

## **MEASURING BODY COMPOSITION IN PATIENTS WITH CHRONIC HEART FAILURE AND TYPE II DIABETES MELLITUS**

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Diabetes mellitus (DM) is an equally pressing global health problem, with its prevalence doubling every 10-15 years. By 2030, it is projected to affect one in 15-20 people worldwide, meaning the number of people affected will increase to more than 400 million (Askerov M.M., 2013). According to the International Diabetes Federation (IDF), the prevalence of diabetes among the adult population in 2014 was 8.5% (Smirnov I.I. et al., 2007).

In the last decade, it has become clear that the unfavorable prognosis of chronic heart failure with preserved ejection fraction (CHF-PEF) is determined by the number and severity of structural and functional myocardial rearrangements, as well as changes and intensity of intramyocardial interactions. The developmental paradigm for chronic heart failure (CHF) suggests the following sequence of events: 1) comorbid conditions and diseases, such as overweight/obesity, type 2 diabetes mellitus (T2DM), chronic obstructive pulmonary disease (COPD), and arterial hypertension (HTN), lead to the development of low-grade, asymptomatic systemic inflammation; 2) it gradually affects the endothelial glycocalyx of the coronary vascular bed and microvascular collaterals; 3) generalized damage to the glycocalyx with the development of endotheliopathy destabilizes the vascular wall with an increase in its permeability and paracellular transport; 4) this results in myocardial infiltration with cardiotoxic, inflammatory and profibrotic agents, a decrease in the bioavailability of vasoactive mediators (nitric oxide and cyclic guanosine monophosphate) and the activity of protein kinase G in cardiomyocytes; 5) this, in turn, causes hypertrophy and a decrease in myocardial elasticity due to titin hypophosphatation;

6) this results in the development of cardiomyocyte stiffness and progressive interstitial fibrosis, leading to diastolic stiffness of the left ventricle and SBP. Myocardial remodeling in CHF-EF differs from that in CHF with a low ejection fraction (CHF-LFEF) due to the loss of cardiomyocytes and structural depletion of the syncytium with the development of predominantly eccentric remodeling, volume overload, and persistent neurohumoral activation [16, 31]. Diagnosis of CHF at an early stage of myocardial remodeling allows for timely initiation of treatment, thereby improving the prognosis and quality of life of patients. Currently, the most informative diagnostic tools are echocardiography, diastolic stress test, and determination of brain natriuretic peptide (BNP) and its N-terminal propeptide (NT-proBNP) [5]. However, these methods have low diagnostic specificity both in the early stages of CHF and in conditions of a stable course of an already formed disease. An alternative is the possibility of identifying a genetic predisposition to CHF, which significantly helps in identifying risk groups. Early detection of known gene polymorphisms associated with CHF has valuable prognostic value [2], but does not allow for the accurate determination of the early onset of the disease and timely initiation of preventive treatment. The presence of predisposition genes suggests the existence of altered metabolic pathways involving the corresponding protein structures involved in the genesis of CHF. Despite the polyetiological nature of CHF, these pathways are apparently associated with the effective functioning of compensatory mechanisms. Therefore, elucidation of the components of the metabolic pathways that determine the development of CHF in predisposed patients is a pressing task aimed at developing new methods for the early noninvasive diagnosis of CHF.

Cachexia is common in the late stages of many chronic diseases, but is rarely diagnosed. Chronic heart failure (CHF) frequently leads to cachexia, with an estimated prevalence of 5% to 15%. Cachexia is an unfavorable prognostic sign in patients with CHF. Patients with cachexia (defined as non-edematous and unintentional weight loss of more than 7.5% of their previous weight for at least 6 months) have a 50% mortality rate within 18 months.

Cachexia is characterized by muscle loss with or without fat loss. Defining cachexia is difficult, although consensus statements suggest features such as a weight loss of at least 5% (without edema) over 1 year or a body mass index (BMI) less than 20 kg/m<sup>2</sup>, along with other symptoms or signs. A weight loss of more than 6% over 9-12 months in patients with CHF is a strong predictor of poor outcome. Conversely, weight gain is associated with an improved prognosis.

Detecting cachexia in patients with CHF can be particularly challenging due to changes in extracellular fluid. Patients may lose muscle and fat mass while their overall weight remains unchanged or even increases. Measuring weight or BMI alone may not detect a decrease in lean mass (LM) or fat mass (FM). Therefore, body composition measurements can help detect cachexia. We compared segmental body composition in patients with CHF using two devices: dual-energy X-ray absorptiometry (DXA), considered the gold standard for body composition analysis, and multi-frequency bioelectrical impedance analysis (BIA).

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