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Diagnosis of Chronic Right Heart Failure

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Abstract

Heart failure caused by a violation of the right ventricular function is associated with late diagnosis and high mortality. Many diseases of the heart and other organs lead to the development of right ventricular heart failure. Analysis of the causes of development, physical data, instrumental methods of diagnostics (electrocardiography, echocardiography, magnetic resonance imaging, catheterization of the right ventricle), laboratory methods (levels of atrial natriuretic peptide) can detect failure of the right heart in the early stages of heart failure development.

Keywords: Heart Failure; Pulmonary Hypertension; Ischemia Myocarditis; Transesophageal Echocardiography; Valvular Heart Disease

Abbreviations: CHD: Coronary Heart Disease; RV: Right Ventricle; HC: Hypertrophic Cardiomyopathy; ARVD: Arrhythmogenic Right Ventricular Dysplasia; DCM: Dilated Cardiomyopathy; ASD: Atrial Septal Defect; VSD: Ventricular Septal Defect; TAPSE: Tricuspid Annular Plane Systolic Excursion; FAC: Fractional Area Change; MPI: Myocardial Performance Index; IVRT: Isovolumic Relaxation Time; IVCT: Isovolumic Contraction Time; ET: Ejection Time; RT3DE: Three-Dimensional Echocardiography in Real Time; ANP: Atrial Natriuretic Peptide; BNP: Brain Natriuretic Peptide.

Introduction

Heart failure due to violation of right ventricle function is often diagnosed as a result of biventricular heart failure, which has developed on a basis of the left ventricle lesion. However many diseases (pulmonary hypertension, cardiomyopathy, congenital and acquired defects of the tricuspid valve, left-right cardiac shunts) cause primarily violations of the right ventricle function and then lead to left ventricle failure [1]. Blood ejection from the right ventricle is energetically less expensive in comparison with the left ventricle due to lower pulmonary vascular resistance; right ventricle performs less work [2]. The smaller thickness of the right ventricle wall leads to a shorter period of right ventricle failure development as compared to the left ventricle. Symptoms of right heart failure at early stages are not specific with poor clinical manifestations, in later progression of the process leading inexorably to manifest heart failure and death. Identification of predisposing conditions and diseases, early signs of right heart failure contributes to the timely implementation of therapeutic measures.

Morphological and Functional Features of a Normal Right Ventricle

The right ventricle is irregular triangular pyramid in shape, which base is directed to the right atrium. Features

of the right ventricle structure include the presence of the three main anatomic areas: sinus - inflow tract, which includes components of the tricuspid valve (annulus, three leafs - front, bottom, septal, leafs' chords and papillary muscles), top and cone - outflow tract, smooth formation, separated from the inflow tract by four muscle structures: the most massive of the heart walls - interventricular septum, parietal, and moderatory and septal surfaces [3]. Three pulmonary valve leafs (right, left, front) are located in the top of the outflow tract. Tricuspid valve and pulmonary artery valve have no common annulus, unlike the mitral and aortic valves. Thinner (3.5 mm) and more stretchable walls of the right ventricle in comparison with the left ventricle wall determine better excess volume tolerability but not the cavity pressure increase. As the pressure in the pulmonary artery increases systolic dysfunction with preserved ejection fraction develops, followed by systolic dysfunction with reduced ejection fraction of the right ventricle after a short period of myocardial adaptation in the form of hypertrophy [3].

Blood supply of the right ventricle is predominantly performed by right coronary artery. 1-2 branches depart from artery supplying the equal flow of the right ventricle anterior wall in systole and diastole. The left anterior descending coronary artery supplies the front two-thirds of the interventricular septum and the posterior descending artery - papillary muscles of the right ventricle, the posterior wall of the right ventricle and the posterior part of the interventricular septum [3].

Etiology And Mechanisms of Chronic Right Heart Failure

Violations of the right ventricle function are formed as a result of many factors, including myocardium, endocardium, pericardium, blood vessels of the pulmonary circulation, pulmonary parenchyma lesions (Table 1).

A common cause of the right ventricular dysfunction is pulmonary hypertension. In the case of pulmonary hypertension right ventricle changes occur under the influence of different clinical conditions and diseases presented in the Recommendations for the diagnosis and treatment of pulmonary hypertension of the European Society of Cardiology and the European Respiratory Society, proposed in 2009 and amended in 2015 [4]. The classification of pulmonary hypertension included 5 groups of clinical conditions:

- Pulmonary hypertension with veno-occlusive disease of the lungs, and persistent pulmonary hypertension of newborns.
- Pulmonary hypertension due to the pathology of the left heart chambers.
- Pulmonary hypertension due to lung diseases and / or hypoxia.
- Chronic thromboembolic pulmonary hypertension and other pulmonary artery obstruction.
- Pulmonary hypertension with unclear or multiple mechanisms.

The main cause	Diseases and conditions
Diseases of the myocardium	CHD - ischemia, RV myocardial infarction; genetic diseases – HC, ARVD, DCM
Volume or pressure overload of right ventricle	Pulmonary arterial hypertension, pulmonary hypertension; Congenital and acquired defects of the tricuspid valve, pulmonary valve, ASD, VSD, RL shunt defects; Pericardial disease - pericardial effusion, constrictive pericarditis

Table 1: Main etiological factors of chronic right heart failure.
Note: CHD: Coronary Heart Disease; RV: Right Ventricle; HC: Hypertrophic Cardiomyopathy; ARVD: Arrhythmogenic Right Ventricular Dysplasia; DCM: Dilated Cardiomyopathy; ASD: Atrial Septal Defect; VSD: Ventricular Septal Defect.

The right ventricle overload resulting from increase in the pulmonary artery pressure and pulmonary vascular resistance leads to compensatory hypertrophy and interstitial fibrosis. Hypertrophied myocardium maintains normal or increased contractile function of the right ventricle for a short period of time, which supports the required stroke volume, at the same time diastolic relaxation is reduced. As the progression of the right ventricle dilatation development, its shape is transformed into a spherical, providing a more substantial mechanical force during systole. Myocardial oxygen demand and myocardial stiffness are increased; coronary perfusion is reduced (especially in the endocardium). Right ventricle wall thickness is reduced later, amplifying its systolic strain and increasing demand in myocardial blood supply. Myocardial contractility becomes weak and the signs of

decompensation occur, characterized by elevated filling pressure, diastolic dysfunction and a decrease in cardiac output of the right ventricle [1]. Stretching of right atrioventricular ring and the appearance of tricuspid insufficiency lead to volume overload of the right ventricle, reduced volume of blood ejected by the pulmonary circulation, reduced left ventricle filling. The appearance of clinical signs of right heart failure of III or IV functional class (NYHA) indicates a high risk of death (> 10%) within a year for patients with pulmonary hypertension [4]. Thereby violation of right ventricle function in pulmonary hypertension due to different clinical conditions affects the prognosis of patients' lives.

Left ventricle heart failure is believed to be a common cause of right ventricle dysfunction [5]. Mechanisms of right ventricle failure development in this case are different and have not yet been completely determined:

- As a result of reduction of the left ventricle contractility afterload, the pressure in the left atrium, the pulmonary veins, pulmonary arteries, in the right ventricle increase [5].
- Abnormalities in cardiomyopathies occur simultaneously in the left and right ventricles [6].
- Blood flow impairments in coronary heart disease (ischemia) may occur in both ventricles simultaneously.
- As a result of reduction of left ventricle function perfusion of the right ventricle is decreased.
- Displacement of the interventricular septum in case of the volume or pressure increase in the left ventricle leads to right ventricle dysfunction.
- The appearance of effusion or constrictive pathological process in the pericardial cavity results in impaired function of both left and right ventricles of the heart.

The progression of congestive heart failure is compounded by yet another mechanism that causes right ventricle failure. As a result of impaired blood flow in the pulmonary circulation and impaired ventilation the diffusion of gases is affected, the diffusion capacity of the alveolar-capillary membrane and blood gas composition are reduced.

Right ventricle dysfunction appearing as a result of the left heart damage is considered to be a pessimistic prognostic factor in many diseases (ischemic cardiomyopathy, acute myocardial infarction of the left ventricle, myocarditis). Right ventricle function, assessed by radionuclide methods of investigation, in congestive heart failure of II and III functional class (NYHA), is treated as an independent predictor of patient survival [4].

The role of many factors, including the violation of neurohumoral and immune regulation is noted in the development and progression of right heart failure. Increase in the activity of the renin-angiotensin-aldosterone and sympathoadrenal systems, endothelial dysfunction, metabolic disturbances, changes in ion channel activity involved in the processes of contraction and relaxation of cardiomyocytes are determined [1]. Genetic studies indicate the development of the right and left ventricles from the different cells during embryogenesis and different regulation of the right and left ventricular function with pressure overload [7].

Clinical Signs of Chronic Right Ventricle Failure

Complaints and objective evidence of right heart failure are non-specific, complaints of the disease resulting in right heart failure are essential.

The main symptoms are signs of heart failure: shortness of breath, orthopnea, weakness, fatigue, decreased exercise tolerance, dizziness, abdominal discomfort, pain in the heart and / or in the right upper quadrant, hemoptysis, wheezing, swelling of the ankles [7].

Physical signs include the appearance of pulsation on the left side of the sternum in the fourth intercostal space, increased right ventricle size (with significant hypertrophy and / or dilatation), and auscultation of the heart - increased second tone in the second intercostal space on the left, pansystolic murmer in the case of tricuspid insufficiency, Graham-Stille murmur, arrhythmia. Signs of right heart failure include pulsation of neck veins, hepatomegaly, peripheral edema, ascites.

Instrumental Diagnostic Methods

The most informative and accessible in clinical practice methods of right heart failure diagnostics are echocardiographic method and the level of atrial and brain natriuretic peptide estimation. Routine research methods (electrocardiography, radiography) are performed, they give a complementary notion of right ventricular failure.

Electrocardiographic examination reveals the changes caused by the myocardium, pericardium damage or heart disease, signs of right ventricle overload or hypertrophy, right atrium dilatation or hypertrophy. However some signs show low specificity - the expansion of the QRS complex, conduction disorders in the form of a right bundle branch block and increased P wave voltage. Ventricular arrhythmias are rare, supraventricular arrhythmias are common in the form of extrasystoles, atrial fibrillation.

X-ray examination of the chest reveals changes characteristic for diseases causing pulmonary hypertension, pulmonary interstitial lesions, damage of the pulmonary circulation vessels, increase in heart size and shape changes. In the case of pulmonary hypertension increased transparency of the lung fields in the periphery due to the depletion of a pulmonary pattern, bulging trunk and the left pulmonary artery branch, forming the second arc of the left cardiac loop (direct view), the expansion of the lungs roots, increased size of the right heart are observed [4].

The most informative method for the diagnosis of heart failure is echocardiography. Transthoracic and transesophageal echocardiography allows to assess the condition of the heart chambers, wall thickness, structure and function of the valve apparatus, the state of the initial portion of the aorta and pulmonary artery with pressure measurement in the latter. Echocardiographic method refers to the basic techniques of non-invasive assessment of right ventricle systolic and diastolic function [8].

Assessment of right ventricle structure and function is carried out using many echocardiographic positions, including positions on parasternal long and short axis, position for the visualization of the right ventricular outflow tract, apical four-chamber and subcostal position.

Since the pliability of the right ventricle wall is high, with an increase in afterload - pulmonary vascular resistance and the load caused by the left side of the heart, there is a concentric hypertrophy and dilatation of the right ventricle, measured at basal (normal RVD1 – 2.0-2.8 cm), median (normal RVD2 – 2.7-3.3 cm) diameter and the longitudinal dimension (normal RVD3 – 7.1-7.9 cm) [8,9].

The amplitude of systolic motion of the lateral tricuspid ring is calculated – it is highly specific indicator of systolic excursion of the tricuspid ring plane (TAPSE - tricuspid annular plane systolic excursion). TAPSE <1.8 cm indicates violation of right ventricle function and increasing the pressure in the pulmonary circulation. According to Forfia PR, et al. 1 mm TAPSE decrease in patients with pulmonary arterial hypertension increases the risk of death by 17% [9].

Echocardiographic method allows to estimate the probability of pulmonary hypertension on the basis of tricuspid regurgitation peak velocity (m/sec), the time of blood flow acceleration in the outflow tract of the right ventricle <105 m/sec and/or midsystolic cover-up, flattening of the interventricular septum, the early diastolic velocity of pulmonary regurgitation> 2.2 m/sec, an increase in pulmonary artery diameter> 25 mm [8].

The degree of pulmonary hypertension severity is determined by the difference between systolic and average pressure in the pulmonary artery, calculated using the Bernoulli equation in the presence of tricuspid insufficiency. A semi-quantitative assessment of the pressure in the right atrium is carried out according to the diameter and degree of inspiratory inferior vena cava collapsing. The diameter of the inferior vena cava > 2.1 cm and its inspiratory collapsing <50% or <20% during quiet breathing indicate high right atrial pressure (10-20 mm Hg). The increase in the area> 18 cm^2 shows increasing size of the right atrium [8].

Unlike the estimated left ventricular ejection fraction, which has a regular shape, the calculation of the right ventricular ejection fraction causes problems because of its shape and disadvantages of standard methods of right ventricular volume assessment. Indicators of right ventricular area change fraction (FAC - Fractional Area Change) are used for quantitative estimation, correlated with ejection fraction of the right ventricle (r = 0.88) according to magnetic resonance imaging [10].

Further assessment of right ventricle function is performed by tissue Doppler study of the tricuspid valve ring velocity or myocardial performance index (Tei index). Performance Index (MPI - Myocardial Performance Index) is calculated by the ratio of the total time of isovolumic activity to the time spent on the systolic expulsion using the formula (IVRT+IVCT)/ET, where IVRT - isovolumic relaxation time; IVCT - isovolumic contraction time; ET - ejection time [10]. Tissue Doppler study allows to study the deformation of the right ventricle and the rate of deformation (Strain/Strain-Rate) - the markers of contractile force, independent of preload.

Strain/Strain-Rate figures reflect regional function of the right ventricle more accurately compared with TAPSE

[10]. Echocardiographic changes in the right ventricle appear in lesions> 25% of the vascular bed of the pulmonary circulation [10].

The three-dimensional echocardiography in real time (RT3DE) reconstructs contours of structures and heart chambers, evaluating the right ventricle and atrium dimensions. Researchers note comparability of volume parameters and right ventricle ejection fraction obtained by RT3DE and magnetic resonance imaging, a high correlation was noted for the global longitudinal strain (RV GLS) [12].

Imaging techniques are used to assess the state of the right ventricle: high-resolution computed tomography, magnetic resonance imaging and radionuclide angiography study.

High-resolution computed tomography, computerized tomography angiography give data on pulmonary artery width (normal <29 mm) or the right ventricle, pulmonary diseases, vascular condition of the pulmonary circulation, congenital heart diseases and other causes of pulmonary hypertension.

Magnetic resonance imaging is the most accurate method of estimation of the size, structure and function of the right ventricle. This method allows to measure blood flow including stroke volume, cardiac output, pulmonary artery distensibility, right ventricle mass.

Catheterization of the right heart chambers is carried out to assess the severity of hemodynamic changes including measurement of pulmonary artery pressure, right atrium pressure, right ventricle pressure, pulmonary artery wedge pressure in pulmonary hypertension and right heart failure and performance of pulmonary vessels vasoreactivity test [13].

Vasoreactivity test is carried out for pulmonary hypertension patients to determine appropriate treatment with high doses of calcium channel blockers. Reducing the average pulmonary artery pressure ≥10 mm Hg up to the absolute value of the mean pulmonary artery pressure ≤40 mm Hg combined with an increased or unchanged cardiac output are regarded as a positive test after the inhalation of nitric oxide or intravenous epoprostenolol either adenosine injection (or inhaled iloprost) [4].

Methods confirming the presence of chronic heart failure include the determination of blood plasma levels of natriuretic peptides: atrial (ANP - atrial natriuretic peptide) and brain natriuretic peptide (BNP - brain natriuretic peptide) and peptide precursor pro-BNP, N-terminal fragment of peptide precursor - NT-proBNP [7]. Secretion of hormones is increased in case of an organic lesion and volume overload of atrial myocardium (ANP), ventricular myocardium (BNP), especially with the increase in end-diastolic pressure in the left ventricle [14,15].

In case of the gradual development of chronic heart failure symptoms diagnostic value of BNP confirming the diagnosis is the level of more than 35 pg/ml, and NT-proBNP more than 125 pg/ml [3]. Human BNP is synthesized as pre-prohormone by ventricular, atrial cardiomyocytes, fibroblasts, brain cells. In response to ventricular cardiomyocytes stimulation (e.g., stretching of the myocardium) proBNP is released, then it subsequently splits into two fragments - active BNP hormone and inactive N-terminal peptide (NT-proBNP).

Physiological effects of natriuretic peptides include reduction of the hemodynamic myocardium load, its increased concentration inhibits excessive activity of renin-aldosterone and sympathetic-adrenal systems increasing during the progression of chronic heart failure. ESC Guidelines for the diagnosis and treatment of heart failure (2001) show indications for natriuretic peptides detection: screening tests to identify patients with a high probability of heart failure; diagnosis of heart failure in the early stages; evaluation of the heart failure therapy effectiveness; assessment of disease prognosis in patients with heart failure [16].

According to the 2016 ESC Recommendations for the diagnosis and treatment of heart failure patients with heart failure and preserved ejection fraction ≥50% (HFpEF), as well as being in a "gray zone" - an ejection fraction of 40% - 49% (HFmrEF) are recommended to determine the concentration of BNP in addition to the assessment of symptoms and signs during echocardiographic study [17].

The detection of blood plasma atrial natriuretic peptide concentration is recommended in case when congestive heart failure is suspected. Heart failure is unlikely with normal range of natriuretic peptide concentration.

Apart from heart failure increased plasma levels of natriuretic peptides have been reported with many conditions and diseases (Table 2) [18].

Cardiac	Heart failure
	Acute coronary syndrome
	Pulmonary embolism
	Myocarditis
	Left ventricular hypertrophy
	Hypertrophic and restrictive cardiomyopathy
	Valvular heart disease
	Atrial and ventricular tachyarrhythmia
	Bruising of the heart
	Surgical procedures involving the heart
	Pulmonary hypertension
Non-Cardiac	Elderly age
	Ischemic stroke
	Subarachnoid hemorrhage
	Kidney failure
	Liver failure (cirrhosis with ascites)
	Paraneoplastic syndrome
	Chronic obstructive pulmonary disease
	Severe infections (including pneumonia and sepsis)
	Severe burns
	Anemia
	Severe hormonal and metabolic disorders (e.g. hyperthyroidism, diabetic ketoacidosis)

Table 2: The cardiac and non-cardiac causes of natriuretic peptides concentrations increase in plasma.

Low levels of BNP and NT-proBNP mark obesity [18].

To sum it up, right ventricle heart failure is caused by many conditions and diseases, including acquired diseases of the myocardium (coronary heart disease) and genetic diseases of the myocardium (cardiomyopathy), pulmonary arterial hypertension and pulmonary hypertension, characteristic for many lung and heart diseases, pericardial lesions. Clinical signs and symptoms of right heart failure appear on the late stages and are often veiled by symptomatic left ventricle heart failure. An integrated approach aimed at identifying the possible causes of right heart failure and signs of structural and functional changes in the right ventricle, obtained by instrumental and laboratory tests, among which echocardiographic study and concentration in blood plasma natriuretic peptides are the most informative, is required for timely diagnosis.

References

1. Ho SY, Nihoyannopoulos P (2006) Anatomy, echocardiography, and normal right ventricular dimensions. Heart 92(1): i2-i13.

2. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, et al. (2010) Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr 23(7): 685-713.

3. Voelkel NF, Quaife RA, Leinwand LA, Barst RJ, McGoon MD, et al. (2006) Right ventricular function and failure: report of a National Heart, Lung, and

Blood Institute working group on cellular and molecular mechanisms of right heart failure. Circulation 114(17): 1883-1891.

4. Galiè N, Humbert M, Vachiery JL, Gibbs S, Lang I, et al. (2015) 2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. Eur Heart J 46: 903-975.

5. Haddad F, Hunt SA, Rosenthal DN, Murphy DJ (2008) Right ventricular function in cardiovascular disease, part I: Anatomy, physiology, aging, and functional assessment of the right ventricle. Circulation 117(11): 1436-1448.

6. Skhiri M, Hunt SA, Denault AY, Haddad F (2010) Evidence-Based Management of Right Heart Failure: a Systematic Review of an Empiric Field. Rev Esp Cardiol 63(4): 451-471.

7. Sheehan F, Redington AN (2008) The right ventricle: anatomy, physiology and clinical imaging. Heart Vessels 94(11): 1510-1515.

8. Melenovsky V, Hwang SJ, Lin G,

9. Kjaergaard J (2012) Assessment of Right Ventricular Systolic Function by Tissue Doppler Echocardiography. Dan Med J 59(3): B4409.

10. Zakeri R, Mohammed SF (2015) Epidemiology of Right Ventricular Dysfunction in Heart Failure with Preserved Ejection Fraction. Curr Heart Fail Rep 12(5): 295-301.

11. Giusca S, Dambrauskaite V, Scheurwegs C, D'hooge J, Claus P, et al. (2010) Deformation imaging describes right ventricular function better than longitudinal displacement of the tricuspid ring. Heart 96(4): 281-288.

12. Maceira AM, Prasad SK, Khan M, Pennell DJ (2006) Reference right ventricular systolic and diastolic function normalized to age, gender and body surface area from steady-state free precession cardiovascular magnetic resonance. Eur Heart J 27(23): 2879-2888.

13. Sadeghpour A, Alizadehasl A (2015) The Right Ventricle: A Comprehensive Review From Anatomy, Physiology, and Mechanics to Hemodynamic, Functional, and Imaging Evaluation. Arch Cardiovasc Imaging 3(4): e35717.

14. Anavekar NS, Gerson D, Skali H, Kwong RY, Yucel EK, et al. (2007) Two-dimensional assessment of right ventricular function: an echocardiographic-MRI correlative study. Echocardiography 24: 452-456.

15. Pruszczyk P (2005) N-terminal pro-brain natriuretic peptide as an indicator of right ventricular dysfunction. J Cardiac Fail 11: S65-S69.

16. Remme WJ, Swedberg K (2001) Guidelines for the diagnosis and treatment of heart failure. Eur Heart J 22(17): 1527-1560.

17. Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland JG, et al. (2016) 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J 37(27): 2129-2200.

18. Madamanchi C, Alhosaini H, Sumida A, Runge MS (2014) Obesity and natriuretic peptides, BNP and NT-proBNP: Mechanisms and diagnostic implications for heart failure. Int J Cardiol 176(3): 611-617.