVMM / body surface area (m2)) were estimated. LV hypertrophy (LVH) was accepted at an LVMMI value of more than 110 g/m2 for females and more than 125 g/m2 for males. The study also covered an analysis of the LV relative wall thickness (RWT) (LV RWT = (LV PWT + IVST) / LV EDS)) as well as categorization of the LV remodeling type. LV RWT ≥ 0.45 and normal LVMMI was classified as concentric remodeling of the LV. Obesity was diagnosed based on body mass index (BMI) following the generally accepted formula: weight (kg)/ height (m2).

The data obtained were statistically analyzed using IBM SPPS software (version 27.0, 2020, IBM Inc., USA, license No. L-CZAA-BKKMKE). For the studied parameters, the normality of the data distribution was assessed using the Shapiro–Wilk test. The continuous variables were expressed as percentages, medians and interquartile ranges (25 and 75 percentiles). The Mann-Whitney ranking criterion was used to compare quantitative indicators between two independent groups as the data were not normally distributed. For the two-group comparisons, the mean values of indicators were compared using the Wilcoxon T test. A significance level of $p<0.05$ was set for testing statistical hypotheses in the study.

Results and their discussion

Table 1 shows the dynamics of anthropometric indicators as well as structural and functional parameters of the LV in diabetic patients after myocardial infarction before the treatment and 1 year after myocardial revascularization. No significant differences were found before treatment and 1 year after the standard medicinal therapy and PCI in relation to the indicators such as body weight, waist circumference, hip circumference, BMI, ESS, EF, IVST, aorta diameter, LV RWT ($p>0,05$). In comparison with the values of some indicators after and before the medicinal treatment and PCI, patients with post-infarction cardiosclerosis and type 2 DM demonstrated significantly lower values of such indicators as systolic blood pressure (SBP) (14.71% and 21.18%, respectively), HR (15.69% and 21.57%, respectively), pulse (15.22% and 21.74%, respectively) and significantly higher values of such indicators as EDV (17.19% and 7.42%, respectively), ESV (30.71% and 10.24%, respectively), SV (32.58% and 10.61%, respectively), ($p<0, 05$). After the treatment with medicinal therapy alone, the patients with post-infarction cardiosclerosis and type 2 DM were revealed with significantly higher values of such indicators as EDS 4.90%, LV PWT 16.67%, LA 18.42%, LVMM 7 .77%, LVMMI 19.24%, while after the treatment with PCI alone, 16.19% significantly lower values of diastolic blood pressure (DBP) were detected in comparison with the values of those indicators in the patients before the treatment ($p<0.05$).

Table 2 demonstrates the dynamics of carbohydrate, energy, lipid, and adipokine metabolism indicators in diabetic patients after myocardial infarction before the treatment and 1 year after myocardial revascularization. There were no significant differences before and 1 year after the standard medicinal treatment and PCI regarding such indicators as TC, VLDL and HDL cholesterol ($p>0.05$). However, when comparing some indicators after and before the combined treatment, patients with post-infarction cardiosclerosis and type 2 DM presented significantly lower values of such parameters as insulin (46.70% and 58.03%, respectively), glucose (35.24% and 39.81%, respectively) and higher values of irisin (37.57% and 79.89%, respectively), ($p<0.05$). After the treatment with PCI alone, type 2 diabetic patients with post-infarction cardiosclerosis were found to have significantly lower values of such indicators as LDL cholesterol 5.90%, TG 13.30%, AI 16.09%, adropin 41.43%, FABP4 27.71%, CTRP3 by 32.91% in comparison with the values of those biochemical parameters before the treatment, respectively, ($p<0.05$), while no differences between those indicators were found in patients who received medicinal therapy alone, ($p>0.05$).

Table 1

Dynamic evaluation of the echocardiographic and anthropometric indicators in diabetic patients after myocardial infarction in the 1-year follow-up

Indicator, unit of measurement	Before treatment, (n=74)	After treatment		Significance (p)
		Patients without PCI, (n=20)	Patients with PCI, (n=50)	
	1	2	3	
SBP, mmHg	170.0 (140.0; 180.0)	145.0 (125.0; 156.5)	134.0 (120.0; 145.0)	p_{1-2}=0.031 p_{1-3}=0.003 p_{2-3}=0.111
DBP, mmHg	105.0 (85.0; 110.0)	94.0 (80.5; 102.1)	88.0 (74.0; 94.0)	p_{1-2}=0.411 p_{1-3}=0.032 p_{2-3}=0.081
HR, bpm	102.0 (87.6; 107.0)	86.0 (68.0; 98.0)	80.0 (66.0; 92.0)	p_{1-2}=0.008 p_{1-3}=0.001 p_{2-3}=0.386
Pulse, bpm	92.0 (77.3; 98.0)	78.0 (64.0; 84.0)	72.0 (62.0; 82.0)	p_{1-2}=0.007 p_{1-3}=0.001 p_{2-3}=0.417
Weight, kg	105.0 (85.8; 110.0)	104.0 (85.3; 110.0)	96.0 (87.4; 102.6)	p_{1-2}=0.061 p_{1-3}=0.055 p_{2-3}=0.491
BMI, kg/m^2	34.1 (28.0; 35.1)	34.5 (27.5; 36.3)	33.0 (29.3; 35.3)	p_{1-2}=0.061 p_{1-3}=0.062 p_{2-3}=0.223
Waist circumference, cm	106.0 (86.8; 115.8)	103.0 (86.0; 119.5)	102.0 (84.0; 115.1)	p_{1-2}=0.101 p_{1-3}=0.081 p_{2-3}=0.981
Hip circumference, cm	110.0 (102.0; 112.0)	112.0 (103.0; 121.0)	109.0 (102.0; 115.0)	p_{1-2}=0.061 p_{1-3}=0.304 p_{2-3}=0.338
EDS, cm	5.1 (4.7; 5.5)	5.4 (5.1; 5.9)	5.2 (5.0; 5.6)	p_{1-2}=0.009 p_{1-3}=0.072 p_{2-3}=0.158
ESS, cm	3.6 (3.3; 4.2)	3.8 (3.4; 4.3)	3.9 (3.6; 4.2)	p_{1-2}=0.350 p_{1-3}=0.081 p_{2-3}=0.614
EDV, mL	128.0 (102.0; 153.25)	150.0 (127.0; 176.0)	137.50 (118.5; 169.3)	p_{1-2}=0.003 p_{1-3}=0.011 p_{2-3}=0.362
ESV, mL	63.5 (47.0; 83.5)	83.0 (61.0; 105.4)	70.0 (62.0; 88.8)	p_{1-2}=0.002 p_{1-3}=0.001 p_{2-3}=0.033
SV, mL	66.0 (50.8; 76.5)	87.5 (82.9; 99.5)	73.0 (62.3; 85.8)	p_{1-2}=0.001 p_{1-3}=0.027 p_{2-3}=0.071
EF,%	50.0 (45.0; 55.3)	47.5 (42.0; 50.0)	55.0 (49.0; 59.0)	p_{1-2}=0.131 p_{1-3}=0.092 p_{2-3}=0.091
IVST, cm	1.3 (1.2; 1.4)	1.4 (1.2; 1.7)	1.3 (1.2; 1.6)	p_{1-2}=0.071 p_{1-3}=0.082 p_{2-3}=0.407
LV PWT, cm	1.2 (1.1; 1.3)	1.4 (1.3; 1.8)	1.3 (1.2; 1.5)	p_{1-2}=0.011 p_{1-3}=0.072 p_{2-3}=0.059
LA, cm	3.8 (3.4; 4.2)	4.5 (4.2; 5.3)	3.9 (3.5; 4.4)	p_{1-2}=0.011 p_{1-3}=0.054 p_{2-3}=0.061
Aorta diameter, cm	3.2 (3.0; 3.5)	3.4 (3.2; 3.6)	3.3 (3.1; 3.6)	p_{1-2}=0.081 p_{1-3}=0.062 p_{2-3}=0.560
LVMM, g	302.3 (250.2; 382.2)	325.8 (281.3; 392.1)	299.0 (248.4; 333.7)	p_{1-2}=0.005 p_{1-3}=0.971 p_{2-3}=0.042
LVMMI, g/m^2	147.1 (116.2; 182.7)	175.4 (139.0; 195.4)	160.4 (136.2; 179.6)	p_{1-2}=0.021 p_{1-3}=0.081 p_{2-3}=0.032
LV RWT	0.5 (0.4; 0.6)	0.5 (0.4; 0.6)	0.5 (0.4; 0.5)	p_{1-2}=0.107 p_{1-3}=0.791 p_{2-3}=0.232

Table 2

Metabolic profile of diabetic patients after myocardial infarction in the 1-year follow-up

Indicator, unit of measurement	Before treatment (n=74)	After treatment		Significance, (p)
		Patients without PCI, (n=20)	Patients with PCI, (n=50)	
	1	2	3	
Glucose, mmol/L	10.5 (7.1; 13.9)	6.8 (6.4; 8.0)	6.3 (5.6; 6.9)	p_{1-2}=0.007 p_{1-3}=0.001 p_{2-3}=0.007
Insulin, mcU/mL	31.2 (23.4; 39.7)	16.7 (15.2; 18.1)	13.1 (11.4; 15.0)	p_{1-2}=0.001 p_{1-3}=0.001 p_{2-3}=0.001
TC, mmol/L	5.1 (4.2; 6.1)	5.6 (5.1; 6.1)	4.9 (4.1; 5.7)	p_{1-2}=0.242 p_{1-3}=0.212 p_{2-3}=0.052
VLDL, mmol/L	1.0 (0.7; 1.3)	1.2 (0.5; 1.3)	0.9 (0.4; 1.2)	p_{1-2}=0.434 p_{1-3}=0.201 p_{2-3}=0.052
LDL, mmol/L	3.1 (2.6; 3.9)	3.6 (2.9; 4.0)	2.9 (2.1; 3.5)	p_{1-2}=0.242 p_{1-3}=0.008 p_{2-3}=0.052
TG, mmol/L	2.2 (1.6; 2.8)	2.4 (2.1; 3.3)	1.9 (1.2; 2.0)	p_{1-2}=0.205 p_{1-3}=0.001 p_{2-3}=0.051
HDL, mmol/L	1.1 (0.9; 1.3)	1.0 (0.7; 1.1)	1.2 (1.1; 1.4)	p_{1-2}=0.061 p_{1-3}=0.076 p_{2-3}=0.051
AI	3.8 (2.7; 4.7)	3.9 (3.4; 4.3)	3.2 (2.5; 3.6)	p_{1-2}=0.223 p_{1-3}=0.001 p_{2-3}=0.052
Adropin, pg/mL	14.1 (9.4; 16.9)	17.6 (11.9; 20.5)	19.9 (17.9; 21.1)	p_{1-2}=0.263 p_{1-3}=0.001 p_{2-3}=0.045
Irisin, ng/mL	1.9 (1.5; 2.2)	2.6 (2.3; 3.9)	3.4 (2.5; 4.1)	p_{1-2}=0.001 p_{1-3}=0.001 p_{2-3}=0.037
FABP4, ng/mL	10.1 (9.1; 11.9)	8.8 (7.8; 10.9)	7.3 (6.4; 8.3)	p_{1-2}=0.592 p_{1-3}=0.001 p_{2-3}=0.031
CTRP3, ng/mL	218.3 (191.9; 268.7)	257.0 (214.8; 288.4)	290.2 (283.6; 295.4)	p_{1-2}=0.189 p_{1-3}=0.001 p_{2-3}=0.001

Comparing the studied indicators between the subgroups after the treatment with medicinal therapy and PCI, a significant reduction in ESV by 15.66%, LVMM by 8.23%, LVMMI by 8.58%, serum levels of glucose by 7.06%, insulin by 21.26% and FABP4 by 16.89% was determined, as well as an increase in the serum levels of adropin by 13.59%, irisin by 30.77% and CTRP3 by 12.91%, respectively ($p<0.05$).

The course of AMI in diabetics was characterized by a high proportion of early AMI complications, which could affect the condition of diabetic patients in the long-term period post-AMI [1]. Serum adropin levels were significantly lower in patients with AMI compared to patients with stable angina or control individuals [13]. The serum adropin concentrations were lower in patients with type 2 DM. In addition, overweight or obese type 2 DM patients have been found to have significantly lower serum adropin levels [14].

Irisin is produced in high quantities by the myocardium. A significant correlation between serum irisin concentrations and different stages of myocardial infarction and cardiac repair has been demonstrated [5]. Several phases of heart repair after myocardial infarction have been described. The inflammatory process is involved in the initial period coupled with immune cell migration. In this phase, the immune cells start to clear and remove cellular debris and necrotic extracellular matrix. Fibrosis formation and neovascularization activation are induced simultaneously during the reparative stage after 3 to 5 days. At that point, ensuring energy metabolism regulated by the AMPK pathway and macrophage migration is essential in achieving the optimum level of repair processes. In the protracted inflammatory phase, the remaining dead cells and necrotic tissues affect subsequent regeneration, causing limited ability for cardiac remodeling and infarct expansion. Impaired heart function results in chamber dilation and disorders of myocardial functions [8]. Heart failure patients with preserved LV EF have been identified with elevated serum FABP4 levels that was associated with cardiac

remodeling and LV dysfunction resulted in unfavorable prognosis [4]. There is evidence of FABP4 involvement in an increased intracellular lipid deposition in patients with type 2 DM, the main consequence of which is myocardial dysfunction [9]. CTRP3 has shown an ability to protect myocardium against ischemic or reperfusion injury via the activation of LAMP1/JIP2/JNK pathway that culminated in attenuated myocardial injury, improved LV function, lowered myocardial infarction incidence, and diminished myocardial apoptosis [10]. Moreover, scientists have determined that serum CTRP3 concentrations were lower in patients with acute coronary syndrome than those in control subjects [6]. We have found that the patients received medicinal therapy and PCI had improved echocardiographic indicators such as EDV, ESV, SV as well as reduced parameters of carbohydrate metabolism. After the treatment with medicinal therapy alone, type 2 DM patients with post-infarction cardiosclerosis were revealed with the significantly increased EDS, LV PWT, LA, LVMM, LVMMI, which indicated dilation of the left heart and increased heart size. Patients after PCI had a gradual tendency to echocardiographic changes, the significant decrease in LDL cholesterol, TG and AI compared to those before treatment. Lipid profile did not differ significantly before and after treatment in patients prescribed medicinal therapy. The study has concluded that there were changes in energy and adipokine metabolism.

Conclusions

1. Imbalanced indicators of energy and adipokine metabolism due to low serum levels of adropin, irisin, CTRP3 and increased concentrations of FABP4 in type 2 diabetic patients after myocardial infarction have been revealed.

2. Moderation of structural and functional changes in the LV myocardium has been found in patients following PCI compared to those in patients who received only medicinal therapy.

3. Increased values of LVMMI and LV RWT have been detected in type 2 diabetic patients after myocardial infarction that corresponded to concentric hypertrophy of the LV myocardium.

Prospects for further research

We consider investigation of markers of energy and adipokine metabolism in type 2 DM patients with complicated course of myocardial infarction to be perspective.

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