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DEVELOPMENT OF CHRONIC CARDIAC DEFICIENCIES IN PATIENTS WITH CORONARY HEART DISEASE

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Abstract. Despite significant advances in the treatment of cardiovascular diseases, the prevalence of chronic heart failure (CHF) not only does not decrease, but, on the contrary, increases. The most common causes of CHF in economically developed countries of the world are coronary heart disease (CHD) and arterial hypertension (AH).

Key words: tumor necrosis factor-α (TNF-α), interleukin-1α (IL-1α).

The prognosis for CHF remains extremely unfavorable, so timely prevention and early diagnosis of this pathology is the most pressing problem of modern cardiology. Recently, a new concept of the development of CHF has been proposed, which is based on the paradigm of systemic inflammation as one of the important independent factors of high cardiovascular risk [2, 3].

According to this concept, nonspecific activation of macrophages and monocytes, which occurs during severe stress disorders of microcirculation, is an inducer of the synthesis of proinflammatory cytokines (in particular, tumor necrosis factor-α (TNF-α), which was previously called cachexin, as well as interleukin-1α (IL-1α) , interleukin-1□ (IL-1□), interleukin-6 (IL-6), etc.), which determine the unfavorable development or regression of ischemic and/or post-infarction cardiac dysfunction, left ventricular (LV) remodeling and the development of LV heart failure [2-4]. Moreover, an increase in the expression of proinflammatory cytokines is directly related to the FC severity of CHF and closely correlates with the concentration of atrial natriuretic peptide, as well as some other neurohormones [3, 5]. The molecular mechanisms underlying cytokinin-induced impairment of inotropic capacity and remodeling of ischemic myocardium are not fully understood. It is assumed that pro-inflammatory cytokines play an important role in the progression of CHF, mediating the nature and intensity of the processes of myocardial and vascular remodeling by the level of cardiomyocyte apoptosis, which is considered as a fundamental mechanism that can cause irreversible impairment of myocardial contractility in CHF [6, 7].

Proinflammatory cytokines are an important and fairly well studied class of biological local cellular pathophysiological regulators that can initiate and aggravate the development of CHF [8-11]. It has been shown that the level of TNF-α in the serum of patients with severe heart failure (NYHA class III-IV) is an order of magnitude higher than in healthy individuals. Moreover, increased levels of TNF-α were recorded

in patients with more severe clinical manifestations of decompensation, a greater degree of cachexia (with a body weight of 82% of ideal weight) and increased activity of the renin-angiotensin-aldosterone system [12]. Other studies draw attention to the close correlation between high levels of TNF-α, IL-1 and IL-6 withthe severity of clinical manifestations [13, 14] and neurohumoral activation in patients with CHF [15, 16]. There is a family of IL-1 cytokines, which includes three cytokines with a high degree of homology: IL-1α, IL-1β and IL-1 receptor antagonist (IL-1Ra). IL-1α and IL-1β have virtually identical activity. If IL-1α is mainly a mediator of local protective reactions, then IL-1α is a secretory cytokine that exerts its action both locally and at the systemic level. IL-1Ra is characterized by the fact that it blocks the biological activity of IL-1 [17]. For more than 10 years, there has been such a field as genetic cardiology, which integrates the concepts and technologies of molecular genetics

to understand the etiology, pathogenesis and mechanisms of clinical polymorphism of human cardiovascular diseases [18, 19].The overall picture of the etiology and pathogenesis of some multifactorial diseases may be dominated by the effect of one or several genes. There are tens of thousands of polymorphic genetic systems, the evolutionary basis of which is the great adaptive value of certain combinations of hereditary factors. But along with well-adapted individuals, there are also individuals with “unfavorable” combinations of hereditary factors. They are the ones who make up the group of people with hereditary predisposition to diseases [20]. In recent years, a number of scientific studies have been carried out in cardiology to assess the contribution of genetic factors to the development of insufficiency blood circulation [21-24]. Thanks to studies of the human genome, identification of its genes, including genes whose mutations lead to hereditary diseases or predispose to the most common polygenic (multifactorial) diseases, for the first time a real possibility has arisennot only conduct accurate molecular diagnostics, but also predict a person’s susceptibility to a particular disease [19, 25]. Association studies are based on searching for population correlations in allele frequencies. Classically, they represent a comparison of sick individuals with healthy individuals from the same population. A genetic marker is considered associated with a disease if its frequency of occurrence amongpatients is significantly higher than in the control sample. It is well known that timely prevention and early diagnosis of various diseases are the most pressing problems of modern medicine. It is noteworthy that the use modern advances in the study of the human genome in clinical cardiology have made early, presymptomatic diagnosis of not only genetic diseases, but also many multifactorial diseases possible [25].

In clinical practice, this goal is achieved through molecular testing of genes called “susceptibility” genes or candidate genes [25, 26]. The latter can be defined as genes whose hereditary polymorphisms are compatible with life, but in combination with unfavorable external factors (for example, drugs, food, bad habits, infections, environmental pollution) can cause various, including such common pathological conditions and diseases as atherosclerosis, ischemic heart disease, diabetes mellitus, etc.

Thus, from the above data it follows that this direction in cardiology is subject to intensive study and research in order to identify genetic risk and predict complications of the disease before the appearance of clinical manifestations. Taking into account modern advances in the study of the pathogenesis of CHF, it can be assumed that polymorphisms of genes encoding proinflammatory cytokines, in particular the cytokines TNF-α and IL-1, influence the development and progression of CHF. To understand the mechanisms of development and progression of CHF, scientific research in recent years has been aimed at assessing the genetic factors in the development of this syndrome. This is a promising approach due to the fact that genetic risk is identified and disease complications are predicted before the onset of clinical manifestations. Taking into account modern advances in the study of the pathogenesis of CHF, the purpose of our work was to study the influence of polymorphic variants of the TNF-α gene, IL-1α and the IL-1Ra gene, as well as the level of TNF-α in the serum on the development and nature of the course of CHF in patients with coronary artery disease and AG.

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