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HISTOGENESIS OF THE CARDIOVASCULAR SYSTEM IN PATIENTS WITH HEART FAILURE

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Abstract

Heart failure (HF) is a complex clinical and pathophysiological syndrome characterized by the inability of the heart to provide adequate blood flow to meet the body's metabolic needs. Structural changes in HF are caused by disturbances in histogenesis and remodeling of cardiovascular tissues. This article examines the embryonic development of the cardiovascular system, as well as changes in cellular differentiation, regeneration, and intercellular interactions in heart failure. Particular attention is paid to the role of cardiomyocytes, endothelial cells, fibroblasts, and the extracellular matrix in disease progression.

In recent years, great importance has been attached to the study of the role of the pineal gland and its main hormone, melatonin, as a key factor determining the endogenous rhythms of the body in the regulation of the cardiovascular system (CVS). It has been shown that the pineal gland plays an important role in regulating the content of sex hormones, and its significant decrease coincides in time with the onset of menopause. Melatonin is a multifunctional hormone with powerful antioxidant, anti-inflammatory and cardioprotective effects. In the work of Girotti L. demonstrated that urinary melatonin sulfate levels in patients with CHF are, on average, 3 times lower than in healthy individuals. Zukhovitskaya E.V. noted a significant decrease in platelet melatonin concentrations in patients with severe CHF compared to controls and other CHF FCs. In patients with severe CHF, nocturnal melatonin secretion in the blood was found to negatively correlate with NT-proBNP levels. The pathogenetic and prognostic role of melatonin in the development and progression of cardiovascular diseases (CVD) and CHF is currently being clarified.

It is known that women are more likely to develop concentric left ventricular remodeling and HFpEF. Currently, patients with HFpEF represent a significant

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and rapidly growing subpopulation of individuals with CHF, with women occupying a leading position. However, HFpEF has been virtually unstudied in its gender context. This has led to a lack of convincing data regarding the prognostic value of numerous CHF markers. Clear treatment guidelines for patients with HFpEF that would reduce morbidity and mortality have not yet been developed. The pathogenesis of HFpEF continues to be clarified and debated, leading to a paradigm shift from the leading role of extracardiac mechanisms to the importance of cardiovascular factors: microvascular coronary dysfunction and symptomatic/asymptomatic myocardial ischemia. Thus, the study of gender-specific features of pathogenetic and molecular mechanisms involved in myocardial remodeling in HFpEF and their impact on the prognosis of patients seems relevant and promising.

Key words: Heart failure, histogenesis, cardiomyocytes, myocardial remodeling, endothelium, fibrosis, angiogenesis, extracellular matrix.

Introduction

The cardiovascular system is one of the first to develop during embryogenesis. Its histogenesis includes the differentiation of mesodermal cells into cardiomyocytes , endothelial cells, and vascular smooth muscle elements. In heart failure, secondary changes occur, affecting both tissue structure and function, due to disruption of normal regeneration and remodeling processes.

To identify gender differences in myocardial structural and functional changes depending on the genesis, functional class, and course of chronic heart failure in patients during the in-hospital and long-term follow-up periods after cardiac surgery and to determine the risk profile of patients with an unfavorable disease course. To evaluate the prognostic value of molecular markers of cardiomyocyte apoptosis and regeneration, extracellular matrix remodeling, microvascular remodeling, membrane melatonin receptor expression, and the role of telocytes in myocardial structural and functional changes in patients of both sexes with different chronic heart failure courses after cardiac surgery. To establish clinical and morphological parallels between left ventricular systolic and diastolic

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function, morphofunctional parameters, molecular markers of myocardial remodeling, CT-proBMP, and melatonin levels in patients with chronic heart failure, depending on gender.

To study the course of chronic heart failure in patients of both sexes during the in-hospital and long-term follow-up periods after cardiac surgery and determine the prognostic significance of the studied laboratory, instrumental, morphological, and molecular parameters. To establish the role of sex hormone deficiency in the development of structural and functional changes in the rat myocardium during experimental menopause and heart failure. To evaluate the effects of melatonin, drospirenone, enalapril, and metoprolol on myocardial remodeling in animals under simulated conditions, taking into account gender.

The aim of the study was to investigate the histogenesis and morphological changes of the cardiovascular system in patients with heart failure.

Research objectives. Describe normal histogenesis of the cardiovascular system. Study changes in cardiomyocytes in heart failure. Assess the role of endothelial dysfunction. Consider the processes of myocardial fibrosis and remodeling . Analyze changes in angiogenesis in heart failure.

Research methods. To study the histogenesis of the cardiovascular system in heart failure, a combination of morphological, molecular, and instrumental methods is used to assess structural and functional changes at various levels of organization. Clinical methods include a general patient examination, medical history, and assessment of heart failure symptoms. Echocardiography, electrocardiography, and cardiac magnetic resonance imaging are used for functional diagnostics. These methods allow us to determine the size of cardiac chambers, myocardial contractility, ejection fraction, and the degree of remodeling.

Morphological methods play a key role in the study of histogenesis. A myocardial biopsy is performed followed by histological examination. Standard staining methods (hematoxylin and eosin, Masson trichrome) are used to detect

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cardiomyocyte hypertrophy, inflammation, and fibrosis. For a more detailed analysis, electron microscopy is used to assess ultrastructural changes in cells, including mitochondria and sarcomeres.

Immunohistochemical methods are used to detect specific proteins and cellular markers. They determine the expression levels of growth factors, apoptotic markers, inflammation, and angiogenesis (e.g., VEGF, caspases , and cytokines). This allows for the assessment of the activity of pathological processes at the molecular level.

Molecular biological methods include polymerase chain reaction (PCR), quantitative gene expression analysis, and proteomic studies. These allow us to identify changes in the regulation of genes involved in myocardial remodeling, fibrosis, and vascular growth.

Biochemical methods are used to determine heart failure markers in the blood, such as natriuretic peptides (BNP, NT- proBNP), troponins, and inflammatory markers. These indicators reflect the extent of myocardial damage and the severity of the disease.

Experimental methods include the use of cell cultures and animal models of heart failure. This allows for the study of mechanisms of histogenesis, regeneration, and remodeling under controlled conditions, as well as the evaluation of the effectiveness of new therapeutic approaches.

Thus, the integrated use of clinical, morphological, molecular and experimental methods provides a comprehensive study of histogenetic changes in the cardiovascular system in heart failure and contributes to the development of new diagnostic and therapeutic strategies.

Study results

Cardiovascular histogenesis in heart failure is a complex pathological process involving disturbances in cellular differentiation, regeneration, and intercellular interactions. Under normal conditions, myocardial and vascular wall cells maintain structural and functional balance, ensuring adequate hemodynamics. However, with the development of heart failure, these mechanisms are significantly disrupted, leading to progressive tissue remodeling.

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One of the key factors is changes in the structure and function of cardiomyocytes. In response to increased stress, they undergo hypertrophy, which is initially compensatory. However, with prolonged exposure to pathological factors, the hypertrophy becomes maladaptive. Energy metabolism is disrupted in the cells, the structure of sarcomeres changes, and contractility decreases. Concurrently, apoptosis and necrosis are activated, leading to a decrease in the number of functionally active cardiomyocytes and a deterioration in the heart's pumping function.

Endothelial dysfunction plays a significant role in the pathogenesis of heart failure. The endothelium normally regulates vascular tone, hemostasis, and inflammatory responses through the production of biologically active substances, particularly nitric oxide. In heart failure, nitric oxide synthesis decreases, while the production of vasoconstrictors and proinflammatory cytokines increases. This leads to impaired vasodilation, increased vascular resistance, and impaired microcirculation.

An important component of histogenetic changes is the activation of fibroblasts and the development of myocardial fibrosis. Under the influence of mechanical stress, hypoxia, and neurohumoral factors, fibroblasts differentiate into myofibroblasts, which possess high synthetic activity. They begin intensively producing extracellular matrix components, primarily collagen. This results in the accumulation of connective tissue, which increases myocardial stiffness, impairs its elasticity, and worsens diastolic function.

At the same time, the structure of the extracellular matrix changes. The balance between the synthesis and degradation of its components is disrupted, leading to disorganization of tissue architecture. This negatively impacts the mechanical properties of the myocardium and intercellular signaling, exacerbating functional impairments.

Angiogenesis also undergoes significant changes in heart failure. Despite hypoxia, which stimulates vessel growth, the process is inadequate. New vessel formation lags behind the needs of the hypertrophied myocardium, leading to chronic tissue ischemia. Insufficient microcirculation contributes to further damage to cardiomyocytes and disease progression.

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Thus, cardiovascular histogenesis in heart failure is characterized by a complex of interrelated processes, including cardiomyocyte hypertrophy and death, endothelial dysfunction, fibrosis, and impaired angiogenesis. These changes underlie cardiac and vascular remodeling and determine the clinical course of the disease.

Conclusions

The histogenesis of the cardiovascular system in heart failure is characterized by complex and interrelated changes at the cellular, tissue, and molecular levels. The underlying pathological process is a disruption of the normal structure and function of cardiomyocytes, which undergo hypertrophy, followed by degenerative changes and death, leading to decreased myocardial contractility.

An important link in the pathogenesis is endothelial dysfunction, accompanied by a decrease in the production of vasodilatory factors and increased inflammatory processes. This contributes to increased vascular resistance and impaired microcirculation.

Fibroblast activation plays a significant role, leading to the development of myocardial fibrosis. Excessive accumulation of extracellular matrix leads to increased cardiac muscle stiffness, decreased elasticity, and deterioration of diastolic function.

Impaired angiogenesis exacerbates tissue ischemia, as vascular growth fails to meet the needs of the hypertrophied myocardium. This contributes to further cell damage and the progression of heart failure.

Thus, histogenetic changes in heart failure create a vicious cycle involving hypertrophy, ischemia, fibrosis, and cell death. Understanding these processes is essential for the development of modern diagnostic methods and pathogenetically based treatments for the disease.

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