Chronic pancreatitis in patients with coronary heart disease: a lipid spectrum of blood, a possibility of correction

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In the course of chronic pancreatitis (CP) and atherosclerosis, metabolic disorders are essential factors in pathogenesis. It is they who act as the unifying mechanisms of CP and coronary heart disease (CHD). Such mechanisms include chronic obstructive oxidative stress, systemic inflammatory response to damage, insulin resistance, hyperlipidemia, hypertriglyceridemia, especially postprandial [14]. Metabolic processes are closely interconnected. For example, in the metabolism of glucose and lipids (including in visceral adipose tissue), regulation of coagulation and inflammatory processes, the key role belongs to the liver and pancreas (pancreas). The liver becomes both a target organ and an important source of some pathogenetic processes that form insulin resistance and lipid metabolism disorders due to damage to the vascular and liver tissue, pancreas. Perhaps the intersection of mechanisms at the level of these organs and inflammatory processes in visceral adipose tissue (which secretes proinflammatory adipocyne) may be significant. In this case, the liver initiates the transcription of a number of proinflammatory genes, which enhances chronic systemic inflammation in both fatty tissue and in the software [3]. It is believed that the early outcome of inflammatory processes in the adipose tissue is also insulin resistance. Systemic insulin resistance reduces the absorption of glucose by hepatocytes, adipocytes, muscle tissue, glycogen synthesis and triglycerides, glycolysis activation, lipolysis, neoglycosis. Increases the circulation and oxidation of fatty acids, causing tissue damage (including hepatocytes, pancreatitis) due to lipotoxicity. Infringement of lipid metabolism is not compensated by hyperinsulinemia, which leads to accelerated development of atherosclerosis.

An important factor in understanding the pathogenesis of CP and the complications of atherosclerosis is the fact that high levels of triglycerides (TG) contribute to the formation of modified low density lipoprotein (LDL) and high density lipoprotein (HDL) rich in thyroid glands, a shift in carbohydrate metabolism and activation of thrombosis [2]. The combination of

hypertriglyceridemia with a decrease in HDL is seen as a predictor of CP, diabetes mellitus (such a mechanism is more significant in women than in men). The leading cause of atherosclerosis in patients with normal levels of lipids in the onset (it is possible and for CP) may be postprandial lipemia. A high concentration of lipids in the blood after eating even is a stronger predictor of cardiovascular disease than the level of triglycerides in the onset [20].

Consequently, hypertriglyceridemia acts as a common mechanism for the formation of fatty infiltration of pancreas (which may contribute to the subsequent formation of steato pancreatitis, non-alcoholic fatty illness with non-alcoholic liver disease, or independently) and the development of vascular atherosclerosis for the comorbid flow of CP with CHD, resulting in some cases before the onset DM type 2, to acute cardiovascular events. [13]

Obstruction of blood vessels in the blood vessels, fatty infiltration of acinar cells, the emergence of a large number of cytotoxic free fatty acids become important in the pathogenesis of hyperlipidemic pancreatitis [11]. They are formed as a result of intense hydrolysis of triglycerides under the influence of lipase, endothelial lipoprotein lipase, which transform it into glycerol and fatty acids. But in the postprandial period, especially in the formation of atherosclerotic processes, the activity of endothelial lipoprotein lipase decreases, in the blood accumulate lipoproteins enriched with triglycerides. It is important that competition for the activity of endothelial lipoprotein lipase increases with hypertriglyceridemia onset. Thus, the vascular endothelium is constantly in contact with the attenuators of atherogenic lipoproteins (lobules that are poor in TG, rich in cholesterol), free fatty acids, which increases the oxidation of proteins and lipid peroxidation, damages the structure of the lipid layer of cell membranes, induces an inflammatory, low-intensity generalized reaction of the immune system [22]. Overloading of cholesterol pancreatic cells can cause mitochondrial dysfunction and start the process of apoptosis [18], which ultimately leads to fibrosis, the more so because of dyslipidemia, fibrosis can be formed through lipoidosis, steatosis [12].

All 5 classes of lipoprotein have a similar structure and components (triglycerides, non-esterified cholesterol, cholesterol esters, phospholipids and apoptoids). Apolipoproteins are the protein components of the lipoprotein and are classified by the letters of the alphabet: A, B, C. More than 20 apolipoproteins are

isolated, their capabilities, mutations in the structural and regulatory genes are determined.

Cholesterol in the blood plasma has two components: exogenous cholesterol, which comes from food, and endogenous — formed in the body. The proportion of exogenous cholesterol is much lower and is approximately 25-30%, which limits the correction of hypercholesterolemia due to diet [5].

It should be noted that in the body cholesterol is not in a free state. It is bound to proteins and is included into lipoproteins transporting cholesterol from the liver to peripheral cells (very low density lipoprotein, LPDH, and LDL) and in the opposite direction, HDL. Cholesterol and triglycerides, which are part of the food, enter into the intestine, where they are absorbed and become a component of large lipoprotein particles — chylomicrons. They account for 90% of TG, 5% are cholesterol, 4% are phospholipids and 1% are proteins. The main function of the chylomicrons is the transport of TG and cholesterol through the lymph to the blood plasma. In capillaries of fatty and muscular tissue, chylomicrons under the influence of lipoprotein lipase hydrolyze with the release of free fatty acids and monoglycerides and are converted into particles that are poor in TG and rich in cholesterol (repairing particles). The more such particles are transformed into muscle under active physical activity or in the cells of the fatty (predominantly abdominal) tissue, the less they will enter the liver and become a material for the formation of atherogenic classes of lipoproteins. The residues of chylomicrons due to the presence of apo-E in their composition are determined by the corresponding liver receptors, admire and enter into it [1]. Consequently, with the comorbidity of CP with CHD, one can not ignore the functional state of the liver, whose activity depends on carbohydrate, fatty metabolism and endocrine status. The further transport of cholesterol is carried out in the LPDNCH. LDL-cells are formed in the liver and consist mainly of TG (65%). They are the main transport form of endogenous triglycerides, in contrast to the chylomicrons that carry exogenous TG. In capillaries of adipose and muscular tissue, LPDHC under the influence of lipoprotein lipase hydrolyzes with the formation of TG and the cleavage of fatty acids. As a result, LDL-content decreases the TG content and increases the specific gravity of esterified cholesterol, which contributes to their conversion to intermediate density lipoprotein (HDL). They partially catalyze in the liver by receptor-mediated endocytosis and partially (due to liver lipase) lose a large

proportion of TG and become LDL-cells. Low density lipoproteins contain the largest amount of cholesterol (70%) and they are given a leading role in the transport of cholesterol from the liver to peripheral cells. Therefore, this class is considered as the most atherogenic, and the level of LDL cholesterol determines the risk of developing atherosclerosis and evaluates the effectiveness of hypolipidemic therapy. [3]. The introduction of cholesterol into liver cells is ensured by the presence of specific receptors on the membrane of cells that recognize the proteins that are part of the LDL apo-b and apo-e). After binding to the LDL receptors through endocytosis, they enter the cells where the trapped lipoprotein particles in the lysosomal apparatus are degraded. In cholesterol lysosomes, free cholesterol is released, which by the feedback mechanism is an inhibitor of endogenous cholesterol synthesis. Reverse transport from the peripheral cells to the liver is carried out by HDL, the composition of which is significantly different from the low density lipoprotein (precisely the composition of aphids). HDLs contain protein particles (Apo-A-I and apo-A-II), 30% phospholipids, 18% cholesterol. The activity of reverse cholesterol transport depends on the number of HDL, receptors to Apo-A-I and their identification. "Charged" HDL cholesterol is considered as an independent risk factor for atherosclerosis, and it is the ratio of HDL/HDL cholesterol HDL that determines the risk of developing the disease. [13]. A known fact is the property of LDL-lowering of insulin production by beta-cells of pancreas (as a manifestation of lipotoxicity) and the property of apolipoprotein D in the action of contrinsular, competing with insulin for specific receptors. Based on the data obtained, the combination of CHD with CP is a complicated process due to some links of the common pathogenesis (dyslipidemia, especially hypertriglyceridemia, insulin resistance, persistence of chronic generalized low-intensity inflammatory reaction, endothelial dysfunction), which predetermines the clinical picture, the course and prognosis of diseases, and presents clinical interest, especially in chronic heart failure, which is a prognostically unfavorable complication of diseases of the cardiovascular system, and for comorbidity and with CP — and for its course. The medication correction of lipid metabolism disorders to reduce the risk of cardiovascular diseases and their complications (taking into account the results of large studies) is reduced to the appointment of statins in order to reduce the level of LDL cholesterol in the blood [9, 10]. But regardless of the aggressive reduction in

LDL cholesterol achieved with the help of statins, 66.7% of patients still undergo repeated cardiovascular events (regardless of the dosage of statins). They also arise irrespective of the improvement of lipidogram rates achieved on statin therapy, since this group of pharmacological agents is not affected by TG and remnant lipoprotein [17]. This group of drugs does not significantly affect atherosclerotic changes in the arterial wall, the rate of delay in it, lipoproteins, the progression of endothelial dysfunction, reduction of cardiovascular mortality. A significant difference is noted only with regard to the reduction of the frequency of hospitalizations for cardiovascular events. In addition, when they are used, the hepatic enzymes and rhabdomyolysis effects are elevated, the difference in clinical efficacy and safety of use for various statins is detected. Given the risk of myopathy and rhabdomyolysis, the FDA recommended caution in 2011 administer simvastatin at a dose of 80 mg per day. In addition, in recent years, the issue of the influence of statins on carbohydrate metabolism has been actively discussed, which had not been previously disturbed in patients. In 2010, K. Koh et al. Was published. [19] where it was shown that atorvastatin 10, 20, 40, and 80 mg dose significantly reduced LDL cholesterol (39%, 47%, 52% and 56% respectively), apolipoprotein B levels (33%, 37%, 42% and 46% respectively) after 2 months of treatment compared to baseline (for all p <0.01). Nevertheless, atorvastatin at a dose of 10, 20, 40 and 80 mg significantly increased the level of insulin oncaps (averaging 25%, 42%, 31% and 45%, respectively) and the level of glycosylated hemoglobin (HbA1c) by 2%, 5 %, 5% and 5%, in comparison with the upward data (for all p <0,05). The drug at a dose of 10, 20, 40 and 80 mg reduced the sensitivity of tissues to insulin (1%, 3%, 3% and 4%, respectively). The results of the Canadian population study on the risk of diabetes in patients who were first treated with statins from August 1997 to March 31, 2010 (authors — AA Carter et al.) Were published in May 2013 in the British Medical Journal (2013, Vol 346), showed that the risk of diabetes arises when using atorvastatin (1.22; 95% CI 1.15-1.29), rosuvastatin (1.18; CI 1.10-1.26) and simvastatin (1.10; CI 1.04-1.17). The absolute risk of developing diabetes mellitus was 31 and 34 cases per 10,000 patients, which was lower for simvastatin (26 cases per 10,000 patients per year) when compared with pravastatin (23 cases). This allowed us to conclude that there is a high risk of diabetes in the use of statins in the treatment of dyslipidemias (especially atorvastatin and simvastatin), the need to carefully apply to their

appointment to the elderly, women with high risk of developing type 2 diabetes. It complicates the treatment of such patients for a long period of time (1-2 years), which can provide a clinical effect against the progression of atherosclerotic complications [8]. The results of the survey showed that the reasons for the refusal of patients from treatment with statins is a negative effect on the liver function, which is manifested by an increase in transaminase rates in 2-2.5 times; the inability to take alcohol; high price; unwillingness to use preparations of synthetic origin; the lack of achievement of the target indicators of the lipid profile, even with the full implementation of the drug regimen [6]. Under such conditions, it is probably strategically viable to find new phytopreparations that would be able to influence, in parallel with statins, the correction of hyperlipidemia, hypertriglyceridemia both for the treatment and prevention of cardiovascular diseases, their progression and the appearance of complications, including comorbidity diseases. At the present stage, preparations of plant origin occupy an appropriate niche among medicamentous means. This is explained, first of all, with the safety of use, a positive effect on the tone of the cardiovascular system. Together with the correction of modifying risk factors for lifestyle phytopreparations can act in accordance with the effect of drug treatment. In Ukraine, a new phytopreparate, which improves the lipid spectrum of blood, — polikozanol. This is a patented product that contains a natural mixture of higher primary aliphatic alcohols, isolated and purified from the waxy weight of sugar cane (Saccharum officinarum, L.). It consists of 1-octacosanol, 1-dithricontanol, 1-triacontanol, 1-tetracazanol, 1-tetrathiacontanol, 1-hexaconazole, 1-heptacosanol and 1-nonocosinol. The content of each component in polycozalol in each batch is standardized in a narrow range and is stable in storage. The tablet is coated with 10 mg of polycosalone. By appointment, it is an adjunct to nutrition and healthy lifestyle recommendations for lowering total cholesterol and LDL cholesterol levels. Important for use in the comorbidity of CP with CHD is its effect with type II hypercholesterolemia, including the subtype IIa (characterized by elevated HR and LDL cholesterol) and subtype IIb (combined hypercholesterolemia, which is characterized by elevated HR, LDL, and TG) accompanying CHD and CP. Polycosanol is effective, safe and easily tolerated in patients with type II hypercholesterolaemia and in patients with secondary hypercholesterolemia, which occurs as a result of diabetes or nephrotic syndrome (considered as risk factors for

cardiovascular events). The drug can also be used as an alternative to aspirin (if necessary), that is, it can be used as an antiplatelet agent. It affects the synthesis of prostaglandines, reduces the level of thromboxane A2, increasing the level of prostacyclin, thereby preventing aggregation processes. It is recommended to start treatment at a dose of 10 mg once a day during the evening, as bosynthesis of cholesterol is activated at night. If necessary, the dose can be doubled in 2 months to 20 mg/day (depending on the level of cholesterol, which is checked every 8 weeks). Course of treatment for 6 months and further. Treatment with a dose of 10 mg reduces LDL cholesterol levels by 20-25% during the first 6 months, with a dosage of 20 mg — by 25-30%, significantly improves the ratio of HDL/HDL/HDL to HDL. This confirms the dose-dependent effect of the drug [20]. Effectiveness has been studied in about 30,000 patients, which is a sufficient evidence base for the use of polycosanol. Clinical trials included short-term, long-term, randomized, placebo-controlled, and comparative trials of statin efficacy (lovastatin, pravastatin and simvastatin), fibrates (bezafibrate and gemfibrozil), acipigomas and tuberculosis. The results showed that 10 mg of polycosanol is equivalent to 20 mg lovastatin, 10 mg pravastatin and 10 mg simvastatin. At the same time, the values of HDL cholesterol increased by 17%, and the number of side effects was somewhat lower than that of statins [15]. Less hepatotoxicity was observed, whereas lovastatin and pravastatin significantly increased the level of transaminases and creatine phosphokinase. Patients with type II hypercholesterolemia [21] and concomitant CD-2 type polycosanol compared to simvastatin (polycosanol and simvastatin administered at a dose of 10 mg/day for 8 weeks) significantly increased the incidence of HDL in the blood and did not cause side effects. In a study conducted by AA Sergienko and sang. (2013), it was found that the effectiveness of polikozanol (20 mg/day for 3 months) in the treatment of dyslipoproteinemia, correction of insulin resistance in patients with type-2 diabetes is comparable to that of the dose of simvastatin. Consequently, polycosanol has a dose-dependent effect on the level of HDL-C, whereas triglycerides are reduced regardless of the dosage of the drug. This fact deserves attention in terms of the appropriateness of the concomitant administration of polycosalone with statins (since they do not affect the TG). There is a difference in the mechanism that reduces the levels of CF, LDL cholesterol. Polycosanol suppresses their synthesis at the time between the formation of acetate and mevalonate, not suppressing

HMG-CoA reductase, significantly increasing receptor-dependent process of LDL, which is confirmed by the absorption of their hepatocytes and stimulation of their catabolism. Polizoxanol not only reduces cholesterol levels in the blood, but also in such tissues as liver, heart and adipose tissue, with antioxidant effects. In addition, it prevents thickening of intima vessels, proliferation of smooth myocytes and the formation of atherosclerotic changes in the vascular wall and thrombosis. Based on the above, it was interesting to determine the state of indicators of lipidogram in patients with comorbidity of chronic CH with CHD and the role of polycosanol ("FitoStatin") in the correction of dyslipidemia in patients with CH with IHD.

The aim of study is to investigate changes in lipidograms in patients with IHD with CHD, including the dynamics of treatment with polycosanol.

Materials and methods

The studies were conducted in 22 patients who were divided into appropriate groups. The group of practically healthy (POPs) was 10 people (men — 5 (50%), women — 5 (50%). The first group consisted of 10 patients with CS with dyslipidemia (men — 7 (70.0%), women — 3 (30%), the second group consisted of 12 patients with CHD (male — 6 (54.5%), female — 6 (45.5%)).The age of the patients varied from 31 to 69. Examined randomized by age, sex and duration of illness.

The diagnosis of CP was established in accordance with the clinical protocol of the Order of the Ministry of Health of Ukraine dated June 13, 2005, No. 271. "Clinical protocol for the provision of medical care for patients with chronic pancreatitis". The diagnosis of CHD was exposed in accordance with the Order of the Ministry of Health of Ukraine No. 436 of 03.07.2006 "On approval of medical treatment protocols in the specialty" Cardiology. "The rationale for the clinical diagnosis was based on the results of clinical and instrumental examination, general clinical trials, electrocardiography, echocardiography, X-ray examination of the chest organs.

Inclusion criteria were characteristic of chronic obstructive pulmonary tuberculosis, alcohol abuse, abdominal pain syndrome, periodic dyspeptic disorders — nausea, feeling of discomfort, severity, dislocation in the upper abdomen, epigastric area, chronic diarrhea or constipation, alternating diarrhea with constipation, etc.; confirmation of morphological changes in the structure of the software, established on the basis of ultrasonographic research, as well as

violation of the external secretory function of the software; presence of informed consent of the patient to participate in the study. Criteria for inclusion in the study of patients with coronary artery disease, stable angina pectoris I-II FK, CHF IIF-B, FC II-III (NYHA) were: offset by CHF I or II FC; absence of angina attacks for 3 months; regular administration of angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers, β-blockers, statins and antiplatelet drugs for 3 months; sinus arrhythmias, violation of myocardial excitability; signed informed consent of the patient.

The exclusion criteria were as follows: patients with cancer, including cancer; persons who have suffered acute pancreatitis, exacerbation/decompensation of a chronic disease or surgical intervention during the last 4 weeks; persons who have been abusing drugs in history; refusal of the patient to participate in the study. The exclusion criteria were also CHF non-ischemic etiology (cardiomyopathy, rheumatism, pulmonary heart, etc.); Angina pectoris requiring nitroglycerin or prolonged nitrates; suffered acute myocardial infarction over the past 3 months; other severe concomitant diseases of the cardiovascular system in a state of decompensation; stroke or transient ischemic attack of less than 6 months, bronchial asthma, oncological and chronic infectious diseases, chronic kidney disease and/or glomerular filtration rate less than 30 ml/min, insulin dependent diabetes mellitus, thyroid dysfunction, and also marked pathology of the musculoskeletal system, due to which it is impossible to perform a 6-minute walk test; refusal of the patient to participate in the study. Patients with CHF for the comorbid flow with CHD received appropriate treatment that did not change, and in addition appointed polikozanol ("Phytostatein") in a dose of 10 mg 1 time in the evening during the evening. Before course treatment for patients with CS with dyslipidemia additionally prescribed polikozanol under the same scheme. In accordance with the Recommendations on the Diagnosis, Prevention and Treatment of Dyslipidemia of the Ukrainian Association of Cardiologists (2011), a hypolipidemic diet and modification of lifestyle were recommended. The course of treatment lasted 3 months.

In the dynamics of the treatment, the lipid plasma spectrum of blood levels was measured by the levels of ZHC, HDL cholesterol, TG (determined by photocolorimetric method using the reagents of the company Lachema (Czech Republic) by the Zlatiks-Zak method). The level of LDL cholesterol was

determined by the calculation method of the Friedewald formula: LDL cholesterol = LDL cholesterol (HDL cholesterol + TG/2,2) (mmol/l), provided that the TG concentration does not exceed 4.5 mmol/L; the definition of LDL cholesterol was performed by the calculation method, using the formula: Cholesterol LDLC = ZHC — (LDL cholesterol + HDL HDL). The index of atherogenicity (IA) was calculated according to the formula IA = (HDL — HDL HDL)/HDL HDL. Statistical processing of the data was performed on a ViewSonic personal computer using standard Microsoft Excel software packages and using BioStat computer software, Statistica for Windows version 6.0 (Stat Soft inc., USA). Calculated the average arithmetic value (M), its error (m), the Student's criterion (t), the probability (p) with a probability of not less than 95%.

Results of research and their discussion

In the group of patients with CP with dyslipidemia, the prevalence of I-II st. obesity and biliary pathology. The average index of BMI in the group of patients with CP was 27, 28 ± 0.67 kg/m2, and in the group for a comorbid flow of 28, 24 ± 1.01 kg/m2.

In the groups of patients who were examined, there was a significant increase in the rates of total lipids and CFCs of blood plasma, TG and reduction of HDL cholesterol indexes, as shown in Table. 1

Table 1

Lipid profile of blood in the examined patients with CP and with dyslipidemia and on CP with CHD, including in the dynamics of treatment with polycosanol

Indexes	Patients with CP n = 10			Patients with CP and IHD n = 11			Healthy n = 10
	before treatment	after treatment	Δ, %	before treatment	after treatment	Δ, %	
Total CS, mmol/l	6,46±0,22*	5,32±1,49	-18%	7,32±0,15*	5,8±0,22**	-21%	4,8±0,8
TG, mmol/l	2,37±0,16*	2,09±0,69**	-12%	2,53±0,2*	2,06±0,14**	-19%	1,43±0,4
LDL, mmol/l	4,32±0,4*	3,18±0,3	-26%	5,14±0,3*	3,68±0.2**	-28%	2,81±0,29
VLDL, mmol/l	1,08±0,14*	0,95±0,14	-12%	1,15±0,11*	0,94±0,12	-19%	0,65±0,06

HDL, mmol/l	1,06±0,2*	1,19±0,29**	12%	1,03±0,18	1,18±0,09**	15%	1,34±0,07
IA	**5,09**	**3,47**	**-32%**	**6,11**	**3,92**	**-36%**	**2,58**

*Note: * — the indices of patients with respect to practically healthy differ significantly (p <0.05);*

*** — the probability of difference (p <0.05) compared with the indexes of patients after treatment* Δ, %

Prior to the appointment of Phytostatemin, all patients had (significantly> <0.05) elevated HRH, LDL cholesterol, LDL cholesterol and TG (Fig. 1).

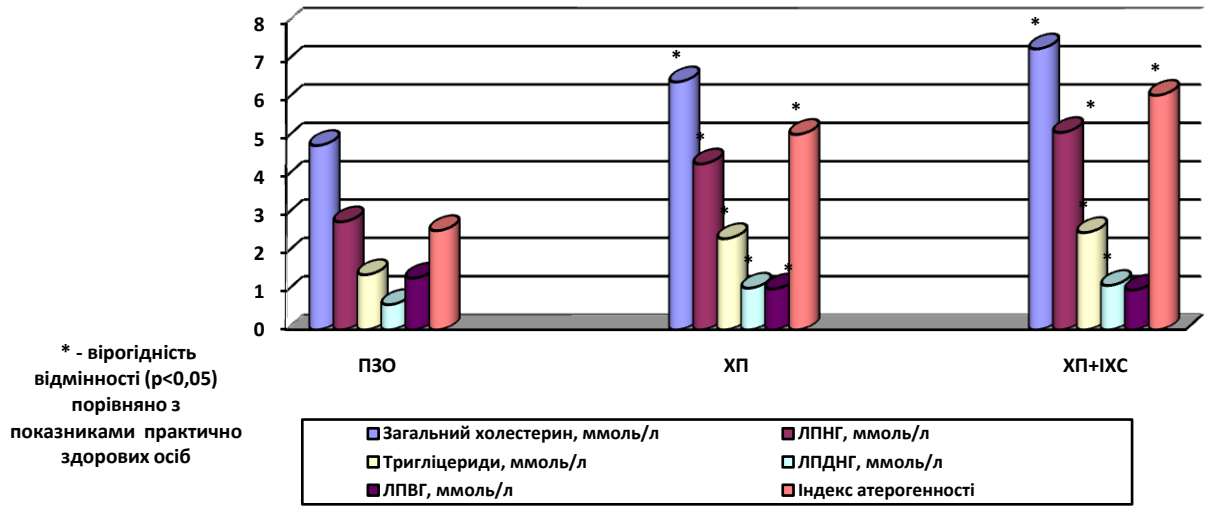

Fig. 1 Lipid profile of blood in the examined patients with CS with dyslipidemia and on CP with CHD.

The level of HDL cholesterol as anti-atherogenic plasma factor was significantly lower in both groups of patients (relative to the indicator of the group of practically healthy), which can be attested not only to the severity of atherosclerotic changes, but also to emphasize the importance of such an etiopathogenetic link in the development of CP as hypertriglyceridemia, especially in the case of lipogenic CP in this groups of patients [7]. Such changes are known to increase the risk of developing atherosclerosis and the possibility of its complications, including in the group of patients with a combined course of CP [4]. Confirmation of such an assumption is an elevated index (coefficient) of atherogenicity in the examined patients for the comorbidity of CP and CHD. In the analysis of the types of dyslipidemia, it was found that IIa and II types of dyslipidemia were more frequent (22 and 25% respectively), but for comorbidity with CHD, dyslipidemia IIa and IU were recorded, which indicated the probability of atherosclerotic changes in the blood vessels, including those with CP

Analyzing the indicators of lipidogram (Figure 2) regarding the effect of 3-month treatment for the appointment of polycosinol, we can say that in patients with CP significantly increased the incidence of HDL-C and significantly decreased TG. This provides the opportunity to talk about sufficient hypolipidemic efficacy that can prevent the development of CP (triglyceride by etiology) and the aggressive development of atherosclerosis, which for a long time has not clinically shown in such patients.

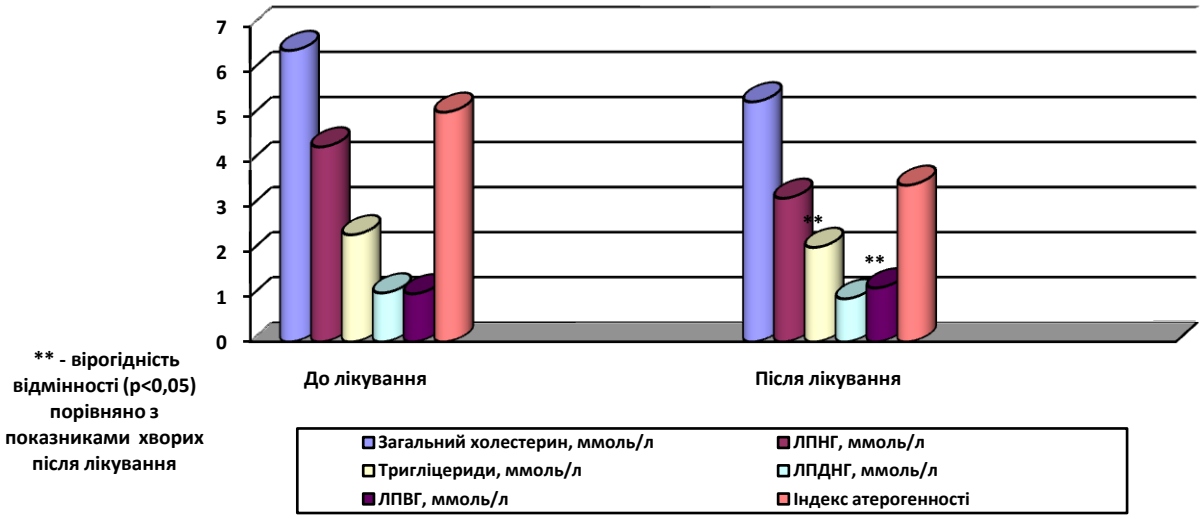

Fig. 2. Lipid profile of blood in examined patients with CP in the dynamics of treatment with polikozanol.

Reduction of hypertriglyceridemia at the time of treatment completion emphasizes the importance of polycosanol as a herbal preparation, which can be recommended for treatment and for the prevention of atherogenic dyslipidemia. Significant hypocholesterolemic effect was also observed in the group of patients with comorbid flow with CHD (Fig. 3), where treatment with polycosanol was performed in combination with statins in starting doses (atorvastatin or rosuvastatin).

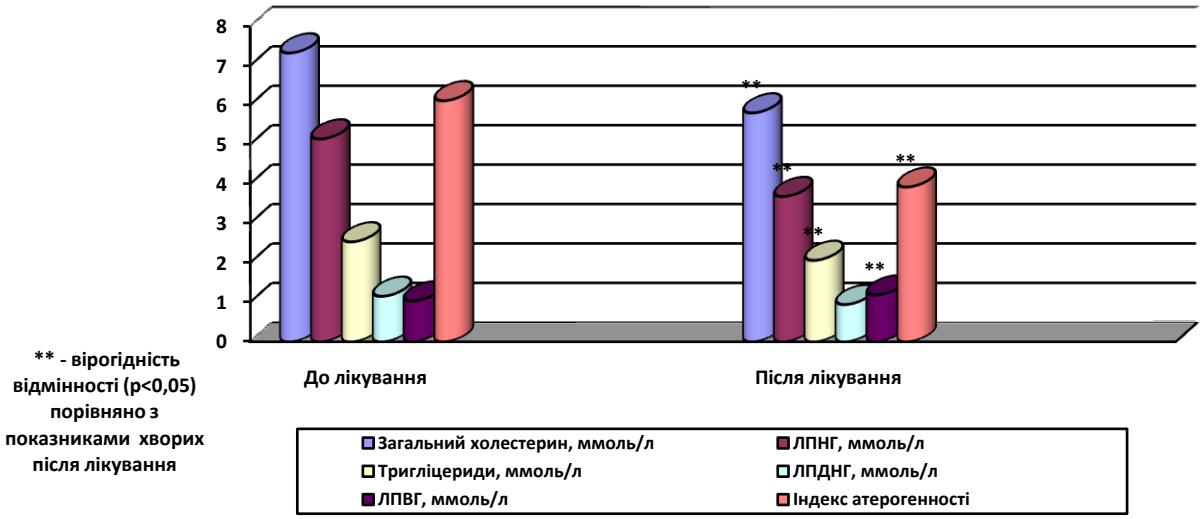

Fig. 3. Lipid profile of blood in the examined patients with CP with CHD in the dynamics of treatment with polikozanol.

However 3 months of treatment in most patients failed to achieve targets lipid spectrum of the blood, which emphasizes the need for further treatment 6-12 months or more, as indicated by the literature.

Consequently, polycosanol ("Phytostate") is a mixture of fatty alcohols, which is isolated from the waxy mass of sugar cane, and has a hypolipidemic effect, increasing the level of HDL cholesterol, protecting LDL cholesterol from damage by free radicals, reducing hypertriglyceridemia, suppressing excess platelet aggregation, improving efficacy of statin therapy in combination therapy. To achieve prognostically significant changes in lipid spectrum would prefer long-term (6-12 months) intended use low initial doses of lipid-lowering drugs (10, 20 mg) daily in combination with policosanol. It is also has a significant effect on lipid spectrum, but will not be associated with symptoms of side effects that are characteristic statynoterapiyi in high doses.

The use of policosanol in combination with statins in starting doses provided comorbid disease course provides an opportunity to reach a wider and significant impact on all parts of the lipid spectrum (hypercholesterolemia, hypertriglyceridemia, low HDL cholesterol level) than just isolated by appointment only statins. Based on this, statin treatment in combination with polycosanol may be recommended for both concomitant diabetes and metabolic syndrome, and with the comorbid flow of CP with CHD, in patients with impaired liver function in patients with chronic heart failure and in patients with hypertriglyceridemia (without the risk of side effects effects)

Conclusions

1. Polycosanol in the complex treatment of patients with CP in combination with hyperlipidemia and CHD has a sufficient hypolipidemic effect (improves the parameters of HDL, TG and atherogenic index).

2. For a comorbid flow of CP with CHD, the drug in combination with statins improves the lipid profile of plasma plasma (when used in starting doses for 3 months). This allows to recommend patients with CP in combination with hyperlipidemia, and also for comorbidity with CHD to use a preparation in combination with statins for 3 months for 10 mg 1 time a day during the evening (in order to improve cholesterol metabolism).

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Chronic pancreatitis in patients with coronary heart disease: a lipid spectrum of blood, a possibility of correction

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Key words: chronic pancreatitis, atherosclerosis, coronary heart disease, indicators lipidogram, statins, policosanol ("FitoStatin")

Aim of research is to evaluate significance of changes in the lipid spectrum of blood in patients with chronic pancreatitis with coronary heart disease in the pathogenesis of the comorbidity of these diseases and in the dynamics of treatment with polycosanol.

Materials and methods. The study was conducted in 22 patients (10 patients with chronic pancreatitis and dyslipidemia, 12 patients with comorbidity of chronic pancreatitis and coronary heart disease in CHD II-II A-B syndrome of stage II-III functional class) and in 10 almost healthy individuals. There were 13 men, 9 women, 31–69 years old. Patients of two groups in addition to protocol treatment were prescribed polycosanol 10 mg 1 time in the evening during dinner, up to 3 months. To study the characteristics of the lipid spectrum of the blood, the level of total cholesterol, high-density lipoprotein cholesterol, triglycerols was determined (using the Zlatix-Zack-based Lachema reagents (Czech Republic)). The level of low-density lipoprotein cholesterol was determined using the Friedewald calculation method, taking into account that the triglycerol concentration did not exceed 4.5 mmol/l. In addition, very low density lipoprotein cholesterol and an atherogenicity index were determined using conventional calculation methods.

Results. In patients with a combined course of chronic pancreatitis with coronary heart disease, in most cases there is a significant ($p<0.05$) increase in total cholesterol, low and very low-density lipoproteins and triglycerols. When analyzing the types of dyslipidemia, it was found that IIa and IIв types were more common (22 i 25%, respectively), but with comorbidity II and IV type of dyslipidemia was more often detected. In the dynamics of a three-month treatment with polycosanol in patients with chronic pancreatitis, the cholesterol levels of high-density lipoproteins increased significantly and the triglycerol values significantly decreased, indicating a hypolipidemic effect of the drug and the possibility of using it in combination with statins in order to reduce the risk of cardiovascular events.

Conclusion. The comorbidity of chronic pancreatitis with ischemic heart disease increases the risk of progression of dyslipidemia and atherosclerosis. This is confirmed by an increased atherogenic index in this group of patients, along with

the severity of lipid spectrum disorders. The addition of polycosanol to patients with chronic pancreatitis and dyslipidemia, as well as in combination with coronary heart disease, contributes to the reduction and normalization of certain parameters of the lipid spectrum of the blood. This allows us to recommend a drug for long-term treatment of these groups of patients, including in combination with statin therapy.