Etiological Factors of Heart Remodeling

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Abstract. The review article is devoted to current issues of the etiology and pathogenesis of cardiovascular diseases leading to heart remodeling, its structural and morpho-functional changes. The authors analyzed modern research by foreign scientists, summarized and systematized the most common etiopathogenetic mechanisms of cardiovascular pathologies.

Key words: cardiac remodeling, cardiovascular diseases, etiology, pathogenesis.

Cardiovascular diseases are the result of various interrelated processes (arteriosclerosis, atherosclerosis, endothelial dysfunction), as well as, as numerous studies have shown, remodeling of the left chambers of the heart, which can increase the risk of developing cardiovascular complications. The course of arterial hypertension (AH) can be influenced by various external and internal factors. Quite often, hypertension is associated with metabolic disorders: obesity, including abdominal obesity, impaired glucose tolerance, dyslipidemia, etc. According to the literature, dyslipidemia with increased levels of atherogenic lipid fractions often occurs in hypertension [12].

The cardiovascular continuum is a continuous chain of interrelated changes in the cardiovascular system from exposure to risk factors, through the gradual onset and progression of CVD to the development of terminal heart disease and fatal outcome. A continuous chain of interrelated changes in the structure and function of several organs and systems of the body within a continuum suggests the presence of common pathophysiological processes, mechanisms of development and progression of organ damage [6].

Over the past decades, the burden of coronary heart disease (CHD) in the world has been consistently decreasing. This decrease is explained by the improvement and improvement of activities for primary and secondary prevention of cardiovascular diseases (CVD). Although mortality from coronary artery disease has declined in developed countries in recent decades, it still accounts for about a third of all deaths among people over 35 years of age. IHD is characterized by a wide range and association of social and clinical-anthropometric factors that influence the clinical course, the risks of complications and the social prospects of the subject in the disease situation. It is known that an independent predictor of morbidity and mortality from CVD is left ventricular myocardial mass (LVMM) [15].

Sex and gender characteristics of men and women manifest themselves in differences in health status, including cardiovascular health. Worldwide, cardiovascular diseases (CVDs) are the leading cause of morbidity, disability and mortality among both men and women. Recent evidence suggests a decline in incidence and mortality from coronary heart disease (CHD) in men, but not in women. In 2013,
morbidity analysis ischemic and hemorrhagic stroke in 188 countries of the world showed that in men this indicator exceeds the incidence rate in women. Moreover, globally, men have a higher rate of ischemic stroke than women, while no significant differences were observed in the prevalence of hemorrhagic stroke. At the same time, standardized mortality rates from CVDs in men in economically developed countries of Europe and Russia are higher, but the absolute number of deaths from CVDs is significantly higher in women [16]. Gender specificity determines the nosological features of damage to the cardiovascular system in men and women. Women are more likely than men to have nonobstructive epicardial artery disease, nonatherosclerotic spontaneous coronary artery dissection, stress cardiomyopathy (broken heart syndrome), plaque erosion, microvascular dysfunction, and a higher risk factor burden, even after adjustment for age. Atypical symptoms of coronary artery disease are also more common in women, due to different perceptions of pain. In-hospital mortality after acute myocardial infarction remains higher among young women compared with their male counterparts [14].

It is known that patients with CVD are at greater risk of developing COVID-19, especially in moderate to severe forms. According to repeated reviews and meta-analyses, ~40% of those infected with COVID-19 have concomitant CVD. Patients with pre-existing hypertension, diabetes mellitus and coronary artery disease are more likely to be hospitalized in the intensive care unit with subsequent severe and extremely severe course of the disease with the need for artificial support of respiratory function, incl. artificial ventilation. The risk of death in patients with COVID-19 and CVD increases 5-10 times [5].

Currently, one of the leading places in hypertensiology is occupied by the study of cardiac and vascular remodeling. Remodeling of the vascular wall is a complex multifactorial process, which is determined not only by the level of hemodynamic load and its association with cardiac remodeling, but also significantly depends on the activation of a number of neurohumoral systems, and also has a genetic predisposition. However, there is still no consensus on the comparative contribution of these characteristics to the formation of myocardial and vascular remodeling [9].

Myocardial remodeling includes hypertrophy of cardiomyocytes, changes in the shape and increase in the volume of cardiac chambers as a compensatory response aimed at maintaining cardiac output (CO). These changes occur in conditions of hyperreactivity of the sympathetic-adrenal (SAS) and renin-angiotensin-aldosterone system (RAAS). Structural changes LV in hypertension according to echocardiography is classified into four geometric models based on myocardial mass and relative LV wall thickness: concentric hypertrophy (increase in myocardial mass and relative LV wall thickness); eccentric hypertrophy (increase in myocardial mass with normal relative wall thickness); concentric remodeling (normal myocardial mass and increased relative wall thickness); normal LV geometry (normal myocardial mass and normal relative wall thickness) [1].

Myocardial hypertrophy is the most common type of cardiac remodeling, as cardiomyocytes increase in size compensatory to maintain cardiac output. Remodeling of the left ventricular (LV) myocardium includes a progressive increase in LV myocardial mass (LVMM), an increase in the volume of the cardiac cavities, and changing its geometric characteristics. Abdominal obesity is an independent pathological factor in myocardial remodeling [2].

Cardiac remodeling, which occurs in response to damage, leading to a change in its geometry and impaired contractility, ultimately determines the prognosis of life for patients with chronic obstructive pulmonary pathology. At the same time, asthma is not mentioned among lung diseases leading to the development of cardiac remodeling. There is also no consensus on the nature of the disorders in both the right and left hearts; their relationships with each other [10].
Myocardial remodeling in hypertension is one of the stages in the progression of cardiac changes, leading to the formation of left ventricular dysfunction and subsequently to the development of heart failure. According to modern concepts, there are four types of LV remodeling characteristic of patients with hypertension: 1) normal LV geometry; 2) concentric hypertrophy (increase in myocardial mass and relative thickness of the LV wall); 3) eccentric hypertrophy (increase in mass with normal relative thickness); 4) concentric remodeling (normal weight and increased relative wall thickness) [11].

Laboratory biomarkers are being considered with interest as tools for prognostic stratification. In recent years, more than 100 new biomarkers have been evaluated in this regard, and more than 4000 clinical studies have been published. Assessing the predictive accuracy of a new cardiovascular biomarker is very difficult. According to the principles of evidence-based laboratory medicine, a biomarker should not only be an independent predictor of outcome in multiple regression models, but also influence patient management, which is a prerequisite for cost-effectiveness. As a result, very few new laboratory biomarkers are recommended for risk prediction. Several studies have demonstrated that cardiovascular risk increases progressively in the general population for cTn values well above the 99th percentile, the recognized threshold for detecting myocardial injury and/or diagnosing myocardial infarction. Highly sensitive cTn methods can quickly identify patients at high risk of developing heart failure, which can lead to early diagnosis and improved prognosis for these patients [13].

However, it was also noted that the degree of increase in blood pressure (BP) and the duration of hypertension do not correlate with the severity of remodeling processes. It has been established that the development of different types of remodeling is associated not only with increased hemodynamic load, but also with the influence on the heart of numerous neurohumoral factors, the degree of activity of which can be genetically determined [3,4]. In this regard, there is an active search for candidate genes that influence the processes of myocardial remodeling and the study of the connection of these genes with a specific type of remodeling. The greatest attention has been attracted to genes encoding components of the renin-angiotensin system, genes of key sympathetic receptors, as well as genes whose defects can lead to endothelial dysfunction [8]. These genes are in one way or another associated with the load on the heart, including blood pressure, vascular resistance, heart rate and other parameters [7].

Conclusion

Cardiac remodeling in hypertension is essentially a compensatory reaction that allows the heart to work under conditions of high blood pressure. Patient risk stratification is an important goal because it guides treatment and follow-up strategies with the ultimate goal of influencing the natural history of the disease. There are two reasons for cardiac remodeling in obesity: hemodynamic and metabolic. Basically, the entire variety of such mechanisms can be reduced to genetic, hemodynamic and neurohumoral factors. Among the latter, one of the central roles belongs to the activation of the renin-angiotensin-aldosterone system (RAAS), which can be traced at almost all stages of the cardiovascular continuum.

Reference:


6. MLA


