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FEATURES OF THE MODERN CLINICAL COURSE OF CHRONIC HEART FAILURE

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Abstract

Despite significant advances in the treatment of cardiovascular diseases, the prevalence of chronic heart failure (CHF) is not only not declining, but is steadily increasing, with the growth in incidence resembling a non-communicable epidemic. The MONICA study, conducted on a large, unorganized population, showed a CHF prevalence of 2%. A study of city residents over 50 years old, conducted in Rotterdam, established a CHF prevalence of up to 4%. In the Russian population-based EPOCHA-CHF study, the increase in CHF prevalence by clinical criteria was more than 4%, especially in older age groups, reaching 9.7%. According to Euro-Heart Survey The study considers the main causes of CHF development to be: ischemic heart disease (IHD) - 60%, valvular heart defects - 14%, dilated cardiomyopathy - 11%, etc. Many independent nosological forms or pathological conditions^ are currently considered as risk factors for CHF. Identification of modifiable and non-modifiable risk factors for CHF, a modern understanding of its pathogenetic mechanisms have allowed us to formulate the phenomenological concept of a "cardiovascular continuum". The essence of the phenomenon is that risk factors for coronary heart disease through hypertrophy and dysfunction of the left ventricle (LV) myocardium, or through the development of stenotic coronary atherosclerosis, myocardial ischemia and acute myocardial infarction lead to the death and hibernation of cardiomyocytes , activation of apoptosis and, as a result, to cardiac remodeling and the development of CHF. The cardiovascular continuum is characterized by the fact that, from a certain stage of cardiac damage, myocardial remodeling progresses with the development of heart failure according to general patterns independent

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of the etiologic factor. An important factor in maintaining normal cardiovascular function is the timely prevention of cardiac disease through early and reliable identification of potential risk factors for the onset and development of pathological myocardial changes.

Pre-symptomatic diagnosis feasible, not only for genetic diseases but also for many multifactorial diseases. The "geneticization" of medicine has led to the emergence of molecular medicine. This, in turn, has given rise to new areas of medical science, one of which is predictive medicine, which appropriately considers the earliest stage of a person's active influence on their body with the goal of timely correction of a potential pathology or pathological process. When studying the genes involved in the development of CHF, of primary interest is the study of gene polymorphisms of components of the SAS and RAAS systems, which play a leading role in the pathogenesis of both underlying diseases (hypertension, coronary heart disease, myocardial infarction, type 2 diabetes mellitus, etc.) and CHF itself. However, recent studies have shown that, despite improvements in the clinical condition of patients and a reduction in cardiovascular risk with the use of inhibitors of these systems, CHF continues to progress. In this regard, the influence of immune activation and systemic inflammation on the progression of heart failure is currently being actively studied. Proinflammatory cytokines are recognized as the most significant components of this activation. A whole group of genes involved in the development of heart failure is currently being studied: these include genes encoding aldosterone synthase , angiotensin-converting enzyme, tumor necrosis factor, adrenergic receptors , atrial natriuretic peptide, and endothelial synthesis. Data from case-control studies are highly variable. A genotype that is predominant in one population may be minor in another, making research for each ethnic and population group unique and meaningful.

The aim of the study was to investigate the clinical and molecular genetic aspects of the neurohumoral mechanisms of initiation and development of chronic heart failure in patients with coronary artery disease to improve the effectiveness of risk stratification, prevention, and drug therapy.

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Research Objectives

1. To assess the role of genes of the neurohumoral and cytokine systems in the mechanisms of initiation of chronic heart failure of ischemic genesis in order to identify genetic determinants of an increased risk of developing this pathology.
2. To identify candidate genes that determine the severity of CHF in patients with coronary heart disease.
3. During a 12-month prospective observation, study the relationship between polymorphic variants of genes and the nature of the course of CHF in patients with coronary heart disease in order to identify early objective predictors of CHF progression.

Study Results

According to Russian and global statistics, arterial hypertension (AH) complicates 4-8 to 29% of pregnancies, which is almost a third more than 10-15 years ago [1,2]. The concept of AH in pregnant women encompasses various clinical and pathogenetic forms that differ significantly not only in epidemiological characteristics but also in the risk of an unfavorable prognosis for the mother and fetus [3,5]. Thus, AH is one of the leading symptoms of preeclampsia (PE), the incidence of which has increased by 40% since the 1990s and amounts to 2-7% among healthy primiparous women [9]. About 15-30% of maternal deaths in developed countries are directly related to PE, which also ranks first among the factors of perinatal morbidity and mortality [1,6]. It has been established that 1-5% of pregnancies occur against the background of chronic arterial hypertension (CAH), which often end favorably. However, the probability of developing PE in patients with CAH is 5 times higher, the risk of premature detachment of a normally located placenta is 3 times higher, and the risk of premature birth and the birth of low birth weight infants is 50% higher than in pregnant women with normal blood pressure (BP) [3,4].

Both PE and CAH are multifactorial diseases with a high heritability coefficient, accounting for up to 50% of cases of these diseases [1,5]. A range of candidate genes associated with each form of hypertension has been identified; however, the reproducibility of the results of single-locus association studies in different

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populations is impossible [7, 13]. In this regard, the genetic mechanisms of inheritance of PE and CAH, as well as the role of intergenic interactions in the formation of a predisposition to these forms of hypertension in women living in the central part of Russia and in the Tver region in particular, remain unexplored. Circulating cell-free fetal DNA in plasma is a new biomarker for prenatal diagnosis [2]. Increased concentrations of fetal genetic material in maternal blood are associated with mechanisms of abnormal chorionic villus invasion or impaired fetal DNA clearance, which is associated with PE [5, 8]. However, the significance of cell-free fetal DNA in maternal plasma in relation to clinical and pathogenetic associations in PE and CAH has not been studied.

In PE, local placental ischemia associated with abnormal chorionic villus invasion triggers a mechanism of total endothelial dysfunction, whereas patients with CAH can maintain the adaptive potential of vascular reactivity throughout pregnancy [3]. However, the role of the vascular tone regulatory apparatus in the formation of gestational features of endothelial vasomotor function in PE and CAH, and how the genetic component of predisposition to these diseases modifies vascular reactivity indicators in pregnant women remains unknown.

The renin- angiotensin - aldosterone system (RAAS) plays a role in the mechanism of development and progression of hypertensive disorders in pregnant women. Thus, there is evidence of RAAS involvement in disruption of uteroplacental blood flow, initiation of endothelial dysfunction and hypercoagulation, electrolyte imbalance, and direct influence on cardiac function [2,6,9]. In addition, a significant number of candidate genes encoding RAAS structures are associated with PE and CAH in different populations [3,5]. However , very little is known about how RAAS activity changes in various forms of hypertension in pregnant women and how the level of its factors is associated with sodium excretion, heart rhythm regulation parameters, and circadian blood pressure profile (CBP) under PE and CAH. At the same time, there is no information on the pathogenetic significance of genetic polymorphism in the implementation of RAAS effects depending on the form of hypertension syndrome in pregnant women.

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Positive experience has been accumulated in the prevention of cardiovascular diseases by limiting table salt intake to reduce the risk of hypertension [3,4]. However, whether preventive interventions to reduce the incidence and severity of hypertension in pregnant women by prescribing a low-salt diet can be justified, and how this conclusion can be substantiated from the standpoint of genotypic and phenotypic analysis of RAAS functioning and daily sodium excretion, requires detailed study. Until recently, the leading role among the causes of CHF was assigned to coronary heart disease (CHD), followed in descending order by heart defects, myocarditis, pericarditis, and cardiomyopathy. These diseases are in most cases accompanied by a decrease in systolic function of the heart. The situation changed significantly when research revealed that more than half of CHF cases are associated predominantly with impaired diastolic function of the heart while maintaining its contractility. Moreover, it turned out that diastolic dysfunction of the heart, as a rule, precedes a decrease in the ability of the myocardium to fully contract, i.e. it occurs in the initial stages of CHF [3]. A classic example of diastolic dysfunction of the heart is arterial hypertension (AH), which, according to a recent ESSE-RF study, occurs in 44% of the population over 15 years of age in Russia [4]. Thus, currently, hypertension (HTN) without the presence of ischemic heart disease (CHD) is the cause of CHF development in 40-50% of cases. The Recommendations adopted by the Russian Medical Society on Arterial Hypertension (RMSAH) indicate that the presence of CHF stage I according to the classification of V.Kh. Vasilenko - N.D. Strazhesko is the basis for diagnosing HTN stage II, and CHF stages II-III – HTN stage III [5].

Conclusions

In patients with coronary heart disease with manifested CHF, a 12-month course of therapy with carvedilol , bisoprolol , fosinopril and enalapril is effective in reducing blood pressure, decreasing heart rate and NYHA functional class of CHF, increasing LVEF and regressing ischemic remodeling of the heart against the background of wide variability in the effectiveness of the classes of drugs used depending on the studied genotypes - homozygotes Arg / Arg polymorphisms of the Gly389Arg locus of the ß- adrenergic receptor gene are

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more sensitive to carvedilol therapy than heterozygotes Gly/Arg , and in carriers of the D/D genotype of the I/D polymorphic marker of the ACE gene, treatment of CHF with fosinopril and enalapril was more effective compared to carriers of the I/I genotype. No associations were found between the gene polymorphism and the effectiveness of therapy in patients with CHF with carvedilol and bisoprolol , between the ßi-adrenoreceptor gene polymorphism and the effectiveness of bisoprolol , or between ATG and AT2P1 polymorphisms and the effectiveness of treatment with fosinopril and enalapril .

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