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DIAGNOSIS AND TREATMENT OF IMMUNOLOGICAL CHANGES IN SOME DISEASES OF THE CARDIOVASCULAR SYSTEM BY MODERN METHODS

Resume: The immune system (IS) is activated not only during infectious aggressions, but also reacts to any stressful effects, including ischemia, hemodynamic overload, intoxication, etc., that is, to those factors that cause the development and heart failure (HF). There are several interrelated components of IC that may be involved in the pathogenesis of HF, and the main ones are pro-inflammatory cytokines, adhesion molecules, autoantibodies, nitric oxide and endothelin-1. Much attention in immunological studies in HF has recently been paid to chemokines that induce migration of phagocytes in the myocardium, as well as shock proteins, components of oxidative stress.

Keywords: immune system, cardiovascular system, diseases.

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ДИАГНОСТИКА И ЛЕЧЕНИЕ ИММУНОЛОГИЧЕСКИХ ИЗМЕНЕНИЙ ПРИ НЕКОТОРЫХ ЗАБОЛЕВАНИЯХ СЕРДЕЧНО-СОСУДИСТОЙ СИСТЕМЫ СОВРЕМЕННЫМИ МЕТОДАМИ

Резюме: Иммунная система (ИС) активируется не только при инфекционных агрессиях, но реагирует также на любое стрессовое воздействие, в том числе ишемию, гемодинамическую перегрузку, интоксикацию и т.д., то есть на те факторы, которые являются причиной развития и сердечной недостаточности (СН). Существуют несколько взаимосвязанных компонентов ИС, которые могут быть задействованы в

патогенезе СН, и главные из них - провоспалительные цитокины, молекулы адгезии, аутоантитела, оксид азота и эндотелин-1. Большое внимание в иммунологических исследованиях при СН в последнее время уделяется хемокинам, индуцирующим миграцию фагоцитов в миокарде, а также шоковым белкам, компонентам оксидативного стресса.

Ключевые слова: иммунная система, сердечно – сосудистая система, заболевания.

Relevance. Recently, more and more attention has been paid to the importance of immune mechanisms in the implementation of pathological processes of various localization, including in the development of cardiovascular diseases [3].

Some researchers believe that atherosclerosis is a manifestation of premature aging of the immune system [2]. It is believed that somatic cell mutations play an important role in the occurrence of autoimmune processes during aging. In this regard, it is believed that the immune system plays a significant role not only in preserving, but also in shortening life. Studies have shown that autoimmune reactions can cause damage to the heart and blood vessels [4]. The resulting autoimmune damage, especially characteristic of the elderly, is characterized by ultrastructural rearrangement of myocardiocytes [5, 6].According to available data, patients of different age groups, including young children with non-coronary myocardial lesions (myocardiodystrophy, myocarditis, myopericarditis, dilated and hypertrophic cardiomyopathy), coronary artery disease, hypertension, heart defects, cardiac arrhythmias may have a variety of immune disorders [1,3]. In the myocardium, as in other organs and tissues, inflammatory changes occur under the influence of many factors: this is a direct invasion of an infectious agent, and immune-mediated damage to the heart muscle, and the effect of various toxins. It is shown that there is a connection between the clinical features of myocardial damage and immunological indicators characterizing different degrees of immune imbalance

[2,5]. Immune mechanisms of heart damage of both inflammatory and non-inflammatory genesis are of particular interest [6]. The basis of inflammatory changes of any localization is the infiltration of the organ by cells of the immune system, accompanied by the secretion of cytokines (TNF-a, IL-1, IL-6), the accumulation of oxidative. Recently, the role of complement system activation and cytokine balance disorders in heart diseases has been discussed [1]. In patients with various cardiac pathologies (myocarditis, dilated, hypertrophic cardiomyopathy, stress cardiomyopathy, chronic heart failure), an important role belongs to mediators of the acute phase response - interleukin-1 (IL-1), IL-6, tumor necrosis factor (TNF-a). These cytokines regulate the immune response and provide homeostasis in physiological concentrations, in high doses, the same cytokines can have a pathological endocrine-like effect, causing microvascular hypercoagulation, hemodynamic disorders and metabolic depletion [2].

There was an increase in the level of TNF-a in the blood serum in heart failure developed against the background of DCMP, while in some cases the degree of hyperproduction correlated with the functional class of circulatory insufficiency [3]. Of great interest is the work of T. V. Bershova and co. (2010), demonstrating the activation of all links of apoptosis in chronic heart failure in children and adolescents, and the severity of the induction of apoptosis depends on the stage of circulatory insufficiency. It is shown that the change in the content of molecular agents of apoptosis depending on the concentration of nitric oxide and superperoxide dismutase indicates their participation in the regulation of programmed cell death. The relationship of apoptosis indicators and echocardiographic parameters confirms the participation of cellular apoptosis in the development of myocardial remodeling in children [2,4].

An increase in the level of IL-2, which plays a major role in the regulation of lymphocyte differentiation, IFN-Y contributing to the biosynthesis of plasma

cells, as well as colony-stimulating factors was detected in patients with myocarditis [3,6].

The purpose of the study. The purpose of this study was to develop and systematize criteria for early diagnosis, severity of damage to the cardiovascular system, immediate and long-term prognosis, based on a comprehensive clinical, instrumental and laboratory assessment, in immune systems..

Materials and methods of research. To solve the tasks, the state of the cardiovascular system in the acute and long-term periods of the disease was assessed in 748 patients, 265 patients with diphtheria were examined, 149 with changes in immune systems.

The results of the study. The manifestation of autoimmunization in patients with coronary heart disease is characterized by sensitization of lymphocytes to the tissues of the vascular wall and is accompanied by a significant increase in the levels of autoantibodies to LDL and vascular tissues, as well as activation of the phagocytic link of immunity — neutrophils and monocytes. This is combined with the presence of such complications of coronary heart disease as myocardial infarction and left ventricular dysfunction according to echocardiography. The results of tissue typing showed that in the HLA DR locus in the group of CHD patients, significant differences in the frequency of occurrence were noted for the DRB1*12 allele (0.075 vs. 0.023 in the control). The DRB1*04 allele is significantly less common compared to the control group (0.050 vs. 0.112 in the control). When comparing the results of HLA typing with the parameters of the immune system in patients with coronary heart disease, characterizing the manifestations of autoimmune reactions, a positive association of the DRB1*03 allele with increased sensitization of lymphocytes was established, which makes it possible to consider it as a genetic marker of the risk of autoimmune reactions. The analysis of the obtained data on the prevalence of known genes in CHD patients in the HLADRB1 region, for which a reliable link with the development of autoimmunization was proved,

was also carried out. It was found that in the genotype of 61.7% of the examined patients in the HLA-DRB1 genotype, both specificities were neutrally or protectively associated with autoimmunity. In 38.3% of patients, only one HLA-DRB1 specificity was positively associated with autoimmunization, while the second was neutrally or negatively associated. Of the patients we examined, there was not a single one in whose genotype both HLA-DRB1 specificities were from the "functional" group marking the development of autoimmunization.

Conclusion. Modern recommendations for the prevention of cardiovascular diseases pay great attention to the identification and correction of risk factors, the assessment of the likelihood of complications and the prognosis of these diseases. Further study of the involvement of autoimmune processes in the pathogenesis of coronary heart disease and prediction of the risk of their development using immunogenetic analysis will contribute to timely prevention and selection of appropriate treatment regimens, taking into account the need to correct the factors that form autoimmune reactions.

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