CELLULAR IMMUNITY IN CHRONIC HEART FAILURE CAUSED BY ISCHEMIC ETIOLOGY

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Abstract: The article summarizes the literature data on the pathogenetic effect of an increased level of pro-inflammatory cytokines on the progression of chronic heart failure (CHF) of ischemic origin. According to a number of modern researchers, immune activation and systemic inflammation play a key role in the pathogenesis of CHF [5]. According to this concept, there is an increase in the synthesis of pro-inflammatory cytokines that determine the evolution of left ventricular dysfunction. The content of cytokines in the blood plasma of patients with CHF, regardless of its etiology, significantly exceeds normal values [2]. However, the reasons for the activation of the immune system in CHF are not fully understood [2]. It has been suggested that the hyperproduction of pro-inflammatory cytokines, mainly tumor necrosis factor α (TNF-α), is mediated by high sympathetic adrenal activation [1].

Key words:chronic heart failure, coronary heart disease, proinflammatory cytokine, tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6).

Activation of the immune system in chronic heart failure (CHF) has been known for more than 20 years. Initially, experimental studies demonstrated the maladaptive role of the immune system. Modern preclinical studies describe the positive and negative effects of immune activation in CHF. These different effects depend on the timing and etiology of CHF. Therefore, here we give a detailed overview of the immune mechanisms and their significance for the development of CHF.

In various types of autoimmune pathology, the role of genetic predisposition is generally recognized and the most informative genetic markers are considered to be alleles of genes associated with autoimmunity of the main histocompatibility complex (HLA)[1]. It is the products of these marker genes, HLA antigens, that are involved in the launch and implementation of autoimmune processes. The autoimmune basis has already been confirmed in more than 40 diseases, and in many other diseases its participation is assumed. To date, HLA-specificity has been established (especially at the HLA-DRB1 locus), some of which are markers of predisposition, others of resistance to autoimmunization. Since in atherosclerosis, autoantigens also play a significant role in the pathogenesis of the development of immune inflammation in the vascular wall (primarily – modified low-density lipoproteins (LDLP) and chaperones), the purpose of our work was to study autoimmune reactions and analyze the nature of their manifestations depending on the HLADRB1 genotype in patients with coronary heart disease (CHD[2]Recent studies show that regulatory T cells (Tregs) can reduce the penetration of proinflammatory cells into damaged myocardial tissue and prevent chronic inflammation, which can lead to CHF [3]. In patients with CHF, an imbalance was observed between the levels of proinflammatory T helper cells 17 (Th17) and antiinflammatory IL-10 in the blood plasma. It should NOT be forgotten that aging is inversely correlated with the scardioprotective role of T cells. With age, the decrease in immunogenicity is directly related to the functional activity of the thymus, which affects the maturation of T cells [4].

In elderly people, a decrease in the pool of naive T cells, in contrast, an increase in the pool of memory T cells such as CD28null, often leads to an increased susceptibility to pathogenic attack and the subsequent development of cardiac dysfunction [5].To assess the state of immunity, the following indicators were determined: the content of subpopulations of lymphocytes carrying CD3+, CD4+, CD8+, CD16+, CD19+ antigens, It was determined by counting antigen-positive cells using fluorescence microscopy.Lymphocyte populations and subpopulations (cell immunophenotyping) were determined using a panel of monoclonal antibodies to human leukocyte surface antigens (CD markers) in Andijan) by immunofluorescence microscopy. The relative and absolute contents of the following cells were studied: CD3+, CD4+, CD8+, CD16+, CD19+, and the ratio CD4+/CD8+ - immunoregulatory index (IRI) was determined.One of the main cytokine effectors of inflammation in the human body at this stage of the pathogenesis of CHF is TNF, which can directly lead to dysfunction of the heart muscle due to direct negative effects on calcium-dependent processes in cardiomocytes [5], inactivation of nitric oxide in the vascular endothelium, induction of cellular apoptosis [4, 5]. At the same time, the release of pro–inflammatory cytokines causes the activation of residual tissue macrophages and leads to the recruitment of different populations of circulating immune cells into the heart under the influence of specific molecules - chemokines.