PATHOGENETIC MECHANISMS OF HEART FAILURE

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Abstract. *The origin of the heart failure disease, the mechanisms of the origin of the disease, the pathogenetic mechanisms of the disease, the importance of the pathogenetic mechanisms in the clinic of the disease and treatment measures.*

Key words: *Chronic Heart Failure, extracardiac, cardiac, renin-angiotensin-aldosterone system, vasoconstriction.*

Extracardiac and cardiac compensatory mechanisms are involved in the pathogenesis of chronic heart failure. These mechanisms are activated in a compensatory way to supply the organ with sufficient blood when the pumping activity of the heart is disturbed, but later it turns into a pathological process. Extracardiac compensatory mechanisms include:

- Sharp limitation of the body's energy expenditure (more in muscles);

- Reflexive acceleration and deepening of breathing in order to provide the body with oxygen at the required level;

- Increase in the number and strength of heart contractions under the influence of impulses sent from the brain;

- Reduction of the load on the heart as a result of a decrease in the tone of arterioles.

- The activation of the following neuroendocrine system causes the occurrence of the mentioned extracardiac mechanisms:

Sympatho-adrenal system and its effectors (adrenaline and noradrenaline).

Their effect is to accelerate the number of heart contractions. to increase of its one-minute blood volume, number of myocardial contractions and venous tone (this increases venous blood return to the heart and increases pre-systolic pressure), systemic vasoconstriction, increase in general peripheral and blood pressure, myocardial compensator It led to the development of hypertrophy, the activation of RAAT due to the effect of RAT β-adrenergic receptors in supraglomerular cells and tissues as a result of endothelium dysfunction.

So, in the early stages of heart failure, SAT activation causes myocardial contraction, blood flow to the heart, preload and increased ventricular filling pressure. This, in turn. for a certain period of time it makes it possible to keep the blood pumping of the heart at a normal level. But this system is used for a long time in patients with SYY! staying in this position leads to an increase in pre- and post-load (vasoconstriction, retention of water and sodium in the body), to an increase in the oxygen demand of the myocardium. catecholaminami directly leads to increased cardiotoxic effect and finally to ventricular arrhythmia.

Activation of the renin-angiotensin-aldosterone system (RAAT) plays a special role in the formation of heart failure. In this, not only the neurohormones of this system (renio-angiotensin, angiotensin-I and aldosterone) circulating in the blood, released from the kidney and adrenal gland, but also limited RAT in bulk tissues (including the myocardium) are important. Any decrease in perfusion pressure in the kidneys causes an increase in RAT in it and increases the release of renin from YGA cells and the breakdown of angiotensin into peptide-angiotensin-1 (AI). AI csa binds to angiotensin II (All) by exerting a strong effect on RAAT under the influence of AAF. Inhibitors of angiotensin-converting enzyme (AAF), which are crucial in this process, are located in the membrane of endothelial cells of pulmonary vessels, in the proximal tubules of the kidney, in the myocardium, and in the blood serum that produces AII.

Its effect (in kidney, heart, arteries, adrenal gland and other places) is carried out through specific angiotensin receptors (ATI and ATP). As a result of the activation of RAT in tissues other than AAF, esp. chymases, chymase-like enzymes,

cathepsinG, tissue activators of plasminogen and others also cause the conversion of Angiotensin I to Angiotensin II. Finally, aldosterone is produced as a result of the effect of AP on ATII-receptors in the brain part of the adrenal gland and in the area of the balls. Under the influence of this hormone, excess oil and water accumulate, and as a result, the volume of blood circulating in the body increases. In summary, RAAT activation leads to:

Obvious vasoconstriction and increase in blood pressure;

- Retention of sodium and water in the body and increase in circulating blood volume;

- Increase in myocardial contractility;

- Heart hypertrophy and remodeling;

- activation of the formation of connective tissue (collagen) in the myocardium;

To increase the sensitivity of the myocardium to catecholamines.

In the early stages of acute and chronic heart failure, activation of RAAT is compensatory, aimed at maintaining blood pressure, blood circulating in the body, perfusion in the kidneys, increasing preload and myocardial contractility. However, due to its long-term active status, the following negative effects are also observed:

-Increased general periteric resistance in vessels and decrease in blood flow in internal organs and tissues;

- A sharp increase in the final load on the heart;

- A significant increase in fluid retention in the body and, as a result, an increase in preload with the formation of swelling syndrome;

-Development of the remodeling process in the heart and vessels, including the occurrence of myocardial hypertrophy and smooth muscle cell hyperplasia:

- Increased collagen synthesis and development of cardiac muscle fibrosis:

- Acceleration of cardiomyocyte necrosis and increasing damage to the myocardium, formation of myogenic dilatation in the ventricles;

- Increased sensitivity of heart muscles to catecholamines and, as a result, severe ventricular arrhythmias.

Arginine-vasopressin (antidiuretic hormone) system. Antidiuretic hormone

(ADH) is produced in the posterior lobe of the pituitary gland and controls the flow of water from the distal tubule of the kidney. ADG production increases in the case of dchydration of tissues, which is observed as a result of a decrease in water in the body, and on the contrary, it decreases in hyperhydration. Disruption of this functional mechanism in heart failure causes excess water retention in the body and leads to the development of edema syndrome. As the heart's pumping activity decreases, it causes more stimulation of the osmo and voloreceptors, which causes an increase in the production of ADG. The last condition increases fluid retention in the body.

Partial natriuretic peptide (BNP). BNP is a unique antagonist of vasoconstrictors (sympatho-adrenal, RAAT, ADG, etc.) in the body. It is produced by the myocytes of the bundles and is added to the blood stream when the vessels are stretched, and has a vasodilating, natriuretic, diuretic effect, and reduces the production of renin and aldosterone. BNP activity increases as heart failure progresses. However, despite the high level of BNP circulating in the blood, its positive effect is significantly reduced in SYY. This is probably due to a decrease in the sensitivity of receptors and an increase in the breakdown of peptides. Therefore, the higher the level of BNP circulating in the blood, the more severe the SYY in patients.

Violation of endothelial activity. In recent years, special attention has been paid to the dysfunction of the endothelium in the formation of SYY. Usually, this process occurs due to the influence of various negative factors (increased catecholamine, AII, serotonin, high blood pressure, increased blood flow, etc.) and endothelium-dependent vasoconstrictor It leads to an increase in blood pressure and, in connection with it, an increase in the tone of the vessel wall, aggregation of platelets, and acceleration of the formation of blood clots in the vessel wall. Endothelin-I, thromboxane A, prostaglandin PGH2, AII, etc. are important endothelium-dependent vasoconstrictor substances that increase vascular tone, increase platelet aggregation and blood clotting. In addition, entothelin I has the property of enhancing protein synthesis and developing cardiac muscle hypertrophy. In severe

forms of SYY, endothelin I exceeds the norm by 2-3 times, and its level in blood serum is directly related to intracardiac hemodynamics, pulmonary artery pressure, and patient mortality.

Hyperproduction of cytokines. Cytokines are protein mediators with low molecular weight in the cell, which participate in the mechanism of intercellular interaction and biological processes (immune reaction in hemopoietic, lymphoid and mesenchymal cells, tissue repair, angiogenesis, inflammation). 'growth and management) controls. They are synthesized by activated immune system, fibroblasts, epithelium, endothelium and stromal cells of bone marrow. The significance of pro-inflammatory cytokines - α-tumor necrosis factor, interleukin-1 and interleukin-6 in the pathogenesis of SYY has been studied in depth. The mechanism of myocardial damage and contractility due to cytokines is different, the main ones are as follows:

> Directly exerts a toxic and damaging effect on the myocardium, causing a reduction in its contractility, activation of the synthesis of connective tissue in the myocardium;

> Enhances the process of apoptosis in cardiomyocytes and peripheral muscle cells;

> It is one of the reasons leading to myocardial hypertrophy and remodeling of the heart;

> Disturbance of endothelium-dependent dilatation of arterioles, long-term preservation of total peripheral vascular resistance at high levels, and as a result, increase myocardial oxygen demand and decrease contractility. In recent years, apoptosis of cardiomyocytes caused by cytokines has been considered as one of the mechanisms leading to irreversible changes in myocardial contractility in SYY. Due to its long-term effect, it is important in the destruction of the intracellular collagenous matrix of the myocardium, in hypertrophy, dilatation and remodeling of the myocardium. Therefore, their activation has a negative inotropic effect on SYY, reduces the volume of blood pumping of the heart, increases the internal pressure and limits the physical activity of the patient, thus leading to the

exacerbation of the pathological process.

Mechanisms of cardiac compensation. Concentric and eccentric hypertrophy of the myocardium develops under the influence of the above-mentioned neurohumoral and etiological factors. Concentric hypertrophy in the myocardium with long-standing high end-load. that is, without expanding the space of the ventricles, it leads to the thickening of the muscle layer. In concentric hypertrophy, an increase in the thickness of the myocardium, an increase in intraventricular pressure during systole, overcomes the final load, and allows sufficient blood supply to the organs and tissues. When the preload increases, eccentric hypertrophy gradually develops, and this situation is due to tonogenic dilatation of the ventricle cavity. Myocardial hypertrophy and pronounced tonogenic dilatation of the left ventricle keep the blood pumping volume of the heart at the required level for a certain period of time. that is, according to Starling's law, the increase in end-diastolic volume in the ventricles overcomes the increased pre- and post-load and increases its contraction. But over time, as a result of progressive hemodynamic stress or direct damage to the myocardium, the compensatory mechanisms of the heart become insufficient, the efficiency of the Starling mechanism decreases sharply, and the blood pumping capacity of the heart decreases. As a result, all the pathogenetic factors of SYY listed above, first of all, the process of cardiac remodeling occurs under the influence of the neurohumoral system.

Remodeling is a change in the composition of the left ventricle and its hemodynamic parameters. It includes myocardial hypertrophy and heart dilatation, which leads to a change in the geometric shape of the heart, systolic and diastolic dysfunction. Key components of left ventricular remodeling.

❖ Changes at the level of individual cardiomyocytes:

-Disturbance of ATF formation during oxidation-phosphorylation and as a result decrease of it and crcatine phosphate reserve;

- Violation of the composition and properties of proteins that provide mobility and contractility;

- Cardiomyocyte desensitization of β-adrenoreceptor apparatus;

- Hypertrophy of cardiomyocytes;

- Violation of protein activity in cardiomyocytes;

❖ Changes in the left ventricular myocardium:

- Decrease in the number of cardiomyocytes (due to necrosis and apoptosis);

- Change of the extracellular matrix (activation of metalloproteinase, degradation of the matrix with fibrotic tissue occupying the tumor).

- Changes in the geometry of the left ventricle:

- Left ventricular dilatation;

- Spherical configuration of the left ventricle;

- Thinning of the heart walls;

- Occurrence of functional (relative) mitral regurgitation.

Approximately 25-30% of all HF patients have left ventricular diastolic dysfunction. that is, during diastole, the heart muscles cannot relax enough and cannot accommodate the required blood. Accumulation of excess collagens in the interstitial tissue of the myocardium increases its stiffness and leads to impaired relaxation during diastole. In accordance with myocardial hypertrophy, the muscle layer thickens. When the heart moves along with the increase of the muscle and interstitial components, this process becomes adaptive and is considered concentric. When excessive connective tissue is continuously produced and perivascular and interstitial fibrosis begins to dominate, myocardial hypertrophy takes on a pathological, i.e., eccentric character and over time leads to first diastolic and then systolic dysfunction.

In short, the activity of the above-mentioned neurohormonal system and endothelial dysfunction play a leading role in the pathogenetic mechanism of the formation of heart failure. At the beginning of the process, they occur as a result of systolic and diastolic dysfunction of the ventricles. has an adaptive nature and is directed to the blood pumping activity of the heart, the systemic blood supply to the organs and tissues of the body. This is an acceleration of the heartbeat. as a result of compensator hyperfunction and the hypertrophy that develops as a result, the heart pumps blood and increases the last and previous load, the amount of blood

circulating in the body. But these extracardiac mechanisms develop after the end of the possibility of cardiac inechanisms, which provide blood circulation for a long time, that is, in the compensation stage.

Compensatory hyperfunction means a situation where the overload on the heart does not have a negative effect on its performance. This process is carried out by the myocardium, which has not undergone hypertrophy in the early stages of heart failure, and causes its hypertrophy in a short period of time.

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