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FEATURES OF DYSLIPOPROTEINEMIA IN TYPE 2 DIABETES MELLITUS PATIENTS WITH PRIOR MYOCARDIAL INFARCTION

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Relevance. In diabetes mellitus (DM), atherosclerotic plaques contain more fat, are more inflammatory and show a higher risk of thrombus formation than in individuals without diabetes. One of the significant factors in the development and progression of atherosclerosis in these patients is atherogenic dyslipidemia, which includes a wide range of disorders and often precedes the onset of diabetes for several years. Therefore, it is relevant to study the features of dyslipidemia in patients with diabetes after myocardial infarction (MI).

Objective: to study changes in blood lipid spectrum parameters, including Apolipoproteins (Apo A-1, Apo B) and lipoprotein (a), in postinfarction patients with type 2 DM.

Materials and methods. 119 patients (77 men and 42 women; mean age 61.09 ± 0.92 years) were examined, of which 42 were patients with coronary heart disease (CHD) who suffered from MI and type 2 diabetes (main group), 39 patients with a history of MI without concomitant diabetes (comparison group I) and 38 patients with type 2 diabetes without MI (comparison group II). The control group consisted of 30 healthy individuals, comparable in age and sex. Total cholesterol (TC), triglycerides (TG) and high-density lipoprotein cholesterol (HDL) were determined in venous serum by enzymatic colorimetric method. According to the formulas, the level of cholesterol in very low density lipoproteins (VLDL) was calculated; cholesterol in low-density lipoprotein (LDL), coefficient of atherogenicity (CA). The level of lipoprotein (a) – LP (a), Apo A-1 and Apo B was determined by immunoturbidimetry. Blood sampling in patients was performed on an empty stomach.

Results. In the main group, significantly higher rates of TC, TG, LDL, VLDL, AF, Apo B and the ratio of Apo B/Apo A-1, compared with non-diabetic postinfarction patients. At the same time, there was a significantly lower concentration of HDL, Apo A-1 and LP (a) – in patients with a history of diabetes mellitus. When comparing the indicators of patients in the main group with patients with isolated type 2 diabetes, there were significantly higher levels of LDL cholesterol and CA, as well as significantly lower concentrations of HDL cholesterol and apo A-1 in patients of the main group. The analysis of lipid metabolism in the comparison groups revealed a difference in the level of TG, LDL cholesterol and Apo B, which have higher levels in patients with isolated type 2 diabetes, and the level of LP (a) – significantly higher in postinfarction patients, in contrast to diabetics. The results indicate deeper disorders of lipid metabolism in post-infarction patients with diabetes than in non-diabetic post-infarction patients, which may be due to insulin resistance, hyperinsulinemia and hyperglycemia.

Conclusions. Dyslipoproteinemia in postinfarction patients with type 2 diabetes is characterized by a decrease in the content of antiatherogenic HDL cholesterol and its protein Apo A-1, moderate hypertriglyceridemia, increased levels of LDL cholesterol, VLDL cholesterol and Apo B, which causes higher values of cholesterol and increases AF. Elevations in lipid profile parameters such as TG, LDL cholesterol, and Apo B are more associated with diabetes, while higher concentrations of LP (a) are characteristic of postinfarction non-diabetic patients.

Key words: type 2 diabetes mellitus, postinfarction cardiosclerosis, dyslipoproteinemia.

Relevance. The population of patients with type 2 diabetes mellitus (DM) is growing steadily, doubling every ten years [6]. The risk of developing coronary heart disease (CHD) in type 2 diabetes is 2-3 times higher in male patients and 3-7 times higher in women [5]. Patients with type 2 diabetes have a 2-4 times higher risk of dying from coronary heart disease than non-diabetic patients [1].

Atherosclerosis, which is the morphological basis of coronary pathology, develops in patients with type 2 diabetes 10-15 years earlier than in the general population, progresses rapidly and more often leads to complications [3, 14]. It is among these patients that the risk of developing myocardial infarction (MI) is 3-5 times higher, depending on gender, higher than in the non-diabetic population [2]. It is among patients with type 2 diabetes mortality during the first year after MI is

15-34%, reaching 45% in the next 5 years, which is twice as high as in the general population. Despite significant advances in the treatment of coronary heart disease and its consequences, patients with type 2 diabetes remain the most vulnerable group, as the reduction in mortality among them is insignificant [15].

Such disappointing prognosis for patients with type 2 diabetes is associated primarily with the accelerated development of atherosclerosis of large subepicardial vessels and its rapid progression [1]. The results of studies have shown that in diabetes atherosclerotic plaques contain more fat, are more inflammatory and show a higher risk of thrombosis than in people without diabetes [17]. One of the important factors in the development and progression of atherosclerosis in these patients is atherogenic dyslipidemia, which includes a wide range

of disorders and often precedes the onset of diabetes for several years [8].

Therefore, based on these data, we decided to investigate the features of dyslipidemia in patients with type 2 diabetes who suffered MI.

Objective: to study changes in blood lipid spectrum parameters, including Apolipoproteins (Apo A-1, Apo B) and lipoprotein (a), in postinfarction patients with type 2 DM.

MATERIALS AND METHODS

119 patients (77 men and 42 women, mean age of patients – 61.09 ± 0.92 years) were examined, of which 42 were patients with coronary heart disease who had MI and had type 2 diabetes (main group), 39 patients with History of MI without concomitant diabetes (comparison group I) and 38 patients with type 2 diabetes without MI (comparison group II). The control group consisted of 30 healthy individuals, comparable in age and sex. The general clinical characteristics of the examined patients are given in table 1.

All patients included in the study were tested for total cholesterol (TC), triglycerides (TG) and high-density lipoprotein cholesterol (HDL) in the serum of venous blood by enzymatic colorimetric method using reagent kits «Human» (2000 Germany) bio (Germany,

2000), the results were expressed in mmol/l. The level of cholesterol in very low density lipoproteins (VLDL) was calculated by the formula: $TG \times 0.45$ (mmol/l); cholesterol in low-density lipoprotein (LDL) was calculated by the formula of Friedwald W.T. [12]: $LDL = TC - HDL - VLDL$. The coefficient of atherogenicity (CA) was calculated according to the generally accepted formula [7]: $CA = (LDL - HDL) / HDL$. The level of lipoprotein (a) – LP (a), Apo A-1 and Apo B was determined by immunoturbidimetry using the test system Roshe Diagnostics (Switzerland) on a Cobas 6000 analyzer (Switzerland). Units of measurement for LP (a) – mg/dl, and for Apo A-1 and Apo-B – g/l. Blood sampling in patients was performed on an empty stomach (last meal – more than 10 hours before blood sampling).

The research results are processed using the methods of variation statistics. Significance of differences when comparing the mean values was determined using Student's t-test (p). The difference was considered significant at $p < 0.05$. The values of the studied indicators are presented in the form of $M \pm m$, where M is the arithmetic mean, m is the standard error.

RESULTS AND DISCUSSION

The analysis of the obtained data in the main group of patients revealed significantly higher rates of TC

Table 1

General clinical characteristics of the examined patients

Indicator	Main group (n=42)	Isolated coronary heart disease: postinfarction cardiosclerosis (n=39)	Isolated type 2 diabetes mellitus (n=38)
Age, M $\pm$ m years	61,66 $\pm$ 1,78	61,17 $\pm$ 1,08	60,04 $\pm$ 1,16
Gender, n (%)	male	27 (64,3%)	24 (63,2%)
	female	15 (35,7%)	14 (36,8%)
Prescription of IM, M $\pm$ m years	4,93 $\pm$ 0,38	5,28 $\pm$ 0,55	-
Prescription of DM, M $\pm$ m years	8,34 $\pm$ 0,67	-	7,66 $\pm$ 0,71

Note: the difference between the groups is statistically insignificant ($p > 0,05$).

Table 2

Indicators of lipid metabolism in patients with CHD who underwent MI and patients with type 2 DM in comparison with patients with MI without DM and patients with history of type 2 DM without MI (M $\pm$ m)

Indicator	Main group (n=42)	Isolated coronary heart disease: postinfarction cardiosclerosis (n=39)	Isolated type 2 diabetes mellitus (n=38)
TC, mmol/l	5,99 $\pm$ 0,29 $\Delta\Delta\Delta$	4,92 $\pm$ 0,20	5,32 $\pm$ 0,22
TG, mmol/l	2,76 $\pm$ 0,28 $\Delta\Delta\Delta$	1,43 $\pm$ 0,15 $^{\#\#\#}$	2,57 $\pm$ 0,28
HDL, mmol/l	0,93 $\pm$ 0,04 $\Delta\Delta^{**}$	1,13 $\pm$ 0,06	1,07 $\pm$ 0,04
LDL, mmol/l	3,81 $\pm$ 0,27 Δ^*	3,13 $\pm$ 0,20	3,07 $\pm$ 0,19
VLDL, mmol/l	1,24 $\pm$ 0,13 Δ	0,64 $\pm$ 0,07 $^{\#\#\#}$	1,16 $\pm$ 0,12
CA	5,57 $\pm$ 0,36 $\Delta\Delta\Delta^{***}$	3,62 $\pm$ 0,33	4,05 $\pm$ 0,27
Apo A-1, g/l	1,29 $\pm$ 0,05 Δ^*	1,42 $\pm$ 0,03	1,40 $\pm$ 0,03
Apo B, g/l	1,24 $\pm$ 0,08 Δ	1,06 $\pm$ 0,04 $^{\#}$	1,23 $\pm$ 0,08
Apo B/Apo A-1	0,94 $\pm$ 0,06 $\Delta\Delta\Delta$	0,72 $\pm$ 0,03	0,81 $\pm$ 0,06
LP (a), mg/dl	23,29 $\pm$ 4,28 Δ	42,17 $\pm$ 7,31 $^{\#\#}$	18,10 $\pm$ 4,90

Notes: Δ – $p < 0,05$, $\Delta\Delta$ – $p < 0,01$, $\Delta\Delta\Delta$ – $p < 0,001$ compared with patients with MI without DM;

* – $p < 0,05$, ** – $p < 0,01$, *** – $p < 0,001$ in comparison with patients with DM without a history of MI;

– $p < 0,05$, ## – $p < 0,01$, ### – $p < 0,001$ compared with patients with DM without MI.

($p < 0.001$), TG ($p < 0.001$), LDLC ($p < 0.05$), LDLC ($p < 0.05$), CA ($p < 0.001$), Apo B ($p < 0.05$) and the ratio of Apo B/Apo A-1 ($p < 0.001$), compared with non-diabetic postinfarction patients. There was a significantly lower concentration of HDLC ($p < 0.01$), Apo A-1 ($p < 0.05$) and LP (a) – $p < 0.05$ in patients with a history of diabetes mellitus).

When comparing the studied indicators of patients of the main group with patients with isolated type 2 diabetes, significantly higher values of LDLC ($p < 0.05$) and CA ($p < 0.001$), as well as significantly lower concentrations of HDLC ($p < 0.01$) and apo A-1 ($p < 0.05$) in patients of the main group.

In the analysis of lipid metabolism of the comparison groups, the difference in the level of TG ($p < 0.001$), LDLC ($p < 0.001$) and Apo B ($p < 0.05$), which have higher levels in patients with isolated type 2 diabetes, is noteworthy. , and the level of LP (a) – $p < 0.01$, which is significantly higher in postinfarction patients, in contrast to diabetics.

Thus, the results indicate deeper disorders of lipid metabolism in postinfarction patients with diabetes than in non-diabetic postinfarction patients, which may be explained by insulin resistance, hyperinsulinemia and hyperglycemia, which are the pathogenetic basis of type 2 DM. It is known that insulin resistance [13] in the fat cell activates lipolysis, which leads to the release of large amounts of free fatty acids (FA) into the bloodstream. Further, there is an increase in the synthesis of TG and LDL due to the availability of their main substrate, as well as a decrease in the degradation of Apo B. Excessive production of LDL with increased secretion of TG and Apo B leads to the formation of small dense particles of LDL. This subtype of LDL plays an important role in atherogenesis, being more prone to oxidation under conditions of impaired antioxidant defense mechanisms in diabetics [10]. Both LDL and LDL residues and particles carry the Apo B molecule, so such dyslipidemia is characterized by an increase in serum Apo B concentration. Apo B levels are more closely associated with diabetes and insulin resistance than LDL and HDL because they are found in the most atherogenic particles of lipoproteins, and therefore can better predict cardiovascular events in such patients and are recognized as a valuable marker of CVD [20]. Regarding the antiatherogenic class of HDL, recent studies have shown that patients with diabetes not only have a quantitative decrease in these lipoproteins, but also may lose their protective function due to disruption of the structure of protein constituents on the background of prooxidant and proinflammatory phenotype [18]. Thus, HDL in diabetics is less effective in preventing LDL oxidation. Determining the level of Apo A-1 as the main protein of HDL, according to epidemiological and interventional studies, in addition to determining Apo B, allows you to more accurately predict cardiovascular risks than the assessment of conventional lipid parameters [20]. In

addition to insulin resistance, hyperglycemia also has a direct negative effect on lipid metabolism in these patients. It was found that the level of modified LDL increases significantly as a result of their glycosylation, which significantly enhances the atherogenic potential of lipoproteins [17].

Therefore, not only quantitative but also qualitative changes of lipids and lipoproteins are used, which increase their proatherogenic effects with corresponding clinical consequences. Thus, routine determination of general lipid profile parameters cannot provide complete information on atherogenic potential in a particular disease.

Today, hypertriglyceridemia in patients with diabetes is considered as a multifactorial process, with each injection of LCD from the periphery into the liver due to insulin resistance is only one component of this process. The decrease in HDL levels in these patients, which is often due to an increase in TG levels and the transition of TG to HDL (in exchange for cholesterol), is likely to be more complex, as the decrease in HDL at normal TG concentrations often changes [9].

Thus, not all pathogenetic mechanisms of diabetic dyslipidemia have been elucidated to date, and this is the subject of further study.

The results also show more pronounced atherogenic changes in the lipid spectrum of the blood in patients with type 2 DM who had a history of MI, compared with patients without MI. The results of the well-known British study UKPDS [19] showed that an increase in LDL cholesterol by 1 mmol/l is associated with an increase in the frequency of CVD endpoints by 57%, and an increase in HDL cholesterol by 0.1 mmol/l – with a decrease in CVD endpoints at 15%. Thus, our data confirm the fact that the increase in the degree of atherogenicity of lipid changes causes cardiovascular events. Therefore, it is important to timely and adequately influence the altered lipid profile in patients with type 2 DM to prevent complications of coronary heart disease and improve cardiovascular prognosis.

The difference in the comparison groups, namely between patients with isolated type 2 diabetes and postinfarction cardiosclerosis without diabetes, in terms of TG, LDL cholesterol and Apo B is explained by the pathogenetic mechanisms of dyslipidemia in diabetes, which were discussed above. As for LP (a), it is considered to be one of the most studied, but still a mysterious lipoprotein. Despite decades of research, the normal physiological role of LP (a) is still unclear. As for pathophysiology, there is more clarity. LP (a) can cause cardiovascular pathology due to proatherogenic LDL cholesterol and stimulate thrombosis due to the thrombogenic properties of Apo A, which are part of it. Thus, numerous studies suggest that high levels of LP (a) are an independent risk factor for atherogenesis and thrombogenesis. The amount of LP (a) is more than 90% determined genetically and depends mainly on the rate

of Apo A biosynthesis and, unlike most lipid risk factors, does not depend on age, sex, diet, living conditions and is very poorly corrected by medication [4, 11, 16].

Thus, going back to our data, we can assume that most patients who suffered from MI and did not have diabetes had a genetically determined high risk of acute coronary complications.

CONCLUSIONS

1. Dyslipoproteinemia in postinfarction patients with type 2 DM is characterized by a decrease in the content of antiatherogenic HDL cholesterol and its protein Apo A-I, moderate hypertriglyceridemia, increased levels of LDL cholesterol, VLDL cholesterol and Apo B, which causes higher values of cholesterol and increases CKD.
2. Elevations in lipid profile parameters such as TG, LDL cholesterol, and Apo B are more associated with the effects of diabetes, while higher concentrations of LP (a) are characteristic of postinfarction non-diabetic patients.

REFERENCES

1. Aleksandrov A.A., Bondarenko I.Z., Kukharensko S.S., Yadrinkhinskaya M.N., Martyanova I.I., Solyanik Yu.A., Drozdova E.N., Mayorov A.Yu. [Diabetes mellitus and coronary heart disease: search for a solution]. Diabetes mellitus. 2005; 3: 34-8. [in Russian]. URL: <https://cyberleninka.ru/article/n/saharnyy-diabet-i-ishemicheskaya-bolezn-serdtsa-poiski-resheniya>
2. Alexandrov A.A. [Myocardial infarction and diabetes mellitus: «Munich Agreement»]. Diseases of the heart and blood vessels. 2007; 2(2): 4-11. [in Russian]. URL: http://old.consilium-medicum.com/media/bss/07_02/4.shtml
3. Alexandrov A.A. [Cardiovascular complications and modern algorithm of antihyperglycemic therapy: «Florentine perspective»]. Russian medical journal. 2010; 14: 879-84. [in Russian]. URL: https://www.rmj.ru/articles/endokrinologiya/Serdechnosudistye_oslozhneniya_i_sovremennyy_algoritm_saharnonighayushey_terapii_Florentiyskaya_perspektiva/
4. Vel'kov V.V. [How to prevent heart attacks and strokes: predictors]. Polyclinic. Laboratory diagnostics. 2008; 3: 56-9. [in Russian]. URL: [http://www.poliklin.ru/articles/08_03/16_\(58-61\).pdf](http://www.poliklin.ru/articles/08_03/16_(58-61).pdf)
5. Dedov I.I., Melnichenko G.A. [Endocrinology: national guidelines]. Moscow: GEOTAR-Media, 2008. 1072 p. [in Russian]. URL: <https://www.rosmedlib.ru/book/ISBN9785970450833.html>
6. Dedov I.I., Shestakova M.V., Mayorov A.Yu. [Algorithms for specialized medical care for patients with diabetes mellitus]. Moscow: Media Sphere, 2007. 112 p. [in Russian]. URL: <http://webmed.irkutsk.ru/doc/pdf/algosd.pdf>
7. Klimov A.N., Nikulcheva N.G. [Lipids, lipoproteins and atherosclerosis]. St. Petersburg: Peter. Press, 1995. 304 p. [in Russian]. URL: <https://www.libex.ru/detail/book703548.html>
8. Mitchenko O.I., Lutai M.I. Dyslipidemias: diagnosis, prevention and treatment. Methodical recommendations of the Association of Cardiologists of Ukraine. Kyiv, 2011. 48 p. [in Ukrainian]. URL: http://strazhesko.org.ua/upload/2014/02/20/guidelines_dyslipid_2011_new.pdf
9. Brunzell J.D., Hokanson J.E. Dyslipidemia of central obesity and insulin resistance. Diabetes Care. 1999; 22 (Suppl. 3): C10-C13. PMID: 10189557.
10. Collins R., Armitage J., Parish S., Sleight P., Peto R. MRC/BHF Heart Protection Study of cholesterol-lowering with simvastatin in 5963 people with diabetes: a randomized placebo-controlled trial. Lancet. 2003; 361: 2005-16. DOI: [https://doi.org/10.1016/s0140-6736\(03\)13636-7](https://doi.org/10.1016/s0140-6736(03)13636-7).
11. Erqou S., Thompson A., Di Angelantonio E. [et al.] Apolipoprotein (a) isoforms and the risk of vascular disease: systematic review of 40 studies involving 58,000 participants. J. Am. Coll. Cardiol. 2010; 55: 2160-7. DOI: <https://doi.org/10.1016/j.jacc.2009.10.080>
12. Friedewald W.T., Levy R.I., Fredricson D.S. Estimation of the concentration of low density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin. Chem. 1972; 18(6): 499-502. PMID: 4337382
13. Ginsberg H.N., Huang L.S. The insulin resistance syndrome: impact on lipoprotein metabolism and atherothrombosis. J. Cardiovasc. Risk. 2000 Oct; 7(5): 325-31. DOI: <https://doi.org/10.1177/204748730000700505>
14. Hurst R.T., Lee R.W. Increased incidence of coronary atherosclerosis in type 2 diabetes mellitus: mechanisms and management. Ann. Intern. Med. 2003 Nov 18; 139(10): 824-34. DOI: <https://doi.org/10.7326/0003-4819-139-10-200311180-00010>
15. Malmberg K., Ryden L., Wedel H., Birkeland K., Bootsma A., Dickstein K., Efendic S., Fisher M., Hamsten A., Herlitz J., Hildebrandt P., MacLeod K., Laakso M., Torp-Pedersen C., Waldenström A., DIGAMI 2 Investigators. Intense metabolic control by means of insulin in patients with diabetes mellitus and acute myocardial infarction (DIGAMI 2): effects on mortality and morbidity. Eur. Heart J. 2005 Apr; 26(7): 650-61. DOI: <https://doi.org/10.1093/eurheartj/ehi199>
16. Nordestgaard BG, Chapman MJ, Ray K., Borén J., Andreotti F., Watts G.F., Ginsberg H., Amarencu P., Catapano A., Descamps O.S., Fisher E., Kovanen P.T., Kuivenhoven J.A., Lesnik Ph., Masana L., Reiner Z., Taskinen M.-R., Tokgözoğlu L., Tybjaerg-Hansen A., European Atherosclerosis Society Consensus Panel. Lipoprotein (a) as a cardiovascular risk factor: current status. Eur. Heart J. 2010 Dec; 31(23): 2844-53. DOI: <https://doi.org/10.1093/eurheartj/ehq386>
17. Ryden L., Grant P.J., Anker S.D. [et al.] ESC Guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed in collaboration with the EASD: the Task Force on diabetes, pre-diabetes, and cardiovascular

- diseases of the European Society of Cardiology (ESC) and developed in collaboration with the European Association for the Study of Diabetes (EASD). *European Heart J.* 2013 Oct; 34(39): 3035-87. DOI: <https://doi.org/10.1093/eurheartj/eh108>
18. Sorrentino SA, Besler C, Rohrer L., Meyer M., Heinrich K., Bahlmann F.H., Mueller M., Horváth T., Doerries C., Heinemann M., Flemmer S., Markowski A., Manes C., Bahr M.J., Haller H., Eckardstein A., Drexler H., Landmesser U. Endothelial-vasoprotective effects of high-density lipoprotein are impaired in patients with type 2 diabetes mellitus but are improved after extended-release niacin therapy. *Circulation.* 2010 Jan 5; 121(1): 110-22. DOI: <https://doi.org/10.1161/circulationaha.108.836346>
 19. United Kingdom Prospective Diabetes Study: Plasma lipids and lipoproteins at diagnosis of NIDDM by age and sex (UKPDS 27). *Diabetes Care.* 1997 Nov; 20(11): 1683-7. DOI: <https://doi.org/10.2337/diacare.20.11.1683>
 20. Vijayaraghavan K. Treatment of dyslipidemia in patients with type 2 diabetes. *Lipids in Health and Disease.* 2010; 9: 144-156. DOI: <https://dx.doi.org/10.1186%2F1476-511X-9-144>

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ОСОБЛИВОСТІ ДИСЛІПОПРОТЕЇНЕМІЇ У ХВОРИХ НА ЦУКРОВИЙ ДІАБЕТ 2 ТИПУ, ЯКІ ПЕРЕНЕСЛИ ІНФАРКТ МІОКАРДУ

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Актуальність. При цукровому діабеті (ЦД) атеросклеротичні бляшки містять більше жиру, в більшому ступені запаленозмінені і демонструють вищий ризик тромбоутворення, ніж в осіб без ЦД. Одним з вагомих факторів розвитку та прогресування атеросклерозу у цих хворих є атерогенна дисліпідемія, яка включає широкий спектр порушень і часто передусе виникненню ЦД на декілька років. Тому є актуальним дослідження особливості дисліпідемії у хворих на ЦД, які перенесли інфаркт міокарда (ІМ).

Мета: вивчити зміни показників ліпідного спектру крові, включаючи аполіпопротеїни (Апо А-1, Апо В) та ліпопротеїн (а), у постінфарктних хворих з ЦД 2 типу

Матеріали та методи. Обстежено 119 хворих (77 чоловіків та 42 жінки; середній вік $61,09 \pm 0,92$ р.), з яких 42 – пацієнти з ішемічною хворобою серця (ІХС), які перенесли ІМ та хворіють на ЦД 2 типу (основна група), 39 хворих з ІМ в анамнезі без супутнього ЦД (І група порівняння) та 38 пацієнтів з ЦД 2 типу без перенесеного ІМ (ІІ група порівняння). Контрольну групу склали 30 практично здорових осіб, зіставних за віком та статтю. Визначали загальний холестерин (ЗХС), тригліцериди (ТГ) та холестерин ліпопротеїдів високої щільності (ХС ЛПВЩ) в сироватці венозної крові ферментативним колориметричним методом. За формулами обчислювали рівень холестерину в ліпопротеїнах дуже низької щільності (ХС ЛПДНЩ); холестерин в ліпопротеїнах низької щільності (ХС ЛПНЩ), коефіцієнт атерогенності (КА). Рівень ліпопротеїну (а) – ЛП (а), Апо А-1 та Апо В визначали методом імунотурбідиметрії. Забір крові у пацієнтів здійснювався натщесерце.

Результати. В основній групі виявлено достовірно вищі показники ЗХС, ТГ, ХС ЛПНЩ, ХС ЛПДНЩ, КА, Апо В та співвідношення Апо В/Апо А-1, порівняно з недіабетичними постінфарктними пацієнтами. При цьому відмічалась достовірно нижча концентрація ХС ЛПВЩ, Апо А-1 та ЛП (а) – у хворих на ЦД з ІМ в анамнезі. При співставленні показників пацієнтів основної групи з хворими на ізольований ЦД 2 типу спостерігалось достовірно вищі показники ХС ЛПНЩ та КА, а також достовірно нижчі концентрації ХС ЛПВЩ і апо А-1 у хворих основної групи. При аналізі показників ліпідного обміну груп порівняння виявлена відмінність за рівнем ТГ, ХС ЛПДНЩ і Апо В, що мають вищі рівні у пацієнтів з ізольованим ЦД 2 типу, та рівнем ЛП (а) – що достовірно вищий у постінфарктних хворих, на відміну від діабетичних. Отримані результати свідчать про більш глибокі порушення ліпідного обміну у постінфарктних хворих за умови наявності ЦД, ніж у недіабетичних постінфарктних пацієнтів, що може пояснюватись інсулінорезистентністю, гіперінсулінемією та гіперглікемією.

Висновки. Дисліпопротеїнемія у постінфарктних хворих з ЦД 2 типу характеризується зниженням вмісту антиатерогенного ХС ЛПВЩ та його білка Апо А-1, помірною гіпертригліцеридемією, збільшенням рівня ХС ЛПНЩ, ХС ЛПДНЩ та Апо В, що зумовлює вищі значення ЗХС та збільшує КА. Підвищення таких показників ліпідогамі, як ТГ, ХС ЛПДНЩ та Апо В, в більшому ступені, пов'язані з впливом ЦД, в той час як вищі концентрації ЛП (а) характерні для постінфарктних недіабетичних хворих.

Ключові слова: цукровий діабет 2 типу, постінфарктний кардіосклероз, дисліпопротеїнемія.

ОСОБЕННОСТИ ДИСЛИПОПРОТЕИНЕМИЯ У БОЛЬНЫХ САХАРНЫМ ДИАБЕТОМ 2 ТИПА, ПЕРЕНЕСШИХ ИНФАРКТ МИОКАРДА

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Актуальность. При сахарном диабете (СД) атеросклеротические бляшки содержат больше жира, в большей степени воспалительно изменены и демонстрируют более высокий риск тромбообразования, чем у лиц без СД. Одним из весомых факторов развития и прогрессирования атеросклероза у этих больных является атерогенная дислипидемия, которая включает широкий спектр нарушений и часто на несколько лет предшествует возникновению СД. Поэтому актуально исследование особенности дислипидемии у больных СД, перенесших инфаркт миокарда (ИМ).

Цель: изучить изменения показателей липидного спектра крови, включая аполипопротеины (Апо А-1, Апо В) и липопротеин (а), у постинфарктных больных с СД 2 типа

Материалы и методы. Обследовано 119 больных (77 мужчин и 42 женщины, средний возраст $61,09 \pm 0,92$ г.), из которых 42 – пациенты с ишемической болезнью сердца (ИБС), перенесших ИМ и страдающих СД 2 типа (основная группа), 39 больных с ИМ в анамнезе без сопутствующего СД (I группа сравнения) и 38 пациентов с СД 2 типа без перенесенного ИМ (II группа сравнения). Контрольную группу составили 30 практически здоровых лиц, сопоставимых по возрасту и полу. Определяли общий холестерин (ОХС), триглицериды (ТГ) и холестерин липопротеидов высокой плотности (ХС ЛПВП) в сыворотке венозной крови ферментативным колориметрическим методом. По формулам вычисляли уровень холестерина в липопротеинах очень низкой плотности (ХС ЛПОНП), холестерин в липопротеинах низкой плотности (ХС ЛПНП), коэффициент атерогенности (КА). Уровень липопротеина (а) – ЛП (а), Апо А-1 и Апо В определяли методом имунотурбидиметрии. Забор крови у пациентов осуществлялся натощак.

Результаты. В основной группе выявлены достоверно более высокие показатели ОХС, ТГ, ХС ЛПНП, ХС ЛПОНП, КА, Апо В и соотношение Апо В / Апо А-1 по сравнению с недиабетическими постинфарктными пациентами. При этом отмечалась достоверно более низкая концентрация ХС ЛПВП, Апо А-1 и ЛП (а) – у больных СД с ИМ в анамнезе. При сопоставлении показателей пациентов основной группы и больных только с СД 2 типа наблюдались достоверно более высокие показатели ХС ЛПНП и КА, а также достоверно более низкие концентрации ХС ЛПВП и Апо А-1 у больных основной группы. При анализе показателей липидного обмена групп сравнения выявлены отличия по уровню ТГ, ХС ЛПОНП и Апо В – более высокие уровни их у пациентов с изолированным СД 2 типа; и уровнем ЛП (а) – достоверно выше у постинфарктных больных, в отличие от диабетических. Полученные результаты свидетельствуют о более глубоких нарушениях липидного обмена у постинфарктных больных при наличии СД, чем у недиабетических постинфарктных пациентов, что может объясняться инсулинорезистентностью, гиперинсулинемией и гипергликемией.

Выводы. Дислипидемия у постинфарктных больных с СД 2 типа характеризуется снижением содержания антиатерогенного ХС ЛПВП и его белка Апо А-1, умеренной гипертриглицеридемией, повышением уровня ХС ЛПНП, ХС ЛПОНП и Апо В, что приводит к более высоким значениям ОХС и увеличивает КА. Повышение таких показателей липидограммы, как ТГ, ХС ЛПОНП и Апо В, в большей степени, связаны с влиянием СД, в то время как высокие концентрации ЛП (а) характерны для постинфарктных недиабетических больных.

Ключевые слова: сахарный диабет 2 типа, постинфарктный кардиосклероз, дислипидемия.