The state of lipid metabolism, lipid peroxidation and antioxidant defense in patients with chronic obstructive bronchitis with hypertrophy and atrophy of the myocardium

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Introduction. In recent years, there has been an increase in the proportion of morbidity and mortality of population from chronic non-specific lung diseases (CNSLD)[1]. This is due to a number of reasons: air pollution, smoking, resistance of the bacterial flora to some antibiotics. A special attention of public health authorities to this problem is determined by the fact that the consequences of CNSLD, complicated by pulmonary heart disease (PHD), eventually lead to patients’ (primarily men) disability and death at able-bodied age (50-60 years)[2]. Thus, according to the WHO Expert Committee, more than half of patients with lung diseases develop PHD, which leads to frequent hospitalization. Statistics show that 16-20% of all hospitalized subjects are PHD patients[3].

According to modern research findings, activation of lipid peroxidation is one of the leading links in PHD pathogenesis in chronic bronchitis[4].

The aim of our research was to study the dynamics of markers of lipid peroxidation (LPO) and antioxidant defense (AOD) in patients with chronic obstructive bronchitis (COB) and heart failure (HF) with myocardial hypertrophy and atrophy.

Materials and methods. To solve the abovementioned tasks, we examined 62 patients of both sexes with COB and HF, stage I-II. Each of them had at least 2 risk factors for COB (smoking, dustiness of the workplace, frequent colds, family history). Determination of nosological form of COB was carried out in accordance with the WHO criteria. The stage of circulatory failure was assessed in accordance with the classification by N.D. Strazhesko and V.Kh. Vasilenko. The diagnosis of COB was made on the basis of anamnestic, clinical and biochemical, instrumental data obtained by examining patients.

30 patients (study group 1) with hypertrophy of the myocardium and 32 patients with myocardial atrophy (study group 2) were examined.

All the patients underwent the following tests: total serum lipids, total cholesterol, β-lipoproteins, phospholipids, peroxide hemolysis of erythrocytes (PHE), malonic dialdehyde (MDA), superoxide dismutase (SOD), ceruloplasmin, catalase according to standard methods.

Results. Our findings indicate that in both groups of patients total lipids, cholesterol, and β-lipoproteins in the blood were significantly increased. Studies of LPO in these groups of patients showed an increase of MDA in both groups of patients, but in the group with myocardial atrophy these rates were significantly higher than in the group with hypertrophy (14.34 ± 0.28 μmol / l and 10.67 ± 0.55 μmol / l, respectively). The increase in peroxide hemolysis of erythrocytes was also more manifested in the group with myocardial atrophy (19.38 ± 1.05%) than in the group with hypertrophy (14.41 ± 0.89%). A statistically significant (p<0.05) decrease in catalase activity in the group with myocardial atrophy (2.11 ± 0.07) was also revealed. From the data obtained it follows that a greater activation of LPO was noted in the group with myocardial atrophy. This can be associated with the release of pro-oxidant factors from the destroyed cardiomyocytes.

Conclusion. Thus, the change in LPO markers indicates its significant activation in the development of myocardial atrophy, which makes it possible to consider the activation of LPO as one of the leading mechanisms in the development of this particular form of PHD in patients with CNSLD.

Prospects for further research. The obtained results are the basis for the development and application of schemes for the diagnosis and treatment of PHD in patients with CNSLD, focused on antioxidant therapy.

Recommendations. It is advisable to use LPO markers to evaluate the course of CNSLD with PHD.
RELATION OF GROWTH-DIFFERENTIATION FACTOR-15 LEVELS AND NUMBER OF CIRCULATING ENDOTHELIAL PROGENITOR CELLS IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Background: risk stratification of patients with established type 2 diabetes mellitus (DM) is under scientific discussion and appears to be controversial issue.

The objective: to investigate relationship between levels of growth differentiation factor-15 (GDF-15) and circulating number of endothelial progenitor cells (EPCs) with angiopoetic phenotypes: CD34+CD14+CD309+, and CD34+CD14+CD309+Tie2+ in patients with type 2 DM.

Materials and Methods. The study retrospectively involved 76 patients with type 2 DM aged 38 to 55 years and 30 healthy volunteers. Data collection included demographic and anthropometric information, hemodynamic performances and biomarkers of the disease. EPCs’ populations were determined by flow cytometry.

Results. The levels of GDF-15 in peripheral blood of diabetics associated with age (r = 0.31, P = 0.044), high-sensitive C-reactive protein [hs-CRP] (r = 0.40, P = 0.001), smoking (r = 0.38, P = 0.001), body mass index [BMI] (r = 0.34, P = 0.001), LDL cholesterol (r = 0.28, P = 0.001), glycated hemoglobin [HbA1c] (r = -0.28, P = 0.001), number of CV risk factors (r = 0.26, P = 0.001). In univariate logistic regression analysis we found that level of GDF-15 ≥ 618 pg/mL, hs-CRP ≥ 7.12 mg/L, HbA1c ≥ 6.4%, fasting glucose ≥ 6.7 mmol/L, and BMI ≥ 27.3 kg/m² predicted deficiency of both angiopoetic phenotypes of EPCs. In multivariate logistic regression model GDF-15 ≥ 618 pg/mL demonstrated the best odds ratio values for declining of EPCs in diabetics in comparison with other predictors including BMI, HbA1c and hs-CRP.

In conclusion, GDF-15 was extremely evaluated in type 2 DM population to healthy volunteers and it was an independent factor that contributes to mobilization and probably proliferation of endothelial precursors with high angiopoetic activity.

Key words: growth differentiation factor-15; endothelial progenitor cells; type 2 diabetes mellitus.

MARKERS OF ENDOTHELIAL INJURIES IN PATIENTS WITH CORONARY HEART DISEASE AND AUTOIMMUNE THYROIDITIS

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According to WHO, coronary heart disease (CHD) has been on the top of the list of 10 leading causes of death in the world over the years, and its share is 12.8% [1,2]. CHD mortality among working age population is 28.3% [3]. At the same time, there is a significant increase in autoimmune thyroiditis (AIT) in society; particularly, in Ukraine the prevalence of AIT has increased by 68% over the past 10 years [3,4]. Today, endothelial dysfunction (ED) is considered to be the pathogenetic basis for the formation of atherosclerotic vascular lesions. ED, formed in conditions of chronic systemic inflammation (CSI), is the earliest stage of atherogenesis, and it plays a leading role in the progression of