WEB OF SCIENTIST: INTERNATIONAL SCIENTIFIC RESEARCH JOURNAL
ISSN: 2776-0979, Volume 4, Issue 1, Jan., 2023

CHRONIC HEART FAILURE IN CORONARY HEART DISEASE

Naimova Sh.A.,
[1]Department of Hematology and Clinical Laboratory Diagnostics,
Bukhara State Medical Institute, Bukhara, Uzbekistan

Rakhimov N. A.
[2]Bukhara Regional Multidisciplinary Medical Center, Uzbekistan

Annotation
Heart failure is a complex syndrome responsible for high mortality and hospitalization rates. Ischemic heart disease is one of the most common causes of heart failure and is usually associated with coronary heart disease, defined by the presence of one or more obstructive plaques that cause a decrease in coronary blood flow, causing myocardial ischemia and, as a result, heart failure. Coronary artery disease (CHD) determines a decrease in the supply of oxygen to the myocardium, which causes a violation of the contraction and relaxation of the myocardium. In this regard, "modern" cardiology pays great attention to the study of epicardial atheromatous plaque, its etiology, prevention, diagnostic and therapeutic interpretation.

Keywords: Heart failure, ischemic heart disease, international classification of diseases, causes of death

ХРОНИЧЕСКАЯ СЕРДЕЧНАЯ НЕДОСТАТОЧНОСТЬ ПРИ ИШЕМИЧЕСКОЙ БОЛЕЗНИ СЕРДЦА

Наимова[1] Ш.А., Рахимов[2] Н.А.
[1]Кафедра гематологии и клиническая лабораторная диагностика
Бухарского государственного медицинского института,
Бухара, Узбекистан
[2] Бухраский Областной Многопрофильный Медицинский Центр,
Узбекистан

Аннотация
Сердечная недостаточность является сложным синдромом, ответственным за высокие показатели смертности и госпитализации. Ишемическая болезнь сердца является одной из наиболее частых причин сердечной недостаточности и обычно связана с ишемической болезнью сердца, определяемой наличием одной или нескольких обструктивных бляшек,

которые определяют снижение коронарного кровотока, вызывая ишемию миокарда и, как следствие, сердечную недостаточность. Ишемическая болезнь сердца (ИБС) определяет снижение снабжения миокарда кислородом, что вызывает нарушение сокращения и расслабления миокарда. В связи с этим «современная» кардиология уделяет большое внимание изучению эпикардиальной атероматозной бляшки, ее этиологии, профилактике, диагностической и терапевтической интерпретации.

Ключевые слова: Сердечная недостаточность, ишемический болезнь сердца, международная классификация болезней, причины смерти

YURAK ISHEMIK KASALLIGIDA SURUNKALI YURAK YETISHMOVCHILIGI

Naimova[1] Sh.A.
Rakhimov[2] N.A.
[1]Gematologiya va klinik laboratoriya diagnostikasi bo'limi Buxoro davlat tibbiyot instituti, Buxoro, O‘zbekiston
[2]Buxoro Viloyat Ko’p Tarmoqli Tibbiyot Markazi, Buxoro, O’zbekiston

Annotatsiya
Yurak yetishmovchiligi yuqori o'lim va kasalxonaga yotqizish ko'rsatkichlariga oshishiga sabab bo’ladigan murakkab sindromdir. Ishemik yurak kasalligi yurak yetishmovchiligining eng keng tarqalgan sabablaridan biri bo'lib, odatda koronar yurak kasalligi bilan bog'liq bo'lib, koronar qon oqimining pasayishiga olib keladigan bir yoki bir nechta obstruktiv pilakchalarning mavjudligi bilan belgilanadi, bu esa miokard ishemiyasini keltirib chiqaradi va natijada yurak etishmovchiligi rivojlanadi. Yurak ishemik kasalligi (YIK) miokardning qisqarishi va kengayishining buzilishiga olib keladigan kislorod bilan ta'minlanishining pasayishini aniqlaydi. Shu munosabat bilan "zamonaviy" kardiologiya epikardial ateromatoz pilakcha, uning etiologiyasi, profilaktikasi, diagnostik va terapevtik talqinini o'rganishga katta e'tibor beradi.

Kalit so'zlar: Yurak yetishmovchiligi, yurak ishemik kasalligi, kasalliklarning xalqaro tasnifi, o'lim sabablari

Despite significant advances in the treatment of cardiovascular diseases, the prevalence of chronic heart failure (CHF) not only does not decrease, but also steadily increases, the increase in the incidence of which resembles a non-communicable epidemic. Ischemic heart disease is one of the most common causes of heart failure and is usually associated with coronary heart disease, defined by the presence of one or more obstructive plaques that cause a decrease in coronary blood flow, causing myocardial ischemia and, as a result, heart failure. However, coronary obstruction is only part of a complex pathophysiological process leading to myocardial ischemia. There is increasing attention in the literature to the role of microcirculation in the pathophysiology of coronary heart disease and heart failure. Coronary microvascular dysfunction determines the inability of the coronary circulation to meet the metabolic needs of the myocardium due to an imbalance in the mechanisms of regulation of coronary blood flow, including ion channels, which leads to the development of hypoxia, fibrosis, and tissue death, which can cause loss of myocardial function, even beyond the presence of atherosclerotic epicardial plaques. For this reason, ion channels may represent a link between coronary microvascular dysfunction, coronary heart disease, and subsequent heart failure. The MONICA study, conducted on a solid unorganized population, showed the prevalence of CHF - 2%. A study of residents of cities over 50 years old, conducted in Rotterdam, established the prevalence of CHF - up to 4%. In a different population study EPOCHA-CHF according to clinical criteria, the increase in the prevalence of CHF was more than 4%, especially in older age groups, reaching 9.7%.

An important factor in maintaining the normal functioning of the cardiovascular system is the timely prevention of the development of cardiac diseases by early and reliable determination of possible risk factors for the occurrence and development of pathological changes in the myocardium.

Heart failure (HF) is a complex syndrome responsible for high rates of death and hospitalization in the general population worldwide. One of the most common causes of HF is coronary heart disease (CHD), which leads to loss of myocardial tissue and contractility [1]. Coronary heart disease (CHD) determines a decrease in myocardial oxygen supply, which causes a violation of myocardial contraction and relaxation [2,3]. In this regard, "modern" cardiology pays great attention to the study of epicardial atheromatous plaque, its etiology, prevention, diagnostic and therapeutic interpretation. As early as the 1970s, the effects of progressive narrowing due to stenosis on coronary blood flow at rest and at peak levels were described [4]. In fact, a decrease in the diameter of a coronary artery by ≥50% limits its maximum vasodilating capacity, and a decrease by ≥85% determines a decrease in blood flow

even at rest. The pathophysiological continuum between epicardial coronary artery obstructive atherosclerosis, myocardial ischemia, and HF is now well defined. Angiographic data confirmed the relationship between the severity and prevalence of coronary atherosclerotic lesions and survival [5]. However, several studies in the literature suggest that coronary obstruction is only an element of a complex multifactorial pathophysiological process leading to myocardial ischemia [6]. In addition, it is also known that impairments in the function and structure of the coronary microvasculature are associated with various clinical conditions [7,8]. In clinical practice, insufficient attention is paid to the coronary microcirculation and its pathophysiological role. What actually is this vasculature, consisting of coronary arterioles with a diameter of 50 to 200 microns? Should a cardiologist take this into account when making difficult decisions?

CAD, and CAD in particular, is a major cause of HF [9,10]. However, CAD, and in particular the presence of atherosclerotic plaque in the epicardial coronary arteries, does not always define myocardial ischemia and, on the other hand, myocardial ischemia is not always justified by the presence of atherosclerotic plaque. In patients with CAD, systolic myocardial dysfunction has been described as the main pathophysiological mechanism associated with HF [11,14]. Classically CAD patients who develop HF have a history of myocardial infarction with atherosclerotic lesions of the epicardial arteries, as confirmed by coronary angiography [12,15]. However, the absence of atherosclerotic plaques according to coronary angiography cannot exclude the presence of coronary microvascular dysfunction (CMD) as a pathophysiological mechanism of HF. It is assumed that diastolic dysfusia predominates in these patients. function [13]. Moreover, AMD may represent a pathophysiological substrate for left ventricular diastolic dysfunction [16,19].

In the literature, a growing number of studies emphasize the central role of AMD in the pathophysiology of CAD and HF, in addition to atherosclerotic disease [3,4,8,9,19]. Moreover, AMD is one of several pathophysiological mechanisms that can cause type II myocardial infarction. CMD arises from impaired microvascular endothelial and non-endothelial adaptation of CBF to the metabolic needs of the myocardium and may be associated with myocardial ischemia independently of CAD [17,18]. Dysregulation of mediators of CBF regulation, such as coronary ion channels, can lead to CMD. In addition, CMD, which alters the hemorheological characteristics of CBF, may contribute to the development of atherosclerotic plaques in epicardial vessels by increasing shear stress and long-term exposure of the coronary vessel wall to low-density lipoprotein (LDL), ROS, inflammatory mediators, and enhanced glycation completion. -products (AGEs). There are several methods by which CMD

can be assessed. Transthoracic echocardiogram, cardiac magnetic resonance imaging, and positron emission tomography (PET) can be used for non-invasive evaluation of CFR, while coronary angiography can be used for its invasive evaluation [18]. During coronary angiography, intracoronary administration of acetylcholine and adenosine can be used to assess endothelial-dependent and independent vasodilation, respectively. CMD is defined by CFR<2.0. Over the past few years, several authors have hypothesized a central role for AMD in the pathophysiology of HF and myocardial remodeling.

Moreover, other authors suggest an association between CMD via endothelial dysfunction and symptom severity in patients with HF [12,13]. According to the ALLAHAT study and the European HF registry MEDIA (The Metabolic Road to Diastolic Heart Failure), these patients often have a high body mass index, and cardiovascular risk factors such as arterial hypertension, diabetes mellitus and dyslipidemia can lead to HF through microcirculatory dysfunction. Endothelial dysfunction is associated with reduced NO bioavailability and reduced KATP activity. In addition, CMD stimulates cardiomyocyte hypertrophy, fibrosis, and microvascular thinning, which are the main histological changes observed in HF [12,14,15].

In addition, Paulus et al. [14] and Franssen et al. [17] focused on the possible role of AMD in the pathophysiology of HF. They identified a possible sequence of events that could lead to heart failure. All cardiovascular risk factors contribute to a systemic pro-inflammatory state. Patients with HF have high blood levels of tumor necrosis factor alpha (TNF-α), interleukin 6 (IL-6), pentraxin 3, and ST2. However, systemic inflammation is not a predictive risk factor for HF. In the microvasculature, an inflammatory condition, as well as each risk factor itself, can cause increased production of ROS, increased expression of chemokines and selectins such as vascular cell adhesion protein 1 (VCAM-1) and E-selectin, and impaired mitochondrial function. and reduced NO availability [17].

Adaptation of myocardial blood flow to various metabolic conditions is essential for normal myocardial function. This adaptive process requires a complex system of factors. Several mechanisms are involved in the regulation of myocardial blood flow, including metabolic and neurohumoral factors, as well as physical influences such as changes in intraluminal pressure or effects caused by shear stress on the vessel wall. In this context, the role of coronary ion channels is critical in matching CBF to metabolic needs. Because of their role in repolarization in coronary vascular cells (endothelial and smooth muscle), changes in ion channel expression or activity often result in abnormalities in vascular tone. Thus, pathophysiological conditions characterized by the development of vascular hyperactivity, including arterial

hypertension, dyslipidemia, diabetes mellitus, and genetic variations such as mutations or polymorphisms, can lead to changes in the expression or function of coronary ion channels. In addition, ROS-induced abnormalities in the activity of these channels during diabetes-induced oxidative stress cause dysfunction in vascular resistance control. This damages the regulatory system dependent on myocyte metabolism, which inevitably leads in the long term to the development of coronary artery microcirculation dysfunction and myocardial insufficiency. This can be considered a new paradigm in the pathophysiology of HF, in which the inability of the coronary circulation to meet the metabolic needs of the heart due to dysfunction of the microcirculation and mechanisms of its regulation, including ion channels, leads to the development of hypoxia, fibrosis and tissue death, which ultimately leads to loss of myocardial function, even beyond the atherosclerotic epicardial plaque [1-13]. In addition, modern therapies for HF, such as beta-blockers, ACE inhibitors, and aldosterone antagonists, reduce myocardial oxygen demand and reduce the effect of metabolic vasodilation dysfunction. The imbalance between supply and demand for oxygen, due to changes in the microcirculation of the heart and coronary ion channels, is even more evident when the load on the heart is high. Thus, pharmacological interventions that can reduce cardiac performance also minimize the reduction in microcirculatory dysfunction and are likely to slow the progression of the disease. In recent years, the attention of scientific literature has been drawn to the study of coronary microcirculation and its regulators, including ion channels, and also taking into account the pathophysiological continuum that links microcirculatory dysfunction with coronary artery disease and heart failure. However, further research is needed to shed light on this intriguing but still unexplored aspect. The significance of the influence of chronic heart failure (CHF) on the magnitude of mortality rates is ambiguous. Much depends on approaches to its definition, the ability to record cases of complications, and the quality of filling in the MSA. Currently, in ICD-10, the rules for determining and placing SNs in the MSC are described in Volume 2, but are not clear enough. On the one hand, ICD10 Volume 2 states that “other cardiac conditions must be accepted as an obvious cause of HF” (i.e., HF cannot be PPP), and on the other hand, there is no explanation in which cases HF can be an immediate cause. cause of death (NPD), “an intermediate cause or fatal complication of PPP, since there is a recommendation to abandon the use of codes I50.- (SN), in cases of a terminal or acute, sudden ... condition of short duration” [3]. Therefore, each specialist can interpret such recommendations as he understands it; moreover, volume 2 is intended for “coders” (trained health statisticians), not physicians.

The National guidelines for determining the risk and prevention of sudden cardiac death indicate that the main causes of death from cardiovascular diseases are the progression of CHF (50% of all deaths) and sudden cardiac death (SCD) (50%) [4]. However, there are no references to studies in which such statistics would be confirmed, and there are no other data based on MSS on the frequency of CHF as NPS, an intermediate cause or a fatal complication in CAD. Based on the foregoing, the study of the frequency of registration of HF and CHF based on MSS data with coronary heart disease (CHD) as PPS is important for understanding the causes of death at the population level, especially in multimorbid pathology, planning and organizing preventive and therapeutic measures aimed at to reduce mortality rates.

Список литературы

1. Edelmann F., Gelbrich G., Düngen H.D., Fröhling S., Wachter R., Stahrenberg R., Binder L., Töpper A., Lashki D.J., Schwarz S., et al. Exercise training improves exercise capacity and diastolic function in patients with heart failure with preserved ejection fraction: Results of the Ex-DHF (Exercise training in Diastolic Heart Failure) pilot study. J. Am. Coll. Cardiol. 2011;58:1780–1791. doi: 10.1016/j.jacc.2011.06.054.
2. Kohr M.J., Davis J.P., Ziolo M.T. Peroxynitrite increases protein phosphatase activity and promotes the interaction of phospholamban with protein phosphatase 2a in the myocardium. Nitric Oxide. 2009;20:217–221. doi: 10.1016/j.niox.2009.01.003.
3. Calderone A., Thaik C.M., Takahashi N., Chang D.L., Colucci W.S. Nitric oxide, atrial natriuretic peptide, and cyclic GMP inhibit the growth-promoting effects of norepinephrine in cardiac myocytes and fibroblasts. J. Clin. Investig. 1998;101:812–818. doi: 10.1172/JCI119883.
4. Severino P., Mather P.J., Pucci M., D'Amato A., Mariani M.V., Infusino F., Birtolo L.I., Maestrini V., Mancone M., Fedele F. Advanced Heart Failure and End-Stage Heart Failure: Does a Difference Exist. Diagnostics. 2019;9:170. doi: 10.3390/diagnostics9040170.
5. Бойцов С. А., Драпкина О. М., Зайратьянц О.В и др. Пути решения проблемы статистики сердечной недостаточности в клинической практике. Кардиология. 2020; 60(10):13-9. doi:10.18087/cardio.2020.10.n1039.
6. Бойцов С. А., Самородская И. В., Галявич А. С. и др. Статистическая, клиническая и морфологическая классификация ишемической болезни сердца — есть ли возможность объединения? Российский кардиологический журнал. 2017;(3):63-71. doi:10.15829/1560- 4071-2017-3-63-71.

7. Boltayev K., Shajanova N. Anemia associated with polydeficiency in elderly and senile people //Galaxy International Interdisciplinary Research Journal. – 2022. – Т. 10. – №. 2. – С. 688-694.
8. Naimova S. A. Principles of early diagnosis of kidney damage in patients of rheumatoid arthritis and ankylosing spondiloarthritis //British Medical Journal. – 2021. – Т. 1. – №. 1.
9. Наимова Н. Ш., Хамидова Н. К., Азамов Б. З. Особенности коагуляционного и клеточного гемостаза при ревматоидном артрите у лиц с сердечно-сосудистой патологией //Новый день в медицине. – 2019. – №. 2. – С. 219-222.
10. Наимова Ш. А., Латипова Н. С., Болтаев К. Ж. Коагуляционный и тромбоцитарный гемостаз у пациентов с ревматоидным артритом в сочетании с сердечно-сосудистом заболеваением //Инфекция, иммунитет и фармакология. – 2017. – №. 2. – С. 150-152.
11. Anvarovna N. S. Features Of Kidney Damage at Patients with Ankylosing Spondiloarthritis //Texas Journal of Medical Science. – 2021. – Т. 3. – С. 18-22.
12. Tulkinjanovna S. G., Anvarovich R. A. The influence of defiency of microelements in children with bronchial hyperreactivity// ACADEMICIA: An International Multidisciplinary Research Journal (ISSN: 2249-7137)–2020. April. - 2020. - Т. 10. - No. 4. - S. 846-853.
13. Boltayev K. J., Naimova S. A. Risk factors of kidney damage at patients with rheumatoid arthritis //WJPR (World Journal of Pharmaceutical Research). – 2019. – Т. 8. – №. 13.
14. Болтаев К. Ж., Ахмедова Н. Ш. Характеристика феномена развития полидефицитных состояний при старении //Проблемы биологии и медицины. – 2020. – №. 1. – С. 24-26.
15. Boltayev K. J., Ruziyev Z. M., Ulug'ova Sh T. FEATURES CHANGES IN THE HEMOSTASIS SYSTEM IN PATIENTS WITH COVID-19 //Web of Scientist: International Scientific Research Journal. – 2022. – Т. 3. – №. 5. – С. 479-486.
16. Naimova N. S. et al. Features of coagulation and cellular hemostasis in rheumatoid arthritis in patients with cardiovascular pathology //Asian Journal of Multidimensional Research (AJMR). – 2019. – Т. 8. – №. 2. – С. 157-164.
17. Наимова Ш. А. The degree of secondary osteoporosis in rheumatological patients and ways of its prevention //Новый день в медицине. – 2020. – №. 1. – С. 56-58.

18. Наимова Ш. А., Рузиева Ф. А. ОСОБЕННОСТИ ПОЧЕЧНОЙ КОМОРБИДНОСТИ ПРИ РЕВМАТОЛОГИЧЕСКИХ ЗАБОЛЕВАНИЯХ //Вестник науки и образования. – 2020. – №. 24-2 (102). – С. 74-78.
19. Калюта Т. Ю., Царева О. Е., Трубецков А. Д.и др. Ближайший и отдаленный прогноз у пациентов с нестабильной стенокардией и анемией. Кардиоваскулярная терапия и профилактика 2005; 2:46–51