Modern American Journal of Medical and Health Sciences
ISSN (E): 3067-803X
Volume 01, Issue 08, November, 2025
Website: usajournals.org
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FEATURES OF THE MODERN CLINICAL COURSE OF CHRONIC HEART FAILURE

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Abstract

Relevance of the problem. 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 of age found a CHF prevalence of up to 4%. In the EPOCHA-CHF population-based 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 Study of the main causes of CHF development consider: ischemic heart disease (IHD) - 60%, valvular heart defects - 14%, dilated cardiomyopathy - 11%. 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 and a modern understanding of its pathogenetic mechanisms have allowed the formation of the phenomenological concept of a "cardiovascular continuum." The essence of the phenomenon is that risk factors for coronary heart disease through hypertrophy and myocardial dysfunction of the left ventricle (LV), or through the development of Stenosing coronary atherosclerosis, myocardial ischemia, and acute myocardial infarction lead to the death and hibernation of cardiomyocytes , activation of apoptosis , and, ultimately, cardiac remodeling and the development of CHF. The characteristics of the cardiovascular continuum are that, from a certain stage of cardiac damage, the progression of myocardial

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remodeling and the development of CHF occur according to general patterns independent of the etiologic factor.

An important factor in maintaining the normal functioning of the cardiovascular system is the timely prevention of the development of cardiac diseases through early and reliable determination possible risk factors for the occurrence and development of pathological changes in the myocardium. Pre-symptomatic diagnosis feasible, not only for genetic diseases but also for many multifactorial ones. 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 is appropriately considered the earliest stage of a person's active intervention in their body, with the goal of timely correction of a potential pathology or pathological process. When studying 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. Therefore, the influence of immune activation and systemic inflammation on the progression of CHF is currently being actively investigated. Proinflammatory cytokines are recognized as the most significant components of this activation. It has long been proven that in the development of cardiovascular diseases in general and CHF in particular, a special place is occupied by endothelial dysfunction, which participates in the development of pathology through an increase in the activity of tissue and circulating RAAS and SAS, suppression of the expression of endothelial NO-synthase, which leads to a chronic decrease in blood flow and a distortion of the vascular response to "shear stress", an increase in the level of proinflammatory cytokines, an increase in the concentration of free radicals, an increase in low-density and very low- density lipoproteins .

Currently, a whole group of genes involved in the development of CHF is being studied. These include genes encoding aldosterone synthase, angiotensin-

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converting enzyme, tumor necrosis factor, adrenergic receptors, atrial natriuretic peptide, and endothelial Synthase. Data from case-control studies are highly variable. A genotype that is predominant in one population may be minor in another, making studies for each ethnic and population group unique and significant. Advances in medical science and the introduction of a huge number of new drugs do not diminish the relevance of the problems of effective and safe pharmacotherapy for patients with various cardiovascular pathologies. It is clear that one of the ways to improve the effectiveness and safety of pharmacotherapy is the introduction of so-called personalized medicine technologies into clinical practice. These technologies are based on an individual approach to the selection of drugs and their dosage regimen, taking into account factors influencing the pharmacological response and the individual characteristics of each specific patient; one of the current areas of genetic research is pharmacogenetics. A number of the largest pharmacogenetic studies have identified significant differences in the effectiveness of CHF therapy depending on the genetic characteristics of the patient. Therefore, clinical pharmacogenetics provides the opportunity to individualize the choice of IgGs and their dosage regimens based on the study of the genotype of a particular patient.

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.

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 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

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with coronary heart disease in order to identify early objective predictors of CHF progression.

Results of the Study

It was established for the first time that a high risk of developing CHF of ischemic genesis is associated with polymorphisms of the CT gene. In this study, a relationship between polymorphisms of the IL-1 gene and the severity of clinical manifestations of CHF in patients with ischemic heart disease was revealed for the first time. In patients with coronary heart disease, genetic determinants that determine the nature of the course of CHF were identified for the first time: predictors of an unfavorable course of pathology - the G allele and the G/G genotype of the TNF-α gene (G-308A), the C allele and the G/C genotype of the gene. As a result of this study, associations between GT polymorphisms of the IL- 1β gene and endothelial dysfunction were identified for the first time. The pharmacogenetic features of β- blockers (carvedilol and bisoprolol), AIF inhibitors (fosinopril and enalairil) in the treatment of CHF were studied . It was established that in carriers of the D/D genotype of the I/D polymorphic marker; ACE gene, treatment of CHF with fosinopril and enalapril was more effective compared to carriers of the 1/1 genotype, and homozygotes Arg / Arg polymorphisms of the Gly389Arg locus of the ßj-adrenergic receptor gene are more sensitive to carvedilol therapy than heterozygotes Gly / Arg.

The study of polymorphic loci of different groups of genes regulating neurohumoral and cytokine status and the functional state of the endothelium allows the use of genetic markers as criteria for assessing the individual prognosis of the development and course of CHF in patients with coronary heart disease.

These prognostic indicators allow us to identify high-risk groups for developing heart failure in the setting of chronic myocardial ischemia (taking into account identified gender differences) and to predict the severity and course of heart failure. This enables an individualized approach to high-tech modern diagnostics, disease prevention, and treatment based on a personalized medicine strategy aimed at improving quality of life and survival. Genetic markers have been substantiated, the typing of which is necessary for identifying patients at high risk

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for developing heart failure in patients with ischemic and post-infarction myocardial dysfunction.

It is advisable to use polymorphic variants of the genes of interleukin, tumor necrosis factor, endothelial NO synthase, inducible NO synthase, aldosterone synthase, atrial natriuretic peptide, ßi-adrenergic receptor, angiotensinogen, angiotensin-converting enzyme (I/D) and angiotensin 2 receptor type 1 as diagnostic markers.

Polymorphisms of the genes IL- 1ß with aldosterone synthase, ßi-adrenoreceptor predetermine neurohumoral mechanisms of initiation and formation of XGH, developed against the background of ischemic and/or post-infarction myocardial dysfunction.

In patients with coronary heart disease (CHD) with ischemic and post-infarction myocardial remodeling with systolic dysfunction; myocardium, interconnected; with CHF, an increased risk of developing; heart failure, associated with: carriage of the G allele and the G/G genotype of the G polymorphic locus of the TNF-a gene, the C allele and the Ç/G genotype of the CT polymorphic locus of the IL-1ß gene , the Glut and the Glu / Glu genotype , the eNOS gene polymorphic locus ; alleles and the T/T polymorphic locus of the gene, ATE, the D allele and the D/D genotype of the I/D polymorphic locus, the ACE gene, the C allele and the A/ C genotype of the ATP1 gene polymorphic locus.

Pathogenetic mechanisms of development of heart failure in conditions of chronic myocardial ischemia and/or post-infarction cardiac remodeling are associated with wide variability in the occurrence of genetic markers of CHF depending on gender: in men, the Glu allele and the Glu / Glu genotype of the eNOS gene , the iNOS gene allele , the D allele and the D / D genotype of the ACE gene were associated with an increased risk of developing the disease; and in women - the G allele and the A / C genotype of the gene. Polymorphisms of the TNF-a, IL-1ß, aldosterone synthase , ßi-adrenoreceptor , ATG genes are associated with the risk of developing CHF in conditions of chronic myocardial ischemia in both men and women.

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Conclusions

In patients with CHF against the background of chronic myocardial ischemia, the severity of endothelial vasomotor function disorders depended on the FC of CHF severity. In patients with FC I CHF (7.4±0.35% and 16.5±0.29%) and FC II (6.8±0.29% and 15.2±0.23%), significantly higher levels of EZD and ENZD were determined ($p< 0.01$ and $p<0.05$, respectively) in relation to those in patients with FC III-IV CHF (5.5±0.19% and 12.7±0.27%).

The severity of endothelial dysfunction is associated with polymorphisms of the studied genes in patients with coronary heart disease complicated by CHF. The most pronounced endothelial dysfunction was recorded in carriers of the C/ C genotype of the IL- 1ß gene (C+3953T), the Glu / Glu genotype of the eNOS gene, alleles of the iNOS gene, the G/G genotype of the endothelin-1 gene (K148N), the Gly / Gly genotype of the ßi-adrenoreceptor gene, the D/D genotype of the ACE gene (I/D) and the C/C genotype of the AT2P gene. In CHF against the background of ischemic and/or post-infarction myocardial remodeling with obvious disturbances4 of the inotropic function of the heart and structural changes in the LV, there is an association with the carriage of the C/ C genotype of the proinflammatory cytokine IL - 1ß gene, alleles of the iNOS gene, the T/T genotype of the aldosterone synthase gene, the Gly / Gly genotype of the ßi-adrenoreceptor gene , and the D/D genotype of the ACE gene (I/D).

In patients with coronary artery 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 drug classes used depending on the studied genotypes - homozygotes Arg / Arg polymorphisms of the Gly389Arg locus of the ßi-adrenergic receptor gene are more sensitive to carvedilol therapy than heterozygotes . Gly / Arg , and in carriers of the D/D genotype »polymorphic marker I/D of the ACE gene Treatment of CHF with fosinopril and enalapril was more effective compared to carriers of the I/I genotype.

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