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HEART FAILURE WITH A REDUCED EJECTION FRACTION ("SYSTOLIC HF")

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Abstract
The article presents heart failure (HF) with a reduced ejection fraction or syndrome of ventricular dysfunction of the heart. Left ventricular failure leads to the development of shortness of breath and fatigue, right ventricular failure leads to peripheral edema and fluid accumulation in the abdominal cavity. Both ventricles or each ventricle separately may be involved in the process. The diagnosis is established clinically, confirmed by chest X-ray data, echocardiography and levels of natriuretic peptides in blood plasma.

Keywords: Left ventricular failure, angiotensin converting enzyme, pacemakers, defibrillators.

Introduction

Approximately 6.5 million people in the United States suffer from heart failure; > 960,000 new cases are reported annually. Approximately 26 million people worldwide suffer from this disease. In heart failure, the heart cannot provide tissues with enough blood for metabolism and an increase in pulmonary or systemic venous pressure can lead to fullness of blood in peripheral organs. A similar condition can occur with disorders of both systolic and diastolic heart function (more often both). Although the primary pathology may be a change in the function of cardiomyocytes, there is also a violation of the synthesis and breakdown of extracellular matrix collagen in the myocardium (1,3,5). Defects in the structure of the heart (for example, congenital and acquired valve defects), rhythm disturbances (including persistent high heart rate) and high metabolic demand (for example, due to thyrotoxicosis) can also be the cause of HF (7,9)

Heart failure with reduced ejection fraction (CH SFV)

With HF SnFV (also called systolic HF), general LV systolic dysfunction prevails. LV contracts weakly and is not completely emptied, which leads to

• Increased diastolic volume and pressure
• Reduction of the ejection fraction (≤ 40%)

There are violations in energy consumption, energy supply, electrophysiological functions, contractility disorders develop with disorders of intracellular calcium metabolism and synthesis of cyclic adenosine monophosphate (cAMP).

The predominance of systolic dysfunction is a common occurrence in heart failure due to myocardial infarction, myocarditis and dilated cardiomyopathy. Systolic dysfunction can develop mainly in the LV or pancreas; LV insufficiency often leads to the development of pancreatic insufficiency (2,4)

Heart failure with preserved ejection fraction (SNFV)

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With NSFV (also called diastolic heart failure), LV filling worsens, which leads to

• Increased end-diastolic LV pressure at rest or during exercise

• Normally normal end-diastolic blood volume of the left ventricle (LV)

The overall contractility and, consequently, the ejection fraction remain normal (≥ 50%).

Nevertheless, in some patients, pronounced restriction of LV filling can lead to an inadequately low end-diastolic volume and, thus, be the cause of low SV (cardiac output) and systemic symptoms. Increased pressure in the left atrium can cause pulmonary hypertension and pulmonary hyperemia.

Diastolic dysfunction usually develops with impaired ventricular relaxation (an active process), increased ventricular rigidity, heart valve disease or stenosing pericarditis. Acute myocardial ischemia may also be the cause of diastolic dysfunction. With age, the resistance to filling increases, probably both due to the dysfunction of cardiomyocytes, and due to a decrease in their number, as well as due to an increase in collagen deposits in the interstitium, therefore diastolic dysfunction is characteristic of the elderly. Diastolic dysfunction prevails in hypertrophic cardiomyopathy, diseases leading to the development of ventricular hypertrophy (for example, hypertension, severe aortic stenosis) and amyloid infiltration of the myocardium. LV filling and functions may also deteriorate in cases where, due to a pronounced increase in pressure in the pancreas, the interventricular septum bulges to the left (6,8).

Recently, diastolic dysfunction has been increasingly recognized as the cause of HF. According to various estimates, about 50% of patients with heart failure have NSFV; at the same time, the incidence of the disease increases with age and in patients with diabetes. Currently, it is known that SNSFV is a complex, heterogeneous, multi-organ systemic syndrome, often with multiple concomitant pathophysiological mechanisms. Current evidence suggests that multiple concomitant diseases (e.g., obesity, hypertension, diabetes, chronic kidney disease) lead to systemic inflammation, widespread endothelial dysfunction, microvascular dysfunction of the heart and, ultimately, to molecular changes in the heart that cause increased myocardial fibrosis and increased ventricular stiffness. Thus, although SNCnFV is usually associated with primary myocardial damage, SNCFV may be associated with secondary myocardial damage due to peripheral pathological changes (1.5)

Heart failure with a moderately reduced ejection fraction (SNUFV)

International scientific societies have put forward the concept of HF with a moderately reduced ejection fraction (SNUFV), in which patients have a LV ejection fraction from 41 to 49%. It is unclear whether this group is a separate population or consists of patients with both SNsFV and SNsnFV.

Insufficiency of the left ventricle of the heart

In heart failure associated with left ventricular dysfunction, SV decreases and pulmonary venous pressure increases. Since the pulmonary capillary pressure exceeds the oncotic pressure of plasma proteins (approximately 24 mmHg), the liquid part of the blood penetrates from the capillaries into the intercellular space and alveoli, reducing pulmonary function and increasing the frequency of respiratory movements. The lymphatic drainage of the lungs increases compensatorily, but this does not compensate for the increase in the amount of fluid in the lungs. Massive accumulation of fluid in the alveoli (pulmonary edema) significantly disrupts the ventilation-perfusion ratio (I/N): non-oxygenated pulmonary arterial blood passes through poorly ventilated alveoli, which leads to a decrease in the partial pressure of oxygen (PaO2) in arterial blood and causes shortness of breath.

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However, shortness of breath may occur before an IV disorder, probably due to increased pulmonary venous pressure and increased respiratory activity; the exact mechanism of this phenomenon is unclear. (2,6)

With severe or chronic LV insufficiency, pleural effusion usually develops, which further exacerbates shortness of breath. Minute ventilation increases, and this leads to hypocapnia (decrease in PaCO2 in arterial blood), CO2 and an increase in blood pH (respiratory alkalosis). Significant interstitial edema in the area of the small airways may interfere with ventilation, increasing PaCO2, which is a sign of threatening respiratory failure.

Insufficiency of the right ventricle of the heart

In heart failure accompanied by dysfunction of the right ventricle, the total venous pressure increases, which is accompanied by the release of fluid into the intercellular space, followed by the appearance of edema, primarily peripheral tissues (feet and ankles in outpatient patients) and internal organs of the abdominal cavity. The liver suffers the most, but there is also an induration of the stomach and intestines; there is an accumulation of fluid in the abdominal cavity (ascites). Right ventricular failure usually causes moderate impairment of liver function, usually with a slight increase in bound and free bilirubin, prothrombin time, and liver enzyme activity (especially alkaline phosphatase and gamma-glutamyltranspeptidase [GGT]). The damaged liver is unable to inactivate aldosterone, secondary aldosteronism develops, which contributes to the accumulation of fluid. Chronic venous congestion in internal organs can cause anorexia, malabsorption of nutrients and drugs, enteropathy with loss of protein (characterized by diarrhea and significant hypoalbuminemia), permanent blood loss through the gastrointestinal tract and sometimes ischemic intestinal infarction.

Reaction from the heart

With SNSFV, the systolic function of the left ventricle is very weakened; therefore, increased preload occurs to maintain normal SV. As a result, ventricular remodeling occurs over time: during such remodeling, the left ventricle becomes less elliptoid and more spherical, expands and hypertrophs; the right ventricle dilates and may hypertrophy. Being initially compensatory, remodeling is ultimately associated with adverse outcomes, because these changes ultimately increase ventricular wall tension and diastolic rigidity (i.e., diastolic dysfunction develops), disrupting heart function, especially during exercise. Increased tension of the heart wall increases oxygen demand and accelerates apoptosis (programmed cell death) of myocardial cells. Ventricular dilation can cause mitral or tricuspid regurgitation (on the background of annulodilation) with a further increase in end-diastolic volumes.

Hemodynamic changes

With reduced SV, oxygen supply to tissues is maintained by increasing oxygen extraction from the blood, which sometimes leads to a shift in the oxyhemoglobin dissociation curve to the right to improve oxygen release (5,7,8)

Reduced CB with reduced systemic arterial pressure activates arterial baroreflex, increasing sympathetic and decreasing parasympathetic tone. As a result, there is an increase in heart rate and myocardial contractility, arterioles in the corresponding areas of the vascular bed narrow, venoconstriction joins and sodium and water retention occurs. These changes compensate for the decrease in ventricular function and help maintain hemodynamic homeostasis only in the early stages of heart failure. However, these compensatory mechanisms lead to increased heart function,

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increased preload and postload, decrease coronary and renal blood flow, cause fluid retention, increase potassium excretion, and can also cause necrosis of cardiomyocytes and arrhythmias (3,6)

Classification of heart failure

The most common classification of heart failure currently in use divides patients into:

• Heart failure with reduced ejection fraction ("systolic HF")

• Heart failure with preserved ejection fraction ("diastolic HF")

Heart failure with a reduced ejection fraction (LVEF) is defined as heart failure with a left ventricular ejection fraction (LVEF) of less than 40%.

Heart failure with preserved ejection fraction (SNFV) is defined as heart failure with LVEF ≥ 50%. Patients with LVEF between 41% and 49% are in the intermediate zone and have recently been classified as having HF with a moderately reduced ejection fraction (SNUFV—1).

The traditional distinction between left and right ventricular insufficiency is somewhat erroneous, since the heart is an integral system that performs a pumping function; pathological changes in one chamber eventually affect the work of the whole heart. However, these terms define the location of the largest lesion leading to heart failure and may be useful for initial diagnosis and treatment. Other, most commonly used terms used to describe heart failure include: acute and chronic, with high or low cardiac output, dilated or not, as well as ischemic, hypertensive or idiopathic dilated cardiomyopathy. Treatment differs depending on whether this manifestation is a symptom of acute or chronic HF.

LV insufficiency usually develops in ischemic heart disease (CHD), hypertension, mitral valve regurgitation or aortic valve regurgitation, aortic stenosis, most forms of cardiomyopathy, congenital heart defects (for example, ventricular septal defect, open ductus arteriosus with large shunts).

Pancreatic insufficiency is usually caused by previous LV insufficiency (leading to an increase in pulmonary venous pressure and pulmonary hypertension, i.e., to an overload of the pancreas) or severe lung diseases (then this condition is called pulmonary heart). Other causes are multiple pulmonary embolism, pancreatic infarction, pulmonary hypertension, tricuspid regurgitation or tricuspid stenosis, mitral stenosis, pulmonary artery stenosis, pulmonary valve stenosis, veno-occlusive lung disease, arrhythmogenic pancreatic cardiomyopathy, or birth defects such as Ebstein's anomaly or Eisenmenger syndrome. Some diseases mimic pancreatic insufficiency, except that heart function may be normal; Such diseases include: volume overload of the heart and increased systemic venous pressure in polycythemia or massive transfusions, acute renal failure with sodium and water retention, obstruction of any vena cava and hypoproteinemia due to any causes leading to low oncotic plasma pressure and peripheral edema.

Biventricular insufficiency occurs in diseases that damage the entire myocardium (for example, viral myocarditis, amyloidosis, Chagas disease) or in cases where long-term left ventricular insufficiency leads to the development of pancreatic insufficiency.

High-emission HF occurs when there is a constant need for high cardiac output, which can ultimately lead to the inability of a normal heart to maintain the necessary output. Diseases that can lead to an increase in SV (cardiac output) include severe anemia, end-stage liver disease, beriberi, thyrotoxicosis, progressive Paget's disease, arteriovenous fistula and constant tachycardia.

Cardiomyopathy is a general term indicating myocardial diseases. Most often, this term is used to refer to primary damage to the ventricular myocardium, which is not caused by congenital

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anatomical defects, valvular, systemic or pulmonary vascular disorders, primary diseases of the pericardium or components of the conductive system, as well as coronary heart disease. Sometimes this term is used to describe the etiology (for example, ischemic or hypertensive cardiomyopathy). Cardiomyopathy does not always lead to HF with symptoms. It is often idiopathic and is classified into dilated, congestive, hypertrophic, infiltrative-restrictive, or apex-enlarged cardiomyopathy (also known as takotsubo cardiomyopathy or stress cardiomyopathy).

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