BODY COMPOSITION COMPONENTS IN PATIENTS WITH CHRONIC HEART FAILURE AND TYPE 2 DIABETES MELLITUS

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Abstract

There is growing evidence that body composition should be considered as a systemic marker of disease severity in chronic organ diseases such as chronic obstructive pulmonary disease, chronic kidney disease, and chronic heart failure (CHF). This article focuses on changes in body composition components in patients with chronic heart failure and type 2 diabetes mellitus.

Keywords: chronic heart failure, diabetes mellitus, body composition components, cachexia, obesity.

SURUNKALI YURAK YETISHMOVCHILIGI BOR VA II TIP QANDLI DIABETI MAVJUD BEMORLARDA TANA TARKIBI KOMPONENTLARINING QIYOSIY TAHLILI

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Annotatsiya

Surunkali obstruktiv o'pka kasalligi, surunkali buyrak kasalligi va surunkali yurak yetishmovchiligi (SYE) kabi surunkali organ kasalliklarida tana tarkibi kasallikning og'irligining tizimli belgisi sifatida qaralishi kerakligi haqida tobora ko'proq dalillar mavjud. Ushbu maqola surunkali yurak yetishmovchiligi va 2-toifa diabet bilan og'rigan bemorlarda tana tarkibi komponentlarining o'zgarishiga qaratilgan.

Kalit so'zlar: surunkali yurak yetishmovchiligi, diabet, tana tarkibi komponentlari, kaxeksiya, semirish.

КОМПОНЕНТЫ СОСТАВА ТЕЛА У ПАЦИЕНТОВ С ХРОНИЧЕСКОЙ СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТЬЮ И САХАРНЫМ ДИАБЕТОМ II ТИПА

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Аннотация

Появляется все больше доказательств того, что состав тела следует рассматривать как системный маркер тяжести заболевания при хронических заболеваниях органов, таких как хроническая обструктивная болезнь легких, хроническая почечная недостаточность и хроническая сердечная недостаточность (ХСН). Данная статья посвящена на изменение компонентов состава тела у пациентов хронической сердечной недостаточностью и сахарным диабетом II типа.

Ключевые слова: хроническая сердечная недостаточность, сахарный диабет, компоненты состава тела, кахексия, ожирение.

There is growing evidence that body composition should be considered a systemic marker of disease severity in chronic organ diseases such as chronic obstructive pulmonary disease, chronic kidney disease, and chronic heart failure (CHF). A large number of clinical studies have shown that involuntary weight loss, commonly referred to as "cachexia syndrome," is associated with increased morbidity and mortality regardless of disease severity. Notably, mortality rates are higher in underweight and normal-weight patients compared to overweight and even obese patients. This relationship differs from the U-shaped survival curve typically observed for BMI in large population studies, which show increased mortality with higher BMI due to increased fat mass (FM). This difference may be related to the specific adverse effects of excessive loss of metabolically and functionally active lean mass (LFM) on functional impairment and associated morbidity and mortality in chronic wasting disease, regardless of LFM.

Muscle mass is the largest component of lean body mass and the primary determinant of skeletal muscle strength. In patients with advanced COPD or chronic heart failure, skeletal muscle dysfunction is considered a better predictor of exercise intolerance than airflow obstruction or left ventricular ejection fraction, respectively. In patients with COPD, loss of lean body mass is consistently observed not only in underweight or lean individuals but also in normal-weight individuals.

Understanding the molecular regulation of muscle atrophy and hypertrophy is a hot topic in experimental research, and significant progress has been made in recent years, which should lead to the development of new pharmacological interventions in the near future. Furthermore, rehabilitation programs are now part of modern heart failure therapy to improve impaired exercise tolerance, and the effects of various rehabilitation regimens on body composition and functional capacity are well studied.

Body composition is important to include in the clinical characterization of patients with CHF for several reasons: to select patients at risk for or actually suffering from (hidden) loss of BM; to identify and evaluate current and new rehabilitation strategies; and to adjust peak oxygen consumption during additional exercise, which

is commonly used as a discriminatory criterion in selecting patients for surgery or transplantation, taking into account the absence of menstrual cycles, which is a better predictor of peak oxygen consumption (VO2) than body weight.

Data on the nature of tissue wasting in patients with CHF are limited. The lack of clinical data may be due to the fact that the two-compartment model of body composition (i.e., separating FFM and FM) is not applicable to patients with congestion, since water retention selectively increases the extracellular water compartment and therefore overestimates FFM, while two-compartment models assume a stable ratio between the intracellular and extracellular water compartments.

Soon after the first description of the relationship between body mass index (BMI) and survival after heart failure (HF), several studies were devoted to finding explanations for the so-called "obesity paradox." Two main hypotheses were proposed. The first proposed that fat, by blocking tumor necrosis factor and lipopolysaccharide production or by producing adipokines, might have a protective effect. The second proposed that a high BMI might be associated with protective factors such as younger age, higher amounts of muscle and serum protein, greater muscle strength, and better functional capacity.

It is important to remember that the obesity paradox is based on BMI. However, obesity is defined as an abnormal accumulation of body fat [7], and BMI measures weight relative to height, not the amount of fat.

HF is a congestive condition, and even non-decompensated patients have excess fluid compared to the normal population [8]; Thus, some patients considered normal weight may actually be underweight. However, few studies have included nutritional parameters other than BMI in their assessment of the obesity paradox. There is no universal standard for assessing nutrition, and heart failure, a chronic disease associated with wasting and water retention, may require specific considerations.

In the last decade, it has become clear that the poor prognosis of chronic heart failure with preserved ejection fraction (CHF-EPF) is determined by the number and severity of structural and functional myocardial changes, as well as the changes and intensity of intramyocardial interactions. The paradigm of chronic heart failure (CHF) development assumes 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 sluggish, 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, increasing its permeability and paracellular transport; 4) this leads to myocardial infiltration with cardiotoxic, inflammatory and profibrotic agents, decreased bioavailability of vasoactive mediators (nitric oxide and cyclic guanosine monophosphate) and protein kinase G activity in cardiomyocytes; 5) this,

in turn, causes hypertrophy and decreased elasticity of the myocardium due to titin hypophosphatation; 6) this leads to the development of cardiomyocyte stiffness and progressive interstitial fibrosis, which leads 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-LF) 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 methods 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 the 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 an 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 polyetiology of CHF, these pathways are apparently associated with the effective functioning of compensatory mechanisms. Therefore, identifying the components of metabolic pathways that determine the development of CHF in predisposed patients is an urgent task aimed at developing new methods for early non-invasive diagnosis of CHF.

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