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Koteliukh Mariia Yuriivna,
Candidate of medical sciences, Associate Professor of Kharkiv National Medical University, Department of Internal Medicine No. 2 and Clinical Immunology and Allergology named after academician L.T. Malaya
ekoteliukh@gmail.com
https://orcid.org/0000-0001-6090-4835
Kharkiv, Ukraina

Kravchun Pavlo Grygorovych,
Doctor of medical sciences, professor of Kharkiv National Medical University, Department of Internal Medicine No. 2 and Clinical Immunology and Allergology named after academician L.T. Malaya
pg.kravchun@knmu.edu.ua
https://orcid.org/0000-0002-8285-6763
Kharkiv, Ukraina

Dobrovolska Inna Mykolayivna,
Candidate of medical sciences, Associate Professor of Kharkiv National Medical University, Department of Internal Medicine No. 2 and Clinical Immunology and Allergology named after academician L.T. Malaya
im.dobrovolska@knmu.edu.ua
https://orcid.org/0000-0003-0458-6734
Kharkiv, Ukraina

Characteristics of the acute myocardial infarction course in patients with type 2 diabetes mellitus before and after reperfusion therapy

Introduction. Scientific research is currently being done to diagnose and manage comorbid conditions in patients. The importance of studying the pathophysiological mechanisms of acute myocardial infarction (AMI) in patients with type 2 diabetes mellitus (DM) has been proven.

The purpose of the study: to conduct a retrospective analysis before and after reperfusion therapy by evaluating the features of the AMI course in patients with type 2 DM.

Material and methods. The study was conducted from January 10, 2020 to January 12, 2021. In total, 74 STEMI patients with or without type 2 DM, aged 59.42±7.66 years were examined at the Intensive Care Unit of the Government Institution "L. T. Malaya National Therapy Institute of the National Academy of Medical Sciences of Ukraine" and the Kharkiv Railway Clinical Hospital No. 1 of the branch "Center of Healthcare" of Public Joint Stock Company "Ukrainian Railway". Serum levels of insulin, adropin, irisin, fatty acid binding protein 4 (FABP4) and C1q/TNF-related protein (CTRP3) were detected by enzyme-linked immunosorbent assay. Analysis and processing of the findings obtained after examinations were carried out using the computer program IBM SPPS software, version 27.0 (IBM Inc., USA, 2020).

Results and discussion. Before the treatment, patients with AMI and type 2 DM had significantly higher values of end-systolic size (ESS) by 5.56%, left atrium (LA) by 10.53%, left ventricular myocardial mass index (LVMMI) by 6.87% and lower values of systolic blood pressure (SBP) by 11.76%, atherogenic index (AI) by 5.80%, glucose by 25.71%, insulin by 39.92%, irisin by 15.34%, FABP4 by 19.76% compared to the values of these indicators 14 days after medicamentous therapy ($p<0.05$). Patients with AMI and type 2 DM had significantly higher values of adropin by 46.10%, irisin by 20.11%, CTRP3 by 28.78% and lower values of SBP by 17.65%, diastolic blood pressure by 12.38%, heart rate (HR) by 13.73%, pulse by 10.87%, body mass index by 5.57%, weight by 9.52%, glucose by 26.67%, insulin by 40, 75%, total cholesterol by 10.04%, very low-density lipoproteins by 24.75%, low-density lipoproteins by 8.52%, triglycerides by 27.06%, AI by 10.55%, FABP4 by 37.04% before treatment as compared to the values of these indicators 14 days after percutaneous coronary intervention (PCI) ($p<0.05$). Comparing the studied indicators between subgroups after medicamentous treatment and PCI, a significant decrease in HR by 8.33%, ESS by 4.11%, LA by 9.52%, LVMMI by 5.53%, FABP4 by 21.53% and an increase in the levels of adropin by 36.99%, irisin by 41.88%, CTRP3 by 30.25%, high-density lipoproteins (HDL) by 10.28% were identified ($p<0.05$).

Conclusions. When compared PCI advantages due to the absence of a tendency to the left heart dilatation over the medicamentous therapy, improvements of energy and adipokine metabolism have been observed amid increased levels of adropin, irisin and CTRP3 and decreased levels of FABP4 in patients with AMI and type 2 DM. It should be noted that there has been a significant improvement in the state of energy and adipokine profiles under the conditions of performing PCI.

Key words: diabetes, myocardial infarction, markers, metabolism.

Котелюх Марія Юріївна, кандидат медичних наук, доцент кафедри внутрішньої медицини № 2 і клінічної імунології та алергології імені академіка Л.Т. Малої, Харківський національний медичний університет, koteliukh@gmail.com, https://orcid.org/0000-0001-6090-4835, м. Харків, Україна

Кравчун Павло Григорович, доктор медичних наук, професор кафедри внутрішньої медицини № 2 і клінічної імунології та алергології імені академіка Л.Т. Малої, Харківський національний медичний університет, pg.kravchun@knmu.edu.ua, https://orcid.org/0000-0002-8285-6763, м. Харків, Україна

Добровольська Інна Миколаївна, кандидат медичних наук, доцент кафедри внутрішньої медицини № 2 і клінічної імунології та алергології імені академіка Л.Т. Малої, Харківський національний медичний університет, im.dobrovolska@knmu.edu.ua, https://orcid.org/0000-0003-0458-6734, м. Харків, Україна

Особливості перебігу гострого інфаркту міокарда у хворих з цукровим діабетом 2 типу до та після реперфузійної терапії

Вступ. Сьогодні ведуться наукові пошуки щодо діагностики та лікування пацієнтів із коморбідним станом. Доведено важливість дослідження патофізіологічних механізмів гострого інфаркту міокарда (ГІМ) у хворих із цукровим діабетом (ЦД) 2 типу.

Мета дослідження: провести ретроспективний аналіз до та після реперфузійної терапії шляхом оцінки особливостей перебігу ГІМ у хворих із ЦД 2 типу.

Матеріал та методи. Дослідження проведено з 10 січня 2020 року по 12 січня 2021 року. На базі відділення інтенсивної терапії Державної установи «Національному інституті терапії імені Л.Т. Малої Національної академії медичних наук України» та Харківської клінічної лікарні на залізничному транспорті №1 філії «Центр охорони здоров'я» Публічного акціонерного товариства «Українська залізниця» було обстежено 74 пацієнтів на ГІМ з елевацією сегменту ST та ЦД 2 типу віком 59,42±7,66 років. Визначення вмісту інсуліну, адропіну, ірисину, білка, що зв'язує жирні кислоти 4 (FABP4) та C1q/TNF – асоційованого білка 3 (CTRP3) у сироватці крові пацієнтів проведено імуноферментним методом. Аналіз і обробка результатів обстеження хворих здійснювалася за допомогою комп'ютерної програми IBM SPPS software version 27,0 (IBM Inc., USA, 2020).

Результати досліджень та їх обговорення. До лікування у пацієнтів із ГІМ та ЦД 2 типу встановлено вірогідно більш високі значення кінцево-систолічний розмір (КСР) на 5,56 %, ліве передсердя (ЛП) на 10,53%, індекс маси міокарда лівого шлуночка (ІММЛШ) на 6,87% та більш низькі значення систолічного артеріального тиску (САТ) на 11,76%, коефіцієнта атерогенності (КА) на 5,80%, глюкози на 25,71%, інсуліну на 39,92%, ірисину на 15,34%, FABP 4 на 19,76% у порівнянні зі значенням цих показників у пацієнтів через 14 днів після медикаментозної терапії відповідно (p<0,05). До лікування у пацієнтів із ГІМ та ЦД 2 типу встановлено вірогідно більш високі значення адропіну на 46,10%, ірисину на 20,11%, CTRP 3 на 28,78% та більш низькі значення САТ на 17,65%, діастолічного артеріального тиску на 12,38%, частоти серцевих скорочень (ЧСС) на 13,73%, пульсу на 10,87%, індексу маси тіла на 5,57%, ваги на 9,52%, глюкози на 26,67%, інсуліну на 40,75%, загального холестерину на 10,04%, ліпопротеїдів дуже низької щільності 24,75%, ліпопротеїдів низької щільності на 8,52%, тригліцеридів на 27,06%, КА на 10,55%, FABP 4 на 37,04% у порівнянні зі значенням цих показників у пацієнтів через 14 днів після перкутанного коронарного втручання (ПКВ) відповідно (p<0,05). Порівнюючи досліджені показники між підгрупами після медикаментозного лікування і ПКВ, визначено достовірне зменшення ЧСС на 8,33%, КСР на 4,11%, ЛП на 9,52%, ІММЛШ на 5,53%, FABP 4 на 21,53% та збільшення рівнів адропіну на 36,99%, ірисину на 41,88%, CTRP 3 на 30,25%, ліпопротеїдів високої щільності на 10,28% відповідно (p<0,05).

Висновки. При порівняні ПКВ над медикаментозною терапією спостерігається відсутність тенденції до дилатації лівих відділів серця, покращення енергетичного та адипокінового метаболізму на тлі збільшення рівнів адропіну, ірисину та CTRP 3 та зниження рівнів FABP 4 у хворих на ГІМ та ЦД 2 типу. Слід зазначити, що за умов виконання ПКВ відбувається значне покращення стану енергетичного та адипокінового обмінів.

Ключові слова: діабет, інфаркт міокарда, маркери, метаболізм.

Introduction. Greater than about 7 million people worldwide are diagnosed with acute myocardial infarction (AMI) annually. For patients with ST-segment elevation AMI (STEMI), coronary catheterization and percutaneous coronary intervention (PCI) within 2 hours of presentation reduce mortality [1]. In patients with STEMI, optimization of primary PCI is crucial to improve myocardial status and prevent reperfusion injury [2]. Higher all-cause mortality rates have been reported among type 2 diabetes mellitus (DM) patients compared to those without DM. Moreover, significantly higher 1-year mortality has been found to be associated with insulin treatment in diabetic patients as compared to diabetic individuals without insulin use [3]. According to the International Study of Comparative Health Effectiveness with Medical and Invasive Approaches (ISCHEMIA) and Complete versus Culprit-Only Revascularization Strategies to Treat Multivessel Disease after Early PCI for STEMI (COMPLETE) trials, PCI helps in preventing cardiac death and the development of AMI in patients with an unstable form of coronary heart disease (CHD) [4]. The combination of AMI with comorbid diabetes is an independent predictor of a worse functional body state, and these patients most often develop complications of AMI and heart failure [5]. It is worth noting that type 2 DM increases 5-year mortality in elderly patients with AMI. While younger patients with AMI and type 2 DM are more likely to have complications in the early period after AMI compared to patients of the same age group but without type 2 DM [6]. The adverse effect of DM on in-hospital mortality rates after AMI was further confirmed by a recent cohort study including more than 5,000 STEMI patients following primary PCI [7]. It has been reported that patients with STEMI and DM had higher hypertension, hyperlipidemia, left main coronary artery disease, and higher in-hospital mortality rates [8]. DM is an independent predictor of major adverse cardiac events in patients after PCI [9]. Pathophysiological processes underlying the development and determining the course of AMI in diabetic patients demand a thorough and comprehensive examination in order to reduce the risk of adverse recurrent cardiovascular events.

The purpose of the study: to conduct a retrospective analysis before and after reperfusion therapy by evaluating the features of the AMI course in patients with type 2 DM.

The scientific study is a fragment of the scientific work of the Department of Internal Medicine No. 2 and Clinical Immunology and Allergology named after Academician L. T. Malaya "Prediction of the course, improvement of diagnosis and treatment of ischemic heart disease and arterial hypertension in patients with metabolic disorders", project execution time 2020 – 2022.

Materials and methods. The study was conducted from January 10, 2020 to January 12, 2021. In total, 74 STEMI patients with or without type 2 DM, aged 59.42±7.66 years, who were hospitalized to the Intensive Care Unit of the Government Institution "L. T. Malaya National Therapy Institute of the National Academy of Medical Sciences of Ukraine" and the Kharkiv Railway Clinical Hospital No. 1 of the branch "Center of Healthcare" of Public Joint Stock Company "Ukrainian Railway".

All examinations were carried out with the prior consent of the patients (written consent was obtained

from all patients regarding the appropriate diagnostic and therapeutic measures), and the technique of performing each test was in accordance with the 1975 Helsinki Declaration of Human Rights and its 1983 revision, the Council of Europe Convention on Human Rights and biomedicine and legislation of Ukraine.

The diagnosis of STEMI was made based on clinical, instrumental and laboratory data according to the criteria proposed by the consensus of the European Society of Cardiology [10]. Diagnosis and treatment of type 2 DM was carried out in accordance with the American Diabetes Association and the European Association for the Study of Diabetes 2018 recommendations [11].

STEMI patients with or without type 2 DM were included in the study.

Exclusion criteria were type 1 DM, non-ST segment elevation AMI (NSTEMI), COVID-19, autoimmune diseases, diseases of the pituitary gland and hypothalamus, thyroid disease, the presence of symptomatic hypertension, pathology of the heart valvular apparatus, IV FC chronic heart failure before myocardial infarction, the presence of chronic obstructive pulmonary disease, severe disorders of liver and kidney functions, severe anemia, oncological diseases.

All tests were performed at the Biochemical Department of the Central Research Laboratory of the Kharkiv National Medical University. Blood serum samples were taken from patients on day 1 and stored at -20 ºC. Serum levels of insulin, adropin, irisin, fatty acid binding protein 4 (FABP4) and C1q/TNF-related protein (CTRP3) were detected by enzyme-linked immunosorbent assay using an analyzer "Labline-90" (Austria) with commercial test systems "Human Insulin" (Monobind Inc, USA), "Human Adropin" (Elabscience, USA), "Human Fibronectin type III domain-containing protein 5" (Elabscience, USA), "Human FABP4" (Elabscience, USA) and "Human CTRP3" (Aviscera Bioscience, USA), respectively, following the instructions from manufacturers. Serum total cholesterol (TC) and high-density lipoprotein (HDL) cholesterol were quantified by peroxidase enzymatic technique with assay kits "Cholesterol Liquicolor" (Human GmbH, Germany) and "HDL Cholesterol liquicolor" (Human GmbH, Germany), respectively. Triglyceride (TG) levels were measured by enzymatic colorimetric method using an assay kit "Triglycerides" (Human GmbH, Germany). The atherogenic index (AI) was calculated by the standard A. M. Klimov formula. The Friedewald formula was used to define plasma levels of very low-density lipoprotein (VLDL) and low-density lipoprotein (LDL). Fasting blood glucose level was detected by glucoseoxidase method with commercial test system "Human Glucose" (LLC NPP "Filisit-Diagnostics", Ukraine). Body mass index (BMI) was calculated as weight divided by height squared (kg/m^2).

All patients underwent coronary angiography. Coronary artery stenting was not performed for patients who refused to receive the procedure or a lesion localization in the coronary artery trunk. Doppler echocardiographic examination was implemented according to the generally accepted methodology on a Radmir ULTIMA Pro30 ultrasound scanner. End-diastolic size (EDS), end-systolic size (ESS), end-diastolic volume (EDV), end-systolic volume (ESV) of the left ventricle (LV), stroke volume (SV), LV ejection fraction (EF), interventricular septum thickness (IVST), aorta diameter, left atrium (LA) size, and LV posterior wall thickness (LVPWT) in diastole were estimated. LV myocardial mass (LVMM), LVMM index (LVMMI = LVMM/body surface area (m^2)) were calculated. Relative wall thickness (RVT) of the LV was calculated based on the formula: LVRVT = (LVPWT + IVST)/LV EDS)). Analysis and processing of the findings after patient examinations were carried out using the computer program IBM SPPS software, version 27.0. (IBM Inc., USA, 2020) using non-parametric methods for evaluating the obtained results. The difference was considered significant at a value of $p<0.05$.

Results and discussion. Tables 1 and 2 present the dynamic indicators of carbohydrate, lipid, energy and adipokine metabolism, structural and functional parameters of the LV in patients with AMI and 2 type DM before treatment and 14 days after myocardial revascularization. There were no significant differences in anthropometric and cardiohemodynamic parameters, values of the lipid profile, except for systolic blood pressure (SBP), LA, LVMMI, glucose, insulin, AI, irisin, FABP4 before the treatment and after medicamentous therapy ($p>0.05$). The same dynamics of indicators, except SBP, diastolic blood pressure (DBP), pulse, heart rate (HR), body mass index (BMI), weight, ESS, parameters of carbohydrate, energy, lipid and adipokine metabolism were identified before and after PCI ($p>0.05$).

Before the treatment, patients with AMI and type 2 DM had significantly higher values of such indicators as ESS by 5.56%, LA by 10.53%, LVMMI by 6.87% and lower values of indicators such as SBP by 11.76%, AI by 5.80%, glucose by 25.71%, insulin by 39.92%, irisin by 15.34%, FABP4 by 19.76% compared to the values of these indicators 14 days after medicamentous therapy ($p<0.05$).

Patients with AMI and type 2 DM had significantly higher values of indicators such as adropin by 46.10%, irisin by 20.11%, CTRP3 by 28.78% and lower values of indicators such as SBP by 17.65%, DBP by 12.38%, HR by 13.73%, pulse by 10.87%, BMI by 5.57%, weight by 9.52%, glucose by 26.67%, insulin by 40.75%, TC by 10.04%, VLDL by 24.75%, LDL by 8.52%, TG by 27.06%, AI by 10.55%, FABP4 by 37.04% before treatment as compared to the values of these indicators 14 days after PCI ($p<0.05$).

Serum adropin levels in patients with CHD were lower than those in healthy subjects, indicating that decreased adropin concentrations may play an essential role in the development of CHD [12]. Serum irisin concentrations were decreased in patients with AMI, and also correlated with the severity of stable CHD [13]. Recall that circulating FABP4 levels have been recognized an independent prognostic predictor of serious cardiovascular events in CHD patients after coronary interventions. In addition, circulating FABP4 levels have been featured among predictors of cardiovascular events in patients after coronary interventions [14]. Meanwhile it is known that CTRP3 activates the anti-inflammatory and anti-atherosclerotic mechanisms of CHD, suppressing endothelial inflammation and reducing plaque formation by inhibiting both the secretion of inflammatory cytokines and the expression of adhesion molecules [15].

Table 1

Dynamics of anthropometric and cardiohemodynamic indicators during the hospital period after the use of medicamentous therapy and PCI in patients with AMI and type 2 DM (Me, Q1; Q3)

Parameter, units of measurement	Before treatment (n=74)	After treatment		Significance (p)
		Patients without PCI (n=22)	Patients after PCI (n=52)	
	1	2	3	
SBP, mm Hg	170.0 (140.0; 180.0)	150.0 (130.0; 162.5)	140.0 (120.0; 150.0)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
DBP, mm Hg	105.0 (85.0; 110.0)	98.0 (81.5; 104.2)	92.0 (78.0; 98.0)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
HR, bpm	102.0 (87.25; 107.0)	96.0 (73.0; 101.0)	88.0 (78.0; 92.0)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}<0.05$
Pulse, bpm	92.0 (77.25; 98.0)	86.0 (68.0; 94.5)	82.0 (76.0; 86.0)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
Weight, kg	105.0 (85.75; 110.0)	103.0 (84.24; 109.0)	95.0 (86.25; 101.5)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
BMI, kg/m^2	34.1 (28.0; 35.1)	33.0 (26.5; 35.3)	32.20 (28.33; 34.1)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
Waist circumference, cm	106.0 (86.75; 115.75)	102.0 (85.0; 118.75)	101.0 (87.0; 105.75)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
Thigh circumference, cm	110.0 (102.0; 112.0)	111.0 (102.0; 119.0)	108.0 (102.0; 112.0)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
EDS, cm	5.10 (4.7; 5.5)	5.2 (4.63; 5.6)	5.1 (4.9; 5.5)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
EDS, cm	3.6 (3.3; 4.23)	3.8 (3.5; 4.08)	3.65 (3.2; 4.23)	$p_{1-2}<0.05$ $p_{1-3}>0.05$ $p_{2-3}<0.05$
EDV, ml	128.0 (102.0; 153.25)	128.5 (98.5; 153.25)	127.0 (113.0; 152.25)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
ESV, ml	63.5 (47.0; 83.5)	60.0 (48.75; 80.0)	66.0 (54.25; 76.0)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
SV, ml	66.0 (50.75; 76.5)	65.5 (51.25; 75.5)	65.0 (51.25; 82.75)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
LV EF, %	50.0 (45.0; 55.25)	49.0 (42.0; 57.25)	53.0 (46.25; 58.0)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
IVST, cm	1.3 (1.16; 1.4)	1.2 (1.1; 1.3)	1.2 (1.1; 1.3)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
LVPWT, cm	1.2 (1.1; 1.3)	1.2 (1.1; 1.33)	1.2 (1.1; 1.3)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
LA, cm	3.8 (3.4; 4.2)	4.2 (3.45; 4.33)	3.8 (3.4; 4.28)	$p_{1-2}<0.05$ $p_{1-3}>0.05$ $p_{2-3}<0.05$
Aorta diameter, cm	3.2 (3.0; 3.5)	3.2 (2.98; 3.5)	3.2 (3.03; 3.5)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
LVMM, g	302.3 (250.2; 382.18)	305.25 (248.4; 351.63)	293.65 (212.5; 392.13)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$
LVMMI, g/m^2	147.1 (116.15; 182.7)	157.2 (111.9; 190.52)	148.5 (128.18; 176.73)	$p_{1-2}<0.05$ $p_{1-3}>0.05$ $p_{2-3}<0.05$
LVRVT	0.48 (0.44; 0.57)	0.48 (0.39; 0.52)	0.44 (0.41; 0.5)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}>0.05$

Table 2

Dynamics of the studied indicators during the hospital period after medicamentous therapy and PCI in patients with AMI and type 2 DM (Me, Q1; Q3)

Parameter, units of measurement	Before treatment (n=74)	After treatment		Significance (p)
		Patients without PCI (n=22)	Patients after PCI (n=52)	
	1	2	3	
Glucose, mmol/L	10.50 (7.10; 13.95)	7.80 (6.65; 10.00)	7.70 (6.24; 8.90)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
Insulin, μlU/mL	31.24 (23.43; 39.66)	18.77 (15.46; 22.63)	18.51 (14.94; 25.04)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
Adropin, pg/mL	14.12 (9.44; 16.94)	15.06 (11.20; 17.53)	20.63 (18.87; 21.45)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}<0.05$
Irisin, ng/mL	1.89 (1.49; 2.21)	1.60 (1.44; 1.97)	2.27 (2.04; 2.57)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}<0.05$
FABP4, ng/mL	10.07 (9.13; 11.92)	8.08 (6.85; 9.41)	6.34 (5.46; 6.96)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}<0.05$
CTRP3, ng/mL	218.32 (191.95; 268.68)	215.86 (204.29; 228.49)	281.15 (258.66; 292.70)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}<0.05$
TC, mmol/L	5.08 (4.21; 6.12)	5.02 (4.12; 5.84)	4.57 (4.01; 5.63)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
VLDL, mmol/L	1.01 (0.74; 1.27)	0.89 (0.40; 1.22)	0.76 (0.40; 1.22)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
LDL, mmol/L	3.05 (2.59; 3.87)	2.64 (2.02; 3.24)	2.79 (2.06; 3.52)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
TG, mmol/L	2.18 (1.57; 2.84)	1.88 (1.22; 3.06)	1.59 (1.13; 2.03)	$p_{1-2}>0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$
HDL, mmol/L	1.13 (0.97; 1.31)	1.07 (0.72; 1.19)	1.18 (1.02; 1.44)	$p_{1-2}>0.05$ $p_{1-3}>0.05$ $p_{2-3}<0.05$
AI	3.79 (2.69; 4.70)	3.57 (2.49; 4.18)	3.39 (2.59; 3.94)	$p_{1-2}<0.05$ $p_{1-3}<0.05$ $p_{2-3}>0.05$

Comparing the studied indicators between subgroups after medicamentous treatment and PCI, a significant decrease in HR by 8.33%, ESS by 4.11%, LA by 9.52%, LVMMI by 5.53%, FABP4 by 21.53% and an increase in the levels of adropin by 36.99%, irisin by 41.88%, CTRP3 by 30.25%, HDL by 10.28% were identified ($p<0.05$).

Conclusions. Medicamentous therapy and PCI have resulted in the decreased values of SBP and AI as well as improved carbohydrate, energy and adipokine metabolism. When compared PCI advantages due to the absence of a tendency to the left heart dilatation over the medicamentous therapy, improvements of energy and adipokine metabolism have been observed amid increased levels of adropin, irisin and CTRP3 and decreased levels of FABP4 in patients with AMI and type 2 DM. It should be noted that there has been a significant improvement in the state of energy and adipokine profiles under the conditions of performing PCI.

Greater insight into potential mechanisms of the AMI development and course with underlying type 2 DM may suggest a modern therapeutic strategy for the treatment of AMI.

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