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ПАТОГЕНЕТИЧЕСКИЕ АСПЕКТЫ РАЗВИТИЯ ХРОНИЧЕСКОЙ СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТИ С СОХРАНЕННОЙ ФРАКЦИЕЙ ВЫБРОСА

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АННОТАЦИЯ

Хроническая сердечная недостаточность (ХСН), являясь исходом сердечно-сосудистого континуума, приводит к ухудшению течения не только причин заболевания, но и к своей декомпенсации, регоспитализациям, что приводит к трудностям в курации этих пациентов и становится социально-экономической проблемой [1,2,11]. Распространенность ХСН в западных странах колеблется от 1 до 2 % в общей популяции, достигая 10% у лиц старше 70 лет [2, 38]. Данные исследования ЭПОХА, отображают появление СН в Российской Федерации в 7–10 % случаев [2,11]. В Узбекистане ХСН также занимает одно из первых мест среди всех осложнений заболеваний сердечно-сосудистой системы (ССС) [1,13].

Ключевые слова: Хроническая сердечная недостаточность, сердечная сосудистая система, фракция выброса.

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PATHOGENETIC ASPECTS OF CHRONIC HEART FAILURE WITH PRESERVED EJECTION FRACTION

ANNOTATION

Chronic heart failure (CHF), being an outcome of cardiovascular continuum, leads to worsening of the course not only of the disease cause, but also to its decompensation, rehospitalizations, which leads to difficulties in care of these patients and becomes a socio-economic problem [1,2,11]. The prevalence of CHF in Western countries ranges from 1% to 2% in the general population, reaching 10% in those over 70 years of age [2,38]. Data from the EPOHA study, show an incidence of CHF of 7-10% in the Russian Federation [2,11]. In Uzbekistan, CHF is also one of the leading complications of cardiovascular disease (CVD) [1,13].

Keywords: Chronic heart failure, cardiovascular system, ejection fraction.

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OTISH FRAKTSIYASI SAQLANIB QOLGAN SURUNKALI YURAK YETISHMOVCHILIGI RIVOJLANISHINING PATOGENETIK JIHATLARI

ANNOTATSIYA

Surunkali yurak yetishmovchiligi, yurak-qon tomirlar tizimi kasaliklarinig notug'ri va davolamaganliklarning natijasi bo'lib, u keyinchalik yurakning dekompensatsiyasini, regospitalizatsiyasini yomonlashishiga olib keladi, buningnatijasida esa ushbu bemorlarni boshqarishda qiyinchiliklarga olib keladi va ijtimoiy-iqtisodiy muammoga aylanadi [1,2,11]. G'arbiy mamlakatlarda surunkali yurak yetishmovchiligi tarqalishi umumiy populyatsiyada 1% dan 2% gacha, 70 yoshdan oshgan odamlarda 10% gacha [2,38]. Rossiya Federatsiyasida 7-10% hollarda surunkali yurak yetishmovchiligi paydo bo'lishini aks ettiradi [2,11]. O'zbekistonda surunkali yurak yetishmovchiligi kasalliklarining barcha asoratlari orasida birinchi o'rinni egallaydi [1,13].

Kalit so'zlar: surunkali yurak yetishmovchiligi, yurak qon tomir tizimi, otish fraktsiyasi.

The main etiologies of CHF in the Russian Federation, Europe and the USA are arterial hypertension (AH) (95.5%) and coronary heart disease (CHD) (69.7%), including more than half of patients with CHF. In Uzbekistan, valvular heart disease plays a role alongside the above causes [1,13]. Over the past 10 years, myocardial infarction (MI) (19.7%) and diabetes mellitus (22.7%) have been the "competing" causes of CHF [2,4,21]. The outcome and course depend on a variety of adverse factors, including anaemia [1,2,38).

One in two patients (49%) is hospitalised with decompensation and the diagnosis of CHF is made in 92% of hospitalised patients (2,38). This accounts for the high overall mortality of patients with CHF (□6%), which is 10 times higher than in the population (OR=10.1;p<0.0001) [2]. Life expectancy in patients with CHF I-IVF is 7.8 years, and among patients with CHF III-IVF - 4.8 years [2,21,38]. Studies have shown a clear association of impaired diastolic function with preserved contractility in more than half of CHF cases (3,30,31). Moreover, diastolic dysfunction (DD) of the heart usually developed before the decline in myocardial contractility, i.e. occurring in the early stages of CHF (2,4,33). One obvious variant of cardiac DD is AH; the ESSE-RF study confirms its presence in 44% of the population over 15 years of age in Russia [2,4]. Currently, AH alone is the cause of CHD in 40-50% of cases [5,10,23].

Epidemiological study conducted in Russia, among patients being treated with the diagnosis of CHF II-IV AC, EpOHA showed that 9% of patients had decreased VEF (nFVLV<40%), 20% had "intermediate" VEF (nFVLV 40-60%), and most patients (71%) had so called "hyperkinetic" type of reduction (sFVLV>60%) [7,10,11]. The prevalence of cFVLV was even higher - 78 %, among all patients with CHF I-IV FK [11]. Similar data on prevalence of CHFsFVLV in Russia (84.1%) were obtained in another population-based IMPRDVEMENT HF study (Russian part of the study) [5]. According to the results of the Russian CHF registry, patients with CHF class I-IV also prevailed in patients with FVLV (83%), while systolic LV dysfunction was noted only in 17% of patients [2,4,7,21]. Patients with CHFsFV are characterized by a significant decrease in exercise tolerance, frequent hospitalizations, and reduced quality of life [2,41]. The process of myocardial remodeling in CHFsFV differs from that in CHFsFV and includes 2 interrelated processes, representing the basis of VD [24]: 1) decreased elasticity and reduced relaxation of LV myocardium, which is due to an imbalance in the mechanical properties of cardiomyocytes [8,27,29]; 2) the state of the extracellular matrix [26]. The molecular basis of these changes is impaired calcium transport, regulation of fibrillar collagen synthesis and transformation, and changes in cardiomyocyte cytoskeleton due to increased expression of a stiffer isoform of the cardiac muscle stiffening sarcomeric titin protein [16,17,32].Massive studies have proven the role of immune inflammation in the development and progression of CHF and, in particular, CHFsFV [5,14,18,22]. The role of proinflammatory cytokines (interleukin (IL)-1, IL-6, TNF-α, etc.) involved in cardiac dysfunction and CHF progression has been identified [19,22]. Chemokines such as IL-8 are also involved in cardiac dysfunction and are defined as markers of tissue destruction (41). Adhesion molecules, autoantibodies, nitric oxide (NO), endothelin-1 and acute inflammatory proteins (C-reactive protein (CRP), fibrinogen, complement system) have also been shown to be involved in the pathogenesis of CHF [31,39,41].

The data collected support the current concept of interconnection and interdependence between such systems as sympathetic-adrenal (CAC), renin-angiotensin-aldosterone (RAAS), endothelin system, immune and inflammatory systems in the pathogenesis of CHF. They create an intricate, multidimensional network of cooperation, including different types of cells (monocytes, macrophages, T- and B - lymphocytes, endothelial cells) and biologically active substances [9,20,21,26].

One of the early triggers of CKD is hyperactivation of the CAC, which has a positive adaptation-compensatory effect in the early stages[9]. It provides pumping function to the heart by increasing heart rate and myocardial contractility, stabilises BP with reduced cardiac output (CV) by activating arteriolar constriction and induces venoconstriction to provide venous return and increase filling pressure in the heart [9,19]. In the early stages this will be a manifestation of compensatory systems to maintain contractile function of the heart. Increased SAS activity manifests as a positive inotropic and chronotropic effect on the heart, whereas the RAAS maintains vascular tone, blood pressure and circulating blood volume [2,9,19,36]. Over time, hyperactivation of the CAAS becomes detrimental and contributes to the progression of CKD due to extreme narrowing of veins and arterioles, which increases pre- and post-load and decreases tissue perfusion [9,36]. At the same time, norepinephrine (NA) increases circulating RAAS activity, leading to low cardiac output by retaining sodium salts, increasing circulating blood volume and increasing myocardial load. Chronic cardiac patients with severely elevated plasma catecholamine (CA) levels (especially with HA above 600 pg/ml) have been shown to have a much worse prognosis, with a 2.3-fold increase in mortality. Elevated HA levels determine a significant rearrangement of the myocardial receptor apparatus. Thus, the number of β1 receptors is significantly reduced. This process is known as down regulation [2,9]. It occurs because the receptors bind to HA molecules. In this case, a contradictory situation occurs: myocardial contractility is reduced in the presence of excess HA. An excess of HA circulating in the blood causes an increase in endothelial production of constrictor factors (endothelin, thromboxane A2, superoxide-anion, endoperoxide), thereby increasing the peripheral blood flow resistance, impairing microcirculation (especially in heart and kidney) and causing progressive remodeling of heart and vessels [5,9,10,19]. Thus, CAC hyperactivation promotes further myocardial hypertrophy and remodeling, development of diastolic and systolic LV dysfunction with CKD progression [2,9].Among a large number of biomarkers involved in CHD development, the most studied are natriuretic peptides (BNP), myocardial fibrosis markers, and biochemical markers of renal damage (renal dysfunction and ischemic damage). These include atrial ANP, urodilantin (isoform of ANP), brain BNP, C-type ANP (CNP) and D-type ANP [5,11,14]. The main biological essence of these neurohormones is to increase sodium excretion in the distal parts of the nephron. ANP and BNP are produced in response to myocardial dilatation under pressure or volume overload and are produced in atrial and ventricular myocytes [5,35]. In addition to natriuresis, ANP and BNP induce vasodilation, providing hemodynamic "unloading" of myocardium under adverse hemodynamic changes. In CHD, the protective effect of BNP is neutralized due to CAC and RAAS activation and increased sodium reabsorption in proximal nephron [15,20]. Thus, despite a significant increase in blood concentrations of these biomarkers, no natriuresis occurs. At the same time, blood ANP and BNP concentrations increase in proportion to the degree of haemodynamic overload of the heart chambers (both left and right) (5,35). In CHF, in general, the increase in ANP concentration reflects the level of haemodynamic disturbances and is associated with the incidence of adverse outcomes [2,5]. In CHF, NUPs are even more important as their increased concentration in the blood is one of the criteria for the diagnosis of doubtful EchoCG results [2,5,11,20].

Signs of LD (impaired active LV myocardial relaxation, decreased wall elasticity) were recommended only for the diagnosis of obvious

clinically pronounced dSN [12,19]. Zile et al. confirmed that all patients with CHFsFV showed LD on Doppler. However, they concluded that the sensitivity and specificity of these parameters are rather low for the diagnosis of CHFV [12,33]. This is due to the fact that EchoCG does not always identify the key passive component of diastole, as it is labile (Doppler wave peak amplitudes), associated with age, hypertension and other comorbid conditions. Some sources indicate that, even in the presence of striking symptomatic CSFV, many patients have not been confirmed with type 2 or type 3 DM on Doppler [5,12,33]. The presence of LD may not always be present or fully explain CSFV. In response to these findings, new diagnostic criteria for CHF have emerged that do not emphasise the mandatory presence of DM, LV and LV hypertrophy and increased BNP. In 2013, criteria based on several signs (presence of typical symptoms and signs of CH; normal or almost normal LVEF; absence of other reasons, including valve pathology, explaining CH symptoms) were proposed [2,5]. Further studies of patients with a history of CH and a PVLD greater than 50% have shown that many patients have moderate resting VD (21,23).

Arterial stiffness, as an independent marker of CVD, including CHF, and mortality, develops in the early stages of these diseases and progresses rapidly with disease duration [6,8,36,37]. Vascular wall transformations are accompanied by collagen accumulation and reduction of its elasticity, the conductive and damping function of vessels is impaired, the pulse wave velocity increases, the aortic root dilates and its stiffness increases [16,17,25,40]. As a result, there is an early return of the reflected wave in late systole, LV VA is formed, the postload increases, the need for oxygen increases, coronary perfusion is impaired, myocardial hypertrophy and microcirculatory disorders develop [16,17,25,34].

Numerous studies indicate increased arterial stiffness in CHF [16,17]. Increased arterial wall stiffness (AWS), assessed by an integral index of structural and functional state of arteries - pulse wave velocity (PWV), has been found to correlate with diastolic and systolic LV function and is considered as a predictor of CHF prognosis [29,30,32]. The results of several large studies have shown that the risk of cardiovascular events with an increase in aortic PWV by 1 m/c increases by 39%, and the increase in PWVcf by 1 m/c is associated with a 10% increase in the risk of death [6]. According to the Framingham criteria, PWV is an even stronger predictor of fatal and non-fatal cardiovascular complications (CVD) than smoking, glucose levels, total cholesterol and other biological markers [6].

An increase in HRV in elastic arteries greater than 11.5 m/c has been found to be a marker of poor prognosis in patients with CHF of coronary etiology [3,6,27]. Murego et al. [37] demonstrated that in patients with CHF, an increase in CPV at the brachial ankle segment was a reliable prognostic marker of rehospitalizations due to SA (PWVcf) [27,37]. A PWVcf value greater than 12 m/c is recognized as an independent predictor of prognosis in patients with AH and CHD [6,37]. Massive studies suggest that increased LV stress, vascular load and impaired ventriculo-vascular interaction are important links in the pathogenesis of CHF development, largely responsible for LV myocardial remodelling [6,29]. The increase of arterial stiffness leads to the increase of reflected wave velocity and premature final LV contraction that leads to the increase of postload on heart, myocardial stiffness, development of LV hypertrophy, deterioration of coronary perfusion [3,6,30]. A. Desai et al. found a direct correlation of MMWL and filling pressure with the main indices of LAD, increasing with the onset of AH and CHF [28].

Diastolic function (DFLD) is known to be dependent on age, AH, presence of HF, elevated HR [32,33]. Increased HR has a negative effect on DPLV by decreasing LV diastolic filling time and coronary perfusion time, increasing myocardial oxygen consumption, which leads to slow LV relaxation [33,41]. Negative correlations between sinus tachycardia and E/A ratio, LV early filling index were also found in healthy individuals, with Viau D. M. et al. [41] noted this correlation only in those under 60 years of age, and no such correlation was observed in those over 60 years of age. Fomin V. et al. [21] found that in elderly patients with dCHFS the presence of anemia and decreased renal function state also influences on LV filling, as well as age, presence of HLV, BP and BPD levels [4,21]. Tartiere J. et al. [40] found correlations between increased arterial stiffness, pulse wave dispersion and LV BP. Endothelial imbalance plays an important role in the pathogenesis of CHF [36,39]. A correlation has been established between the degree of endothelial dysfunction and the severity of CHF, determining its progression and outcome, both in asymptomatic LV dysfunction and clinically manifest CHF [18,19]. Damage to the endothelium leads to perversion of its dilating function in response to conventional stimuli, vasoconstriction and proliferation. Proliferation of arterial smooth muscle cells with a subsequent increase in their stiffness and the formation of fibrosis determines the processes of vascular remodelling in CHF [16,17,18].

It has been revealed that structural and functional changes in arteries increase with the development of CHF. E.S. Yavorova et al. [24] found that the increase of CCF is associated with a decrease in amplitude of endothelium-dependent vasodilation, thickening of intima-media complex of carotid arteries and an increase in aortic stiffness. F. Osmolovskaya et al. [16] showed that with increasing severity of CHF, with preserved and reduced LV contractile function, there is a decrease of microcirculatory vasoreactivity, LAS, central reflected wave.

The recommendations of the European Society of Cardiology (ESC) Working Group say that the diagnosis of primary (isolated) diastolic CHF is eligible when the following criteria are mandatory [2,12]:

1. Clinical signs of CHF;

2. Normal (LVEF 55% or >), mildly reduced (LVEF 50-54%) and moderately reduced (LVEF 40-49%) myocardial contractility;

3. Increased levels of BNP (BNP more than 35 pg/ml and/or NT-proBNP more than 125 pg/ml) in serum;

4. Other functional and structural changes underlying the development of CHF;

5. In case of doubt, a stress test or invasive detection of increased LV filling pressure. The initial assessment of CH involves clinical data combined with an assessment of LV systolic function (LVEF measurement). The analysis of LV diastolic function should begin with an assessment of LV systolic function (LVEF) and/or LV strain. A ventricular VEF of 50% is borderline for the diagnosis of CHFVL [2,12]. Patients with a ventricular VEF of 40-49% are often classified as LVEFL [2,10,11]. Recent guidelines define these patients as those with moderately reduced LVEF. The clinical features of CH patients with preserved, moderate and reduced LVEF are identical. Echocardiogram is the main diagnostic method to look for CHFVL. While the hallmark of the pathophysiological process in CHFVLV is the DD, EchoCG is mainly focused on the DD data. There is currently no unified measure of LV diastolic function. The main structural indices of LVD are an indexed LV volume greater than 34 mL/m2 or an indexed LV myocardial mass of 115 g/m2 or greater for men and 95 g/m2 or greater for women [5,12,30].

The implementation of optimal transmission of LV shock volume to body tissues requires a commensurate interaction between the LV and the arterial system [30]. This interaction between the LV as a pump and the vascular system as a load has been termed the left ventricular-arterial coupling (LVAC) and is measured as the ratio of arterial elastance (Ea) to ultimate systolic ventricular elastance (Ees) [8,27].

The concept of LVAS is important in the concept of CVD formation [8,27]. Normally, the interaction between the LV and the arterial system ensures that LV shock work is transmitted as efficiently as possible to the vessels. In HF, this interaction is impaired. In impaired LVAS, the energetic and mechanical efficiency of LV work decreases, especially with decreased LVEF [3,8]. The analysis of LVAS allows to note the effectiveness of interaction between the heart and vessels, remodelling and fibrosis [3,8,27]. Increased arterial stiffness has been shown to be a predictor of CVD and a major contributor to LVAS. At the same time, the relationship between aortic stiffness and CHF has not been sufficiently studied, although this issue is also significant [8,24,27].

Thus, the pathogenesis of CHF illustrates the multiple changes occurring in the body, from disturbances in the immune and CAS systems to changes in LVA with arterial stiffness, which in turn is a predictor of CVD and its complications.

Conclusion

CHF is the major complication of CVD and is the cause of hospital admission in every 2 patients (49%). The main causes of CHF are AH

(95.5%), CHD (69.7%), and their combination. More than 50% of CHF cases are associated with cardiac fibrillation while the heart retains its contractility. Cardiac DD usually precedes myocardial impairment, i.e. occurs in the early stages of CHF. A significant decrease in exercise tolerance, frequent hospitalisations and reduced quality of life are typical of patients with CHFsFV. The modern concept of CHF pathogenesis indicates interrelation and interdependence between such systems as CAC, RAAS, endothelin system, immune and inflammatory. In recent years, attention has been drawn to the increase in GAS, vascular load and impaired ventriculo-vascular interaction, which determine LV myocardial remodelling and the development of CHF.

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