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Relationship between Biomarkers of Carbohydrate, Energy and Adipokine Metabolism in Patients with Acute Myocardial Infarction and Type 2 Diabetes Mellitus

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The purpose of the study was to examine the indicators of carbohydrate, energy and adipokine metabolism in acute myocardial infarction patients with or without type 2 diabetes mellitus.

Materials and methods. A total of 134 patients with ST-segment elevation acute myocardial infarction in the presence or absence of type 2 diabetes mellitus aged 58.97 ± 7.92 years were examined in the Government Institution "L. T. Malaya Therapy National Institute of the National Academy of Medical Sciences of Ukraine" and Kharkiv Railway Clinical Hospital No. 1 of the "Center of Healthcare" branch of Public Joint Stock Company "Ukrainian Railway". The main group (group 1) included 74 patients with acute myocardial infarction and type 2 diabetes mellitus. The comparison group (group 2) was represented by 60 patients with acute myocardial infarction without type 2 diabetes mellitus. The control group was composed of 20 otherwise healthy individuals. Serum concentrations of insulin, adropin, irisin, fatty acid binding protein 4 and C1q/TNF-related protein were measured by enzyme-linked immunosorbent assays.

Results and discussion. It was found that the levels of glucose, insulin, HOMA-IR index were increased in acute myocardial infarction patients with or without type 2 diabetes mellitus when compared with the control group ($p<0.05$). In acute myocardial infarction patients with type 2 diabetes mellitus, the serum levels of adropin, irisin and C1q/TNF-related protein were significantly lower, while the fatty acid binding protein 4 levels were higher as compared to those in the control group ($p<0.05$). Acute myocardial infarction patients with type 2 diabetes mellitus demonstrated significantly lower serum concentrations of adropin and C1q/TNF-related protein in comparison with acute myocardial infarction patients without type 2 diabetes mellitus ($p<0.05$). Negative correlations between blood glucose and adropin ($r = -0.499$, $p<0.001$), irisin ($r = -0.495$, $p<0.001$), fatty acid binding protein 4 ($r = -0.518$, $p<0.001$), between HOMA-IR and adropin ($r = -0.304$, $p<0.02$), fatty acid binding protein 4 ($r = -0.429$, $p = 0.001$), as well as positive correlations between blood glucose levels and C1q/TNF-related protein ($r = 0.435$, $p = 0.001$) were revealed in acute myocardial infarction patients. In acute myocardial infarction patients with type 2 diabetes mellitus, negative correlations were found between blood glucose and adropin ($r = -0.504$, $p<0.001$), irisin ($r = -0.520$, $p = 0.03$), C1q/TNF-related protein ($r = -0.546$, $p<0.001$); between blood insulin and adropin ($r = -0.361$, $p = 0.017$), irisin ($r = -0.396$, $p = 0.01$), C1q/TNF-related protein ($r = -0.361$, $p = 0.018$); between adropin and HOMA-IR ($r = -0.508$, $p = 0.001$), C1q/TNF-related protein ($r = -0.436$, $p = 0.003$), as well as positive correlation between blood glucose and fatty acid binding protein 4 ($r = 0.508$, $p = 0.007$); between blood insulin and fatty acid binding protein 4 ($r = 0.501$, $p = 0.001$); between HOMA-IR and fatty acid binding protein 4 ($r = 0.516$, $p<0.001$).

Conclusion. Characteristics of adropin, irisin, fatty acid binding protein 4 and C1q/TNF-related protein serum levels are evidence of violated energy and adipokine metabolism in both diabetic and non-diabetic patients with acute myocardial infarction. Relationships between adropin, irisin, fatty acid binding protein 4 and C1q/TNF-related protein could indicate involvement of these markers in carbohydrate metabolism.

Keywords: adipokine, carbohydrate metabolism, acute myocardial infarction, energy homeostasis, type 2 diabetes mellitus.

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Introduction. Type 2 diabetes mellitus (DM) is one of the most common diseases worldwide. Globally, an estimated 462 million individuals are affected by type 2 DM, corresponding to 6.28% of the world's population [1]. The prevalence of this disease has increased significantly worldwide in recent years and its increase of 9.9% is expected by 2045 [2]. Diabetes influences the functionality and quality of life resulting in significant morbidity and premature mortality [3]. Type 2 DM is associated with two-fold higher in-hospital and long-term mortality rates as well as with a higher risk of recurrent cardiovascular events, and also it is a common comorbidity in acute myocardial infarction

(AMI) [4]. According to the World Health Organization, the death rate from coronary heart disease (CHD) was 8.9 million cases around the world in 2019 [5].

Both diabetic and non-diabetic patients with elevated serum glucose levels were at increased risk for developing AMI complications [6, 7]. The mechanisms of early and late AMI complications development in diabetic patients are not well understood and still the subject of comprehensive discussion. Various pathophysiological pathways of AMI development and course are currently studied. One of these ways is the examination of energy and adipokine homeostasis.

Biomarkers of energy homeostasis (adropin and irisin) and adipokine system (fatty acid binding protein 4 (FABP4) and C1q/TNF-related protein (CTRP3)) are involved in the regulation of carbohydrate metabolism and associated with cardiovascular diseases [8-11]. The effect of energy and adipokine homeostasis markers on carbohydrate metabolism in diabetic patients with AMI needs to be studied in a separate patient cohort.

The purpose of the study was to examine the indicators of carbohydrate, energy and adipokine metabolism in AMI patients with or without type 2 DM.

Materials and methods. The study was conducted from September 1, 2018 to December 31, 2020. A total of 134 patients with ST-segment elevation AMI (STEMI) in the presence or absence of type 2 DM aged 58.97 ± 7.92 years hospitalized in the intensive care unit of Government Institution "L. T. Malaya Therapy National Institute of the National Academy of Medical Sciences of Ukraine" and Kharkiv Railway Clinical Hospital No. 1 of the "Center of Healthcare" branch of Public Joint Stock Company "Ukrainian Railway" were enrolled in the study. The main group (group 1) included 74 patients with AMI and type 2 DM with a mean age of 59.42 ± 7.66 years. The comparison group (group 2) was represented by 60 patients with AMI without type 2 DM aged 58.42 ± 8.25 years. Control indicators of biochemical and enzyme-linked immunosorbent assays were obtained from 20 otherwise healthy individuals.

The study was carried out in compliance with the basic provisions of the "Rules of ethical principles of scientific medical research with human participation", approved by the Declaration of Helsinki (1964-2013), ICH GCP (1996), EEC Directive No. 609 (dated 24.11.1986), Orders of the Ministry of Health of Ukraine No. 690 (dated 23.09.2009), No. 944 (dated 14.12.2009), No. 616 (dated 03.08.2012). All the participants were informed about the goals, organization, methods of examination and signed an informed consent to participate in the completely anonymous study.

STEMI was diagnosed based on clinical, instrumental and laboratory data, according to the criteria proposed by the Expert Consensus of the European Society of Cardiology [12]. Diagnosis and treatment of type 2 DM was performed according to the American Diabetes Association and the European Association for the Study of Diabetes (2018, 2019) [13, 14].

The inclusion criteria were the presence or absence of type 2 DM in patients with STEMI.

The 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, chronic heart failure (CHF) FC IV to myocardial infarction, chronic obstructive pulmonary disease, severe liver and renal dysfunction, severe anemia, malignancy.

All diagnostic tests were carried out in the Biochemical Department of the Central Research Laboratory of Kharkiv National Medical University. Blood serum samples were collected from the patients on day 1 and stored at -80 ºC. Serum concentrations of insulin, adropin, irisin, FABP4, CTRP3 of patients were measured by enzyme-linked immunosorbent assay using an analyzer "Labline-90" (Austria) with commercial test reagents "Human insulin" (Monobind Inc., Lake Forest, USA), "Human adropin (AD)" (Elabscience, Houston, USA), "Human Fibronectin type III domain-contact protein 5 (FNDC5)" (Elabscience, USA), "Human Fatty Acid Binding Protein 4 (FABP4)" (Elabscience, USA), Human CTRP3 (Aviscera Bioscience Inc., Santa Clara, USA) following the manufacturers' instructions, respectively. Fasting capillary blood glucose level was determined by glucose oxidase method. Insulin resistance (IR) was calculated by the Homeostatic Model of Insulin Resistance Assessment (HOMA-IR).

The results obtained were processed using statistical techniques and the computer program IBM SPPS software, version 27.0, (IBM Inc., USA, L-CZAA-BKK-MKE license, 2020). The Shapiro-Wilks test was used to assess whether indicators of all patient groups were normally distributed. Data were presented in the conventional form (arithmetic mean (M) and standard deviation (SD)). Significant differences between the means of normally distributed numerical parameters were statistically compared using one-way analysis of variance (ANOVA) with Bonferroni correction for multiple comparisons. Correlations between parameters were analyzed by the Pearson correlation coefficient (r). A p value ≤ 0.05 was considered statistically significant.

Results and discussion. Based on the study analysis, it was found that the levels of glucose, insulin, HOMA index were significantly increased in AMI patients with or without type 2 DM when compared with the control group ($p<0.05$) (**Table 1**).

Table 1 – The state of carbohydrate, energy and adipokine metabolism in AMI patients with or without type 2 DM

Indicators	AMI patients with type 2 DM, n=74	AMI patients without type 2 DM, n=60	Control group, n=20	Significance, (p)		
	1	2	3			
Glucose on day 1, mmol/L	11.23±4.57	7.12±2.45	4.75±0.34		2	3
				1	<0.001	<0.001
				2	-	0.031
Insulin on day 1, μIU/mL	32.51±10.56	26.65±7.82	5.97±1.92		2	3
				1	0.001	<0.001
				2	-	<0.001
HOMA-IR on day 1	17.17±11.16	8.51±4.54	1.20±0.38		2	3
				1	<0.001	<0.001
				2	-	0.002
Adropin on day 1, pg/mL	13.65±5.12	16.92±6.12	23.58±2.56		2	3
				1	0.002	<0.001
				2	-	<0.001
Irisin on day 1, ng/mL	1.86±0.43	2.22±0.93	5.97±2.1		2	3
				1	0.107	<0.001
				2	-	<0.001
FABP4 on day 1, ng/mL	10.53±2.01	9.76±2.07	5.02±1.92		2	3
				1	0.095	<0.001
				2	-	<0.001
CTRP3 on day 1, ng/mL	226.06±52.11	272.31±22.32	325.97±42.22		2	3
				1	<0.001	<0.001
				2	-	<0.001

The adropin serum levels were by 19.33% and 42.11% lower in AMI diabetic patients on day 1 as compared to those in AMI non-diabetic patients and the control group (p<0.05), respectively. The irisin serum levels were significantly by 16.22% and 68.84% less in AMI patients with type 2 DM on day 1 in comparison to those in non-diabetic AMI patients and the control group (p<0.05), respectively. The measured adropin serum levels on day 1 in AMI patients without type 2 DM were found to be by 28.24% lower compared to those of the control group (p<0.05), and the irisin serum levels were significantly by 62.81% less as compared to those of the control individuals (p<0.05) (**Table 1**).

In diabetic AMI patients, the FABP4 serum levels on day 1 were significantly by 7.89% and 2.1 times higher compared with non-diabetic AMI patients and the control group (p<0.05), respectively, while the serum concentrations of CTRP3 on day 1 were significantly by 16.98% and 30.65% lower compared with AMI non-diabetic patients and the control group (p<0.05), respectively. The FABP4 serum levels were significantly by 1.94 times higher in AMI patients without type 2 DM as compared to those of the control group (p<0.05). The serum concentrations of CTRP3 in non-diabetic AMI patients were significantly by 16.46% less as compared to those of the control group (p<0.05) (**Table 1**).

Analyzing the results of energy and adipokine metabolism components, correlations between blood glucose and adropin (r = -0.499, p <0.001), irisin (r = -0.495, p <0.001), FABP4 (r = -0.518, p < 0.001), CTRP3 (r = 0.435, p = 0.001); between HOMA-IR and adropin (r = -0.304, p <0.02), FABP4 (r = -0.429, p = 0.001) were revealed in AMI patients.

In AMI patients with type 2 DM, correlations were found between blood glucose and adropin (r = -0.504, p <0.001), irisin (r = -0.520, p = 0.03), FABP4 (r = 0.508, p = 0.007), CTRP3 (r = -0.546, p <0.001); between blood insulin and adropin (r = -0.361, p = 0.017), irisin (r = -0.396, p = 0.01), FABP4 (r = 0.501, p = 0.001), CTRP3 (r = -0.361, p = 0.018); between adropin and HOMA-IR (r = -0.508, p = 0.001), FABP4 (r = 0.516, p <0.001), CTRP3 (r = -0.436, p = 0.003).

In a study of 116 patients with type 2 DM, adropin serum levels were reduced, particularly in overweight/obese diabetic individuals. Adropin serum levels were negatively correlated with fasting plasma glucose [15]. In a study of 392 participants, diabetic patients

had lower serum adropin levels and higher degrees of coronary atherosclerosis angiographic severity. In addition, low adropin levels were identified as an independent predictor of clinically significant coronary atherosclerosis in both diabetic and non-diabetic patients [16]. Low adropin serum levels were found in AMI patients compared to those in patients with stable angina [17].

Patients with type 2 DM were revealed with lower irisin serum levels compared to healthy individuals [18]. Scientists suggest that irisin plays an essential role in glucose utilization and lipid metabolism, and therefore irisin is a promising pharmacological target for the treatment of diabetes and its complications [19]. Lower irisin serum levels were reported in AMI implying that irisin production may depend on myocardial blood supply [20].

It was demonstrated that high circulating FABP 4 concentrations predicted the incidence of metabolic syndrome and type 2 DM, both of which are associated with increased risks of cardiovascular disease (CVD) and mortality [21]. Significantly elevated FABP4 levels were demonstrated at early hours after onset of AMI and were greatly increased in out-of-hospital cardiac arrest survivors, probably due to rapid lipolytic release of FABP4 from epicardial fat tissue by adrenergic overdrive, which is a characteristic of acute CVD [22].

Patients with obesity and type 2 DM had significantly lower plasma CTRP3 concentrations in comparison with healthy subjects, and plasma levels of CTRP3 were strongly associated with glucose metabolism, chronic inflammation, and insulin resistance. Correlation analysis revealed significant negative correlations between plasma CTRP3 concentrations and fasting plasma glucose, 2-h plasma glucose, fasting insulin, HOMA-IR [23]. CTRP3 is currently regarded as a novel antiapoptotic, proangiogenic, and cardioprotective adipokine, the expression of which is significantly suppressed after myocardial infarction [24].

Conclusions

1. Decreased serum concentrations of adropin, irisin, CTRP3 and increased levels of FABP4 in both diabetic and non-diabetic patients with AMI indicate a violation of energy and adipokine metabolism.
2. The significant correlations between carbohydrate, energy and adipokine metabolism in AMI patients with or without type 2 DM have been revealed.

Perspectives of further research. The planned direction is to study adropin, irisin, FABP4, CTRP3 serum levels in the dynamics among AMI patients with or without type 2 DM.

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ВЗАЄМОЗВ'ЯЗОК МІЖ БІОМАРКЕРАМИ ВУГЛЕВОДНОГО, ЕНЕРГЕТИЧНОГО ТА АДИПОКІНОВОГО ОБМІНУ У ПАЦІЄНТІВ НА ГОСТРИЙ ІНФАРКТ МІОКАРДА ТА ЦУКРОВИЙ ДІАБЕТ 2 ТИПУ

Котелюх М. Ю.

Резюме. *Метою* дослідження було дослідити показники вуглеводного, енергетичного й адипокінового обміну у хворих на гострий інфаркт міокарда за умов наявності та відсутності цукрового діабету 2 типу.

Матеріал та методи. Було обстежено 134 пацієнтів на гострий інфаркт міокарда з елевацією сегменту ST за умов наявності та відсутністю цукрового діабету 2 типу віком 58,97±7,92 років у Державній установі «Національному інституті терапії імені Л.Т. Малої Національної академії медичних наук

України» та Харківській клінічній лікарні на залізничному транспорті №1 філії «Центр охорони здоров'я» Публічного акціонерного товариства «Українська залізниця». Першу групу склали 74 хворих на гострий інфаркт міокарда та цукровий діабет 2 типу. До другої групи (група порівняння) увійшло 60 пацієнтами на гострий інфаркт міокарда з відсутністю цукрового діабету 2 типу. Контрольну групу склали 20 практично здорових осіб. Визначення вмісту інсуліну, адропіну, ірісіну, білка, що зв'язує жирні кислоти 4 (FABP 4) та C1q/TNF-асоційованого білка (CTRP 3) у сироватці крові пацієнтів проведено імуноферментним методом.

Результати. Було встановлено, що рівні глюкози, інсуліну, індексу HOMA зростали у пацієнтів на гострий інфаркт міокарда за умов наявності та відсутності цукрового діабету 2 типу при зіставленні із групою контролю ($p<0,05$). Встановлено вірогідне зниження адропіну, ірисину та CTRP 3 та зростання FABP 4 у крові хворих на гострий інфаркт міокарда на фоні цукрового діабету 2 типу в порівнянні з групою контролю ($p<0,05$). Визначено значне зменшення концентрації адропіну та CTRP 3 у хворих на гострий інфаркт міокарда та цукровий діабет 2 типу порівняно із пацієнтами на гострий інфаркт міокарда із відсутністю цукрового діабету 2 типу ($p<0,05$). Визначено зворотній кореляційний зв'язок у хворих на гострий інфаркт міокарда між глюкозою крові та адропіном ($r=-0,499$, $p<0,001$), ірисином ($r=-0,495$, $p<0,001$), FABP 4 ($r=-0,518$, $p<0,001$); між HOMA та адропіном ($r=-0,304$, $p<0,02$), FABP 4 ($r=-0,429$, $p=0,001$) та прямий взаємозв'язок між глюкозою та CTRP 3 ($r=0,435$, $p=0,001$). У хворих на гострий інфаркт міокарда та цукровий діабет 2 типу виявлено зворотній взаємозв'язок між глюкозою крові та адропіном ($r=-0,504$, $p<0,001$), ірисином ($r=-0,520$, $p=0,03$), CTRP 3 ($r=-0,546$, $p<0,001$); між інсуліном крові та адропіном ($r=-0,361$, $p=0,017$), ірисином ($r=-0,396$, $p=0,01$), CTRP 3 ($r=-0,361$, $p=0,018$); між адропіном та індексом HOMA ($r=-0,508$, $p=0,001$), CTRP 3 ($r=-0,436$, $p=0,003$); прямий кореляційний зв'язок між глюкозою крові та FABP 4 ($r=0,508$, $p=0,007$); між інсуліном крові та FABP 4 ($r=0,501$, $p = 0,001$); між індексом HOMA та FABP 4 ($r=0,516$, $p<0,001$).

Висновки. Особливості змін вмісту адропіну, ірисину, FABP 4 та CTRP 3 доводять про дисбаланс в енергетичному та адипокіновому гомеостазі при гострому інфаркті міокарда за наявності та відсутності цукрового діабету 2 типу. Взаємозв'язок між адропіном, ірисином, FABP 4 та CTRP 3 може стверджувати про вплив цих маркерів на вуглеводний обмін.

Ключові слова: адипокіни, вуглеводний обмін, гострий інфаркт міокарда, енергетичний гомеостаз, цукровий діабет 2 типу.

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