



The comorbidity of chronic pancreatitis with ischemic heart disease worsens clinical symptoms, determines the progression and prognosis of diseases. The reason for this may be latent-running chronic systemic low-intensity inflammation, which manifests itself as a systemic impression and wavelike activation of the cascade of proinflammatory cytokines. The development and persistence of the immune cytokine mechanism in a comorbid flow of chronic pancreatitis with coronary heart disease creates a precedent in such target organs as myocardium and pancreas. In this connection, in order to control the intensity of chronic systemic inflammation, one may propose to take into account the high level of CRF, proinflammatory cytokines when determining the treatment tactics and carrying out rehabilitation and preventive measures.

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THE ROLE OF ENDOTHELIAL DYSFUNCTION IN THE PROGRESSION OF CHRONIC CHOLECYSTITIS

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Endothelial dysfunction is the main factor that leads to the development and progression of atherosclerosis.

The aim of the study was to set the degree of development and the role of endothelial dysfunction in the genesis and progress of chronic cholecystitis (CC) in patients with ischemic heart disease (IHD) and obesity.

136 patients were examined: Group 1 (n = 28) - CC; Group 2 (n = 30) - CC on the background of IHD; Group 3 (n = 30) - CC against the backdrop of IHD and 1-2 grade obesity; Group 4 (n = 30) - CC, cholesterosis gallbladder (CG), IHD, obesity 1-2 grade; Group 5 (n = 18) - CC and CG. The functional state of the endothelium was studied by blood levels of stable metabolites of nitrogen monoxide (NO), the activity of endothelial (eNOS) and inducible (iNOS) NO-synthase and endothelin-1 (ET-1) by ELISA.

Results of the study showed that in 97,8% of examined patients with CC a significant increase in the content of stable NO metabolites in the blood ($p < 0,05$) was found. Patients of 3rd group experienced substantial growth content of NO in blood (2,4 fold) compared to the 1st group (1,9 fold) and 2nd group (1,6 fold) ($p < 0,05$). It was established that the stress intensity increased by joining IHD and obesity for CC and cholesterosis (an increase of 2,8 times to 2,1 times, $p < 0,05$). The 4th group: the content of NO in blood exceeded compared to the 1st group by 17,7% ($p < 0,05$). The 4th group found most pronounced indicators: overproduction of iNOS (growth 5,2 times) and eNOS deficit (down by 53,0%) ($p < 0,05$). So, revealed endothelial dysfunction in patients with comorbid disorders CC by pathological induction of iNOS activity and increasing of nitrate causes hypokinetic gallbladder dysfunction and progression CC that deepens with increasing degree of obesity.

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CLINICAL AND INSTRUMENTAL MARKERS OF ACUTE MYOCARDIAL INFARCTION COMPLICATED WITH ACUTE HEART FAILURE FLOW EVALUATION

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Determination of prognosis within first year after acute myocardial infarction (AMI) remains one of the most topical issues in cardiology.

368 patients were examined with the purpose to create a prognostic model of acute myocardial infarction complicated with acute heart failure flow. Some risk factors of lethal outcome were distinguished.

Transmural AMI occurred in 141 (38,32%), macrofocal AMI – in 166 (45,11%) and microfocal AMI – in 61 (16,57%) cases as determined by results of a comprehensive clinical-instrumental examination including detailed complains, taking anamnesis, careful clinical investigation, electrocardiography in dynamics. 123 (33,42%) persons out of 368 examined died throughout observation period, in particular, 94 (25,54%) patients – during 28-day staying in hospital, and 29 (7,88%) – during a year of follow up.

With a purpose of creating prognostic models of AMI complicated by acute left-ventricular failure (ALVF), all patients were divided into 2 groups: group 1 – with favorable AMI outcome, and group 2 – with fatal outcome.

Patients who died were averagely 9 years older as compared to those with favorable outcome. Males were prevalent amongst ($p < 0,001$). Besides, relapsed AMI was registered much more frequently in 2 group patients (79,6% vs 39,19% in group 1, $p < 0,001$). Class 2-4 ALVF signs by T. Killip were significantly more frequent in group 2 patients ($p < 0,001$). Frequency of arterial hypertension (AH) and diabetes mellitus (DM) presence in anamnesis was significantly higher in group 2 patients as well ($p < 0,01$). Risk factors prevalence analysis among patients of both groups revealed significant prevalence of active smoking ($p < 0,01$) and obesity ($p < 0,001$) in group 2 patients as well.

Single-factor regression analysis results were indicative of the fact that risk of lethal event occurrence increased with age: increase of risk by a factor of 1.5 follows each additional 5 years over 50. Risk of lethal event appearance raised twice with every ALVF class by Killip increase, 1.02 times more with income IIR increase on 10 b.p.m. after 60 b.p.m., 1.3 times more in patients with DM, 1.15 times more in case of obesity presence, three times more in patients with chronic heart failure (CHF), 1.2 times more in case of ejection fraction (EF) below 40% detection during 1-2 days after patient's admission, and 4.5 times more in case of anterior AMI localization.



IL-1 α content analysis revealed its significant predominance in group 2 patients ($48,94 \pm 7,05$ vs $22,43 \pm 3,41$ pg/ml (group 1), $p < 0,01$). IL-6 level was markedly higher in group 2 patients as well ($51,63 \pm 7,86$ vs $16,84 \pm 3,94$ pg/ml, $p < 0,01$), and level of anti-inflammatory cytokine IL-10 was some less in group 2 patients comparing group 1 ($2,45 \pm 0,51$ vs $4,03 \pm 0,73$ pg/ml, $p > 0,05$).

Tumor-necrotizing factor (TNF) and neopterin (Np) levels analysis in groups indicates significant predominance of these both values in group 2 patients comparing group 1: $63,41 \pm 3,78$ vs $43,1 \pm 2,62$ pg/ml for TNF ($p < 0,01$) and $24,28 \pm 4,32$ vs $15,08 \pm 1,76$ nmol/l for Np ($p < 0,05$).

Elder patients age, higher class of ALVF, presence of DM and CHF, anterior localization of AMI, smoking and obesity, EF low then 40% are independent predictors of lethal event development in patients with AMI and ALVF. Besides, increase in pro-inflammatory cytokines level (IL-1 α , IL-6, TNF and Np) parallel to worsening of EchoCG EF results in favor of increase of lethal event onset probability in the mentioned category of patients.

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CHANGES OF THE IMMUNE DEFENCE IN DIABETIC PATIENTS WITH PYOINFLAMMATORY PROCESSES

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The immune system disorders in diabetic patients lead to a significant decrease in non-specific and specific immune anti-infectious defense by inhibiting phagocytic function of polymorphonuclear leukocytes, lowering of complement system activity, lysozyme, interferons, bactericidal activity of blood serum.

Diabetic patients with pyoinflammatory processes treated by traditional methods ($n=40$); diabetic patients with pyoinflammatory processes treated by ozonotherapy along with traditional treatment ($n=53$). The obtained results confirm changes in the absolute and relative number of immune cells in the peripheral blood of DM patients associated with pyoinflammatory processes. A relative number of lymphocytes decreases in these patients, at the same time a tendency to growth in the absolute number of the total pool of lymphocytes is formed. The research of the immune disorders degree confirmed that therapeutic measures, including ozonotherapy, against pyoinflammatory processes in patients with DM show their effectiveness. On admission 65,0% of patients were diagnosed with the I-II degree of immune disorders, which required immunorehabilitation; after pyoinflammatory processes therapy only 55,0% of diabetic patients were left. Special efficiency is shown in the III stage of immune disorders.

Pyoinflammatory processes in patients with diabetes occur on the background of decrease in the appropriate number of lymphocytes; increase in the absolute and relative number of monocytes, the absolute number of leukocytes due to the increase in the relative amount of neutrophilic polymorphonuclear leukocytes, as well as decrease in the absolute number of eosinophils, erythrocytes and hemoglobin.

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METFORMIN IMPROVES ENDOTHELIAL VASCULAR REACTIVITY IN FIRST-DEGREE RELATIVES OF TYPE 2 DIABETIC PATIENTS

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Endothelial dysfunction is an early marker of atherosclerosis seen in type 2 diabetic subjects. Metformin is commonly used in the treatment of type 2 diabetes and has besides hypoglycemic, a known vascular protective effect. We aimed to investigate the vascular effects of metformin in first-degree relatives with metabolic syndrome of type 2 diabetic patients.

The study included 43 subjects (age $38,3 \pm 7,6$ years and BMI $36,3 \pm 5,2$ kg/m²), who were first-degree relatives of type 2 diabetic patients and who had metabolic syndrome and normal glucose tolerance. The subjects were randomly assigned 1:1 in a double-blind fashion to receive placebo ($n = 13$) or metformin ($n = 30$). Endothelial function was assessed by venous occlusion plethysmography, measuring forearm blood flow (FBF) and vascular resistance responses to three intra-arterial infusions of endothelium-dependent (acetylcholine 7,5, 15, and 30 μ g/min) and independent (sodium nitroprusside 2, 4, and 8 μ g/min) vasodilators. Weight, BMI, systolic and diastolic blood pressure, waist, and laboratory parameters (lipid profile and fasting plasma glucose [FPG]) were assessed before and after treatment.

The metformin and placebo groups did not differ in anthropometric, clinical, laboratory, and vascular measurements at the beginning of the research. The metformin group had decreased weight, BMI, systolic blood pressure, and FPG and improved lipid profile. Endothelium-dependent FBF responses were also improved, without any effect on endothelium-independent responses. There was no correlation between the improvement on FBF responses and the observed changes on anthropometric, clinical and laboratory parameters.

We concluded that metformin improved vascular endothelial reactivity in first-degree relatives with metabolic syndrome of type 2 diabetic patients, regardless of its known antihyperglycemic effects. Accelerated atherosclerosis seen in type 2 diabetes raised the question about pathogenetic factors that initiate the development of vascular disorders in the pre-diabetic population. Metabolic syndrome, a pre-diabetic state, includes a number of cardiovascular risk factors such as abdominal obesity, dyslipidemia, hypertension, impaired glucose tolerance, and insulin resistance.