FEATURES OF CYTOKINE PROFILE AND ENDOTHELIAL FUNCTION IN COMORBID COURSE OF ESSENTIAL HYPERTENSION

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Appearance of the new terminology such as “endothelial dysfunction” and “chronic systemic inflammation” has designated a new round in the study of essential hypertension (EH), namely – to defining the role of the immune inflammatory mechanisms in the development and progression of vascular pathology [1, 2, 3]. It should be noted that the peculiarity of internal pathology at present time is its comorbidity [4, 5]. Combination of EH with duodenal peptic ulcer (DPU) is frequent. It can complicate diagnostics, modify clinical symptoms and worsen treatment quality [6]. The foregoing formed basis of this study. The study is aimed to identify the features of the changes of the cytokine profile and endothelial function in patients with EH and comorbid DPU.

Materials and methods. Totally 65 patients with second stage of EH (medication control) and 33 patients (main group) had EH in combination with DPU (remission period). The study population had a mean age of 44.3±2.9 years. Reference indicators were obtained while studying 23 practically healthy individuals, sex and age of whom did not differentiate with those of examined patients.

Spectrophotometrical method was used to evaluate the indices of endothelial function: the level of ultimate stable metabolites of nitrogen oxide (Gris reagent) – nitrates (NO$_2$), nitrates (NO$_3$), their total content (NO) in blood serum and risk-marker of thrombogenic complications (according to ristomycin-induced platelet aggregation (RIPA)) [7, 8]. Blood test for pro- (TNF-α, IL-1β, IL-6) and anti-inflammatory (IL-10) cytokines (Ck) was conducted by immunoenzyme method.

Results. Present study revealed that patients with comorbid EH (compared to reference data) had 1.5 times decreased level of NO$_2$, 1.6 times decreased level of NO$_3$ and 1.6 times decreased NO (р<0.001 in all cases). Unlike patients with isolated course of EH, in EH comorbidity RIPA not only exceeded 1.45 times reference data (р<0.001) and 1.12 times index of the comparison group (р<0.05), but also the physiological threshold of this index on the whole, and, moreover, the inverse correlation was found between RIPA and NO$_3$ (r = -0.27; р<0.01).

In patients of the main group an essentially increased level (in comparison with reference data) of Ck was revealed - TNF-α (2.6 times higher; р<0.001), IL-1β (2.3 times higher; р<0.001), IL-6 (1.6 times higher; р<0.001) and IL-10 (1.3 times higher; р<0.05). Noteworthy that absolute content of proinflammatory Ck in the patients of the main group had been higher than in the comparison group: TNF-α – 1.5 times higher (р<0.001), IL-1β – 1.4 times higher (р<0.05), IL-6 – 1.3 times higher (р<0.05). At the same time, TNF-α/IL-10 index (almost twice higher (р<0.01) than in the reference data) was 1.3 times higher than in comparison group (р<0.01); IL-1β/IL-10 and IL-6/IL-10 indices exceeded reference data values 1.9 times higher (р<0.001) and 1.3 times higher (р<0.05), respectively. Patients of the main group had negative correlations between TNF-α and NO$_2$, IL-1β and NO$_3$ (r = -0.30 and r = -0.28 accordingly; р<0.01) and their severity was higher than in patients of comparison group (r = -0.27 and r = -0.24 accordingly; р<0.01). Direct correlation was found between TNF-α and RIPA (r = 0.28; р<0.01) as well as between RIPA and IL-1β (r = 0.26; р<0.05), they were more pronounced than in patients of the comparison group (r = 0.25 and r = 0.22 respectively; р<0.05).

Patients with EH in conditions of comorbis course with DPU have changes of cytokine profile with predominance of pro-inflammatory Ck. Presence of correlation relationships between pro-inflammatory Ck and indices of endothelial dysfunction, pro-inflammatory Ck and risk-marker of thrombogenic complications reflects their community in the mechanisms of comorbis pathology formation. It should be considered as burdening criterion in conditions of comorbis course of EH and DPU.

It is necessary to take into account the results of this study during stratification of risk-factors in patients with EH in conditions of comorbis course.

References:
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 (14.11 ± 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.