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NEUROHUMORAL MECHANISMS IN THE PATHOGENESIS OF HEART FAILURE

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Abstract

Heart failure is a complex clinical syndrome characterized by high disability and mortality among cardiovascular diseases. This condition is characterized by a decrease in the ability of the heart to pump blood in sufficient quantities to meet the body's needs. In the pathogenesis of heart failure, along with hemodynamic disorders, the activation of neurohumoral mechanisms plays an important role.

Studies have shown that as a result of a decrease in cardiac output, the sympathetic nervous system, the renin–angiotensin–aldosterone system, antidiuretic hormone and other hormonal systems are activated as a compensatory response. Initially, these mechanisms serve to maintain arterial pressure, improve perfusion of vital organs and ensure hemodynamic balance. However, their long-term and constant activity increases the load on the myocardium, leads to structural and functional changes in cardiomyocytes, myocardial remodeling and progression of heart failure.

Also, protective mechanisms such as natriuretic peptides are activated, trying to regulate fluid balance in the body and limit the harmful effects of neurohumoral systems. However, in severe stages of heart failure, the compensatory capabilities of these mechanisms are insufficient. This article analyzes the main neurohumoral mechanisms involved in the pathogenesis of heart failure, their clinical significance and role in the development of the disease.

Keywords

Heart failure, pathogenesis, neurohumoral mechanisms, sympathetic nervous system, renin–angiotensin–aldosterone system, vasopressin, natriuretic peptides, myocardial remodeling.

Relevance of the topic: Heart failure is one of the most urgent problems of modern cardiology, and is among the diseases that reduce the quality of life of

millions of people around the world and lead to high mortality rates. The aging of the population, the widespread prevalence of cardiovascular risk factors such as arterial hypertension, ischemic heart disease, diabetes mellitus and obesity are leading to an increase in the incidence of heart failure every year. Therefore, in-depth study of the mechanisms of development of this disease is extremely important for clinical practice.

In recent years, it has been scientifically proven that heart failure is not only a consequence of impaired pumping function of the heart, but also a disease accompanied by disruption of complex neurohumoral regulatory systems. Long-term activation of the sympathetic nervous system, renin–angiotensin–aldosterone system and antidiuretic hormone system leads to myocardial remodeling, fibrosis, cardiomyocyte apoptosis and further deterioration of heart function. Without a proper understanding of these processes, effective treatment of heart failure and improvement of prognosis are impossible.

Modern drugs used in the treatment of heart failure today - angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta-adrenoblockers, aldosterone antagonists and new generation drugs - are aimed at suppressing neurohumoral mechanisms. This indicates that this topic is not only theoretical, but also practical. Also, a deep analysis of neurohumoral pathogenesis plays an important role in improving the quality of life of patients with heart failure, reducing the number of hospitalizations and reducing mortality. Therefore, a comprehensive study of the role of neurohumoral mechanisms in the pathogenesis of heart failure, analyzing their interrelationships and integrating them with clinical practice is one of the current and priority areas of modern medical science

Purpose of the topic: The main purpose of this article is to scientifically shed light on the role of neurohumoral mechanisms in the development of heart failure, to deeply analyze their inextricable link with pathophysiological processes. In particular, one of the important tasks of this study is to determine the effect of activation of the sympathetic nervous system, renin-angiotensin-aldosterone system, antidiuretic hormone and other biologically active substances on structural and functional changes in the heart muscle.

The article also analyzes in detail the stages of transformation of neurohumoral systems from compensatory mechanisms into pathogenic factors, their role in myocardial remodeling, fibrosis processes, hemodynamic disorders and a decrease in the pumping function of the heart. By shedding light on the importance of these mechanisms in the clinical course and exacerbation of heart failure, it is intended to achieve a deeper understanding of the pathogenesis of the disease.

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In addition, one of the important goals of this article is to demonstrate the scientific basis of modern treatment approaches aimed at suppressing neurohumoral activity and to evaluate their clinical effectiveness from the point of view of pathogenesis. This is planned to provide scientific conclusions that will improve the prognosis of patients with heart failure, reduce the development of complications, and improve the quality of life.

In general, the purpose of this article is to systematically reveal the leading role of neurohumoral mechanisms in the pathogenesis of heart failure and to integrate them with clinical practice.

Main part: Heart failure is a complex clinical syndrome characterized by a decrease in the heart's ability to pump blood in accordance with the body's needs, in its development, along with hemodynamic disorders, neurohumoral mechanisms play a leading role. Initially, when cardiac activity decreases, the body activates a number of neurohumoral systems as a compensatory response. Although these processes support blood circulation in the short term, they lead to a worsening of heart failure in the long term.

When cardiac output decreases, arterial pressure decreases and tissue perfusion is impaired. As a result, the sympathetic nervous system is activated through baroreceptors. As a result of the activity of the sympathetic system, the heart rate increases, myocardial contractility increases, and peripheral vessels constrict. Initially, these changes serve to restore cardiac output to a certain extent. However, prolonged activity of the sympathetic system increases the need for oxygen in myocardial cells, causes cardiomyocyte fatigue, apoptotic processes, and the development of arrhythmias. The renin-angiotensin-aldosterone system (RAAT) is of particular importance in the pathogenesis of heart failure. As a result of a decrease in blood flow in the kidneys, renin secretion increases, which leads to the formation of angiotensin II. Angiotensin II is a potent vasoconstrictor, increases peripheral resistance and increases the workload on the heart. At the same time, aldosterone secretion increases, causing sodium and water retention in the body. This leads to an increase in blood volume, the formation of edema, and an increase in the clinical symptoms of heart failure.

Antidiuretic hormone (vasopressin) is also activated in heart failure, contributing to water retention. This causes hypervolemia and increased pressure in the vascular system. As a result, the pumping function of the heart deteriorates further. In this way, neurohumoral mechanisms that were initially compensatory become pathogenic over time.

Chronic activity of neurohumoral systems leads to myocardial remodeling. The walls of the heart thicken or dilate, and fibrous tissue increases between

cardiomyocytes. This reduces the elasticity of the heart and causes a deepening of diastolic and systolic dysfunction. In particular, angiotensin II and aldosterone are the main factors that increase myocardial fibrosis.

In response, natriuretic peptides (ANP and BNP) are secreted in the body. They increase the excretion of sodium and water and try to dilate the vessels. However, in severe stages of heart failure, the effect of this system is insufficient and pathological neurohumoral activity prevails.

Thus, neurohumoral mechanisms play a central role in the pathogenesis of heart failure, determining the development, severity and clinical course of the disease. A deep understanding of these mechanisms provides a scientific basis for the use of drugs such as ACE inhibitors, beta-blockers, aldosterone antagonists in the treatment of heart failure.

Conclusion: In conclusion, neurohumoral mechanisms are one of the leading pathogenetic factors in the development of heart failure. A decrease in the pumping function of the heart leads to the activation of the sympathetic nervous system, the renin-angiotensin-aldosterone system and the secretion of vasopressin as a compensatory response in the body. Initially, these systems serve to support blood circulation and maintain perfusion of vital organs. However, their long-term and uncontrolled activity creates the basis for the aggravation of heart failure.

Chronic activity of neurohumoral systems leads to vasoconstriction, fluid and sodium retention in the body, increased cardiac workload, and structural changes in the myocardium - remodeling processes. These conditions are manifested by damage to cardiomyocytes, increased fibrous tissue, expansion or thickening of the heart walls, which leads to a further exacerbation of systolic and diastolic dysfunction. As a result, the clinical symptoms of heart failure increase, and the disease acquires a chronic and progressive character.

At the same time, although counter-regulatory mechanisms such as natriuretic peptides are activated in the body, their effect is insufficient in the stages of severe heart failure, and pathological neurohumoral activity prevails. This once again confirms the important role of neurohumoral imbalance in the pathogenesis of heart failure.

The obtained scientific conclusions form the basis of modern pharmacological approaches to the treatment of heart failure. In particular, the use of ACE inhibitors, angiotensin II receptor blockers, beta-blockers and aldosterone antagonists slows down the progression of the disease by suppressing the activity of neurohumoral systems, increasing the quality of life and survival of patients.

Therefore, a deep study of neurohumoral mechanisms in the pathogenesis of heart failure is of great scientific and practical importance for early detection of the

disease, development of effective treatment strategies and prevention of severe complications. An integrated approach taking into account these mechanisms allows achieving the most optimal results for patients with heart failure.

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