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THE ROLE OF BIOMARKERS IN PREDICTING OF HEART FAILURE IN PATIENTS WITH TYPE 2 DIABETES MELLITIS

«Literature review»

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✓ ***Resume***

In this article, the authors performed a review of literature on the value of biomarkers in predicting complications after endovascular revascularization in patients with diabetes mellitus type 2. The authors concluded that the future use of biomarkers with heart failure stay on the multimarcere panel strategy, which will include a specific combination of biomarkers (galectin 3, growth differentiation factor-15, GDF-15), renal dysfunction (lipocalin associated with neutrophil gelatinase, NGAL; kidney damage molecule-1, KIM-1), neurohumoral activation (adrenellin, MR-PROADM; Coppentine) and oxidative stress (Ceruloplasmin; Myeloperoxidase, MPO; 8-hydroxy-2'-deoxyiguanosine, 8-OHDG; Tyoracedoxin 1, TRX1) in patients with heart failure.

Key words: diabetes mellitus 2 type, ischemic heart disease, markers

РОЛЬ БИОМАРКЕРОВ В ПРОГНОЗИРОВАНИИ СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТИ У БОЛЬНЫХ САХАРНЫМ ДИАБЕТОМ 2 ТИПА

«Обзор литературы»

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✓ ***Резюме***

В данной статье авторы провели обзор литературы о значении биомаркеров в прогнозировании осложнений после эндоваскулярной реваскуляризации у больных сахарным диабетом 2 типа. Авторы пришли к выводу, что дальнейшее использование биомаркеров при сердечной недостаточности остается на стратегии мультимаркерной панели, которая будет включать специфическую комбинацию биомаркеров (галектин 3, фактор дифференцировки роста-15, GDF-15), почечную дисфункцию (липокалин, связанный с нейтрофильной желатиназой, NGAL; молекула повреждения почек-1, KIM-1), нейрогуморальную активацию (адренеллин, MR -PROADM; коппентин) и окислительный стресс (церулоплазмин; миелопероксидаза, МПО; 8-гидрокси-2'-дезоксиигуанозин, 8-OHDG; тиорацедоксин 1, TRX1) у больных с сердечной недостаточностью.

Ключевые слова: сахарный диабет 2 типа, ишемическая болезнь сердца, маркеры.

II - TOIFA QANDLI DIABET BILAN OG'RIGAN BEMORLARDA YURAK ETISHMOVCHILIGINI BASHORAT QILISHDA BIOMARKERLARNING ROLI

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✓ ***Rezyume***

Ushbu maqolada mualliflar 2-toifa qandli diabet bilan og'rigan bemorlarda endovaskulyar revaskulyarizatsiyadan keyin asoratlarni bashorat qilishda biomarkerlarning ahamiyati haqidagi adabiyotlarni ko'rib chiqdilar. Mualliflarning xulosasiga ko'ra, yurak etishmovchiligida biomarkerlardan keyingi foydalanish multimarkerlar paneli strategiyasida qoladi, bunda biomarkerlarning o'ziga xos kombinatsiyasi (galektin 3, o'sish differentsiatsiyasi omili-15, GDF-15), buyrak disfunktsiyasi (neytrofil jelatinaza bilan bog'liq lipokalin, neytrofil jelatinaza, NGAL bilan bog'liq; buyrak shikastlanishi molekulasi-1, KIM-1), neyrohumoral faollashuv (adrenellin, MR-PROADM; kopeptin) va oksidlovchi stress (seruloplazmin; miyeloperoksidaza, MPO; 8-gidroksi-2'-deoksiguanozin, 8-OHDG; tioradoksin11) yurak etishmovchiligi bo'lgan bemorlarda.

Kalit so'zlar: 2-toifa qandli diabet, yurak tomirlari kasalligi, markerlar.

Background

Over the past decades, as is known, type 2 diabetes mellitus (DM 2) is one of the most common diseases. In addition, according to medical and social significance, the DM 2 type occupies 3rd place after cardiovascular and oncological diseases. It was shown in the works that DM 2 type and heart failure (HF) mutually deteriorate the course and forecast of each other. Thus, the increased blood content of glucose deteriorates both metabolism in myocardial tissues and the functional-structural condition of the heart. At the same time, the HF worsens a significantly course of the type of type 2, causing pathogenetic shifts that contribute to an increase, and so high glucose levels. [43, 44].

One of the classical definitions reads: "HF is a pathophysiological condition in which the anomaly of the heart function is responsible for the inability of the heart to pump blood at a rate commensurate with the needs of metabolizing tissues, or only at elevated filling pressure. " [1].

The recommendations of the American College of Cardiologists (ACC) / American Cardiology Association (ACA) define "HF as a complex clinical syndrome that occurs as a result of any structural or functional violation of ventricular filling or blood emissions." [2].

Recognizing the importance of predisposing states in the etiology of HF. ACC / AHA allocates 4 stages of HF. Stages C and D are clinically recognized forms, while Stages A and B are forms to HF. [2].

Traditionally, it is classified as HF with reduced emission fraction (HFrEF) and HF with saved EF (HFsEF). The HFrEFF is usually defined as LVEF <40%, and the HFsEF is usually considered as LVEF ≥50%. This difference is very important, since both of these forms have different etiology, risk profiles, demographics and related diseases.

Many studies have been confirmed that the DM2 type and HF are connected bi-directionally, namely there are an increased risk of HF's development at DM2 type, and HF is a factor of the development risk of DM 2 type. Such an association of DM 2-type and HF meets very often and has a surrounding impact on the choice of treatment tactics and a patient's forecast. It is believed that with an increase in the population, its aging, increasing the prevalence of obesity and atherosclerosis, the frequency of development of HF and DM 2 type will noticeably increase. [39].

Officially, the researchers adopted two Phenotype with a DM type 2- it's a low emission fraction (HFrEF), or a dialing phenotype, and HF with a stored emission fraction (HFsEF), or restrictive phenotype [40, 41]. Clinically, the presence of these phenotypes is justified by the fact that, according to the results of the leading tests of drugs for the treatment of HF, HFrEF and HFsEF, are clinical variants of the disease with different pathophysiological links and responses to the therapy conducted [42].

The prevalence of HF. Heart failure (HF) amazes about 26 million people all over the world and its management creates a significant burden on the country's health care resources. Aging of the population, an increase in the prevalence of cardiological risk factors and improving the survival of patients with acute cardiovascular diseases led to the fact that HF became a serious public health problem worldwide. HF is one of the main causes of hospitalization in high-income countries (HIC), which accounts for from 1% to 2% of the total number of hospitalizations. [3]. HF is a disease associated with significant mortality, which is higher than in many common types of cancer, such as breast cancer or colon. It is also associated with high morbidity and constitutes a significant share of health expenditures in developed countries. [4]. . Although we have good data on HF epidemiology, data from low and middle-income countries (MIC) are very limited, and most of them are forecasts based on Western data or based on risk factors. [5, 6].

According to Nhanes, updated [7]. According to cardiovascular diseases and strokes in 2017, it is reported that 6.1 million Americans at the age of ≥20 years suffer from HF. [8]. According to the forecast data, the prevalence of HF in the United States will increase by 46% from 2012 to 2030

Key Biomarkers of HF diagnostics with type 2 DM. Routine diagnostic assessments of HF should include laboratory tests, including HF Biomarkers, which can be used to assist in diagnostics and as prognostic predictors. The use of biomarkers for the diagnosis of HF is more convenient than echocardiography, as a first line tool in outpatient services or emergency departments. However, these biomarkers can also be increased not only with HF. Therefore, biomarkers should be used with caution and limited by the exception, especially in patients with atypical manifestations. [twenty].

A multicenter study performed by USA, Australia and Canadian clinics in 2019, included 263 patients who were randomized to receive Dapagliflowin at a dose of 10 mg per day or placebo for 12 weeks. [45]. In patients with heart failure and reduced ejection fraction, the use of Dapagliflozin for 12 weeks did not affect the average level of NT-PROBNP, but increased the proportion of patients who had a clinically significant improvement in the state of health associated with HF, or sodium systemic peptides.

Another study was also a multicenter, in the USA in 2021 its data in patients with SD 2 and HSN were published. [46]. The assessment of biomarkers included highly sensitive heart troponin T ≥6 ng / l, N-terminal sodium peptide pro-B-type ≥125 pg / ml, highly sensitive C-jet protein ≥3 mg / l and left ventricular hypertrophy according to electrocardiography, 1 point For each pathological parameter. The 5-year risk of HF was evaluated among participants with diabetes and prediabet for groups of assessing biomarkers (from 0 to 4). It was concluded that among adults with diabetes and predominant, the assessment of biomarkers can strateg the risk of HF and inform about the appointment of prophylactic therapy of the HF.

Russian authors in 2020 g posed data on galectine -3 as a heart fibrosis marker in patients with DM2. Patients with DM 2 type and was a link between the galectine -3 and MMLL / surface surface area (R = -0.58, p = 0.001), PiiInp, Timp-1 and the finite-diastolic volume of LV (R = -0 , 68 and p = 0.042 and r = 0.38 and p = 0.02, respectively). The authors concluded that the dynamics at various stages of the continuum of cardiovascular diseases of serum markers of fibrosis may reflect the increase in fibrous and reduction of antifibrotic processes on the preclinical stage of HF. . [47].

According to numerous literature reviews, it has been shown that both elevated and reduced levels of expression of Galektin-3 are observed in different types of diseases, including heart disease, kidney and liver, cancer and infection. Medvedev et al., 2015, Yu and others, 2015, Anand et al., 2013, Van der Velda et al., 2013, Mayolino et al., 2015, Tunon and others ., 2014 g, etc. showed that Galektin -3 has prognostic value in mortality from heart failure. Halektin-3 as a biomarker fibrosis and inflammation participates in the development and progression of HF and can predict increased morbidity and mortality. Two recent meta-analysis showed that elevated levels of the expression of galektin-3 are associated with mortality in acute and chronic heartfelt [9, 10].

Since there is a significant amount of data on sodium-uretical peptide biomarkers (NP), they are already included in the consensus of American experts on HF [12]. In European Recommendations, NP are important elements for the diagnosis of HFsEF and acute HF [11].

Since there is a significant amount of data on sodium-uretical peptide biomarkers (NP), they are already included in the consensus of American experts on CH [12]. In European Recommendations, NP are important elements for the diagnosis of SDSFV and acute CH [11].

The number of new biomarkers is growing. Biomarkers of myocardium damage and fibrosis, such as soluble oncogenic-2-2 (ST2) suppression, galectin-3 and highly sensitive cardiac troponin, can be used for additive risk stratification in patients with HF [12, 13]. The soluble ST2, cardiac stress marker and fibrosis, showed good results to stratify the additive risk of death during acute HF in meta-analysis based on individual patients [14]. In addition, a smaller decrease in soluble ST2 48 hours after treatment was an independent predictor of 1-year mortality in patients with acute SN, who applied to the emergency room [15] .. The value of soluble ST2 as a prognostic indicator was also demonstrated in ambulatory patients with HF [16]. It is noteworthy that soluble ST2 can be useful in patients with renal failure, since it does not affect the renal function [17, 18]. Galektin-3 is a marker of inflammation and fibrosis. It can also provide prognostic information in patients with acute or chronic HF and identify patients who can use drug therapy [19]. In addition, Galektin-3 can also be a potential target for the treatment of fibrosis. Several inhibitors of Galektin-3 are developed in experimental studies. Along with this, other new biomarkers are investigated, such as prokalcitonin, the average-

regional NP and a growth differentiation factor-15. Separate new biomarkers can be useless as prognostic indicators, but combinations of these biomarkers can show better efficacy in predicting mortality.

Researchers from Taiwan (a multicenter study) recommend measuring the B-type sodium peptide (BNP) or N-terminal sodium peptide Prob-type (NT-PROBNP) to confirm or exclude the diagnosis of HF. [20].

Sodium-repeat peptides (NP) are biomarkers associated with stretched myocardial myocytes [21]., Which can counteract stress, induced vasodilation, sodium and diuresis and inhibition of the growth of heart and vascular myocytes. The data of some large cohort studies support the use of NP, especially BNP and NT-PROBNP [22]., For forecasting and diagnostics for the first time identified by the HF. Other studies also confirm the possibility of predicting the HF forecast, including hospitalization and overall mortality. Recent studies conducted in Taiwan in patients with acute HF decompensation have shown that a higher BNP has been associated with a worst functional class and a two-time increase in the risk of nosocomial mortality [23]. It was also shown that in patients with HFsEF, elevated NP levels are a marker associated with an adverse forecast, including mortality and hospitalization due to HF. [24].

The authors also propose to use NP as a prognostic predictor to monitor the effectiveness of HF's therapy before discharge from the hospital. Nevertheless, the efficiency and benefit from serial control measurements or directivity to a specific decrease in NP as a treatment of treatment is still unclear. Although some small studies have shown improved clinical outcomes, [25, 26]. Further research is needed to clarify benefits.

In addition to HF, ischemic heart disease, uncontrolled arterial hypertension, elderly age, renal dysfunction, anemia, pulmonary diseases and sepsis can also increase the level of NP. The gray bnp zone as a diagnostic tool for HF is 100-400 pg / ml and 300-450 pg / ml for NT-PROBNP. In elderly patients (75 years old), the gray zone of NT-PROBNP can be expanded to 300-1800 pg / ml. [27]. Since the level can also be enhanced with different states other than HF, NP preferably as an exception tool when screening the first line. Normal concentration in the untreated patient has a high negative prognostic value for the diagnosis of HF. Moreover, the level of the LC may be lower in patients with a higher body mass index (BMI) (> 35 kg / m 2) due to an increase in receptor clearance in adipocytes, thus, the use of NP in these groups of patients should be used with caution. For patients receiving ARNI treatment, NP-PROBNP is preferable to estimate the patient's forecast, since the ARNI action mechanism increases the BNP level. [28].

Cardiac troponins should also be explored in patients with suspicion of HF or for the first time diagnosed HF. Cardiac troponins are the heart damage marker. Several factors are associated with an elevated level of troponins, including subendocardial ischemia, cardiomyocyte necrosis, inflammatory cytokines, oxidative stress, apoptosis and leakage of troponins from the cytosolic pool due to increased permeability of membranes. [29]. In patients with recently diagnosed HF, it is possible to measure triponins to evaluate possible etiology, as well as for predicting the forecast. Patients with sn, caused by acute coronary syndrome, should be considered the possibility of revascularization. However, cardiac troponins can also be enhanced in patients with myocarditis and severe HF. The increased level of heart troponin in patients with HFrEF is largely associated with mortality and cardiovascular (CVC) complications. [30-32]. However, data on the prognostic value in patients with HFsEF [33, 34].
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In addition to HF and cardiac troponins, other markers are connected with SN. It has been shown that the cardiomyocyte remodeling markers, such as ST-2 and galectin-3, are predictors and HF markers. Increased soluble ST-2 suggests reduction of cardiac protection during heart injuries. Studies also showed that ST-2 can be an independent marker to predict hospitalization on HF and mortality. [35]. It was shown that patients with acute myocardial infarction (OIM) high level of ST-2 in serum is also a predictor of HF. [36]. Macrophages secrete galektin-3, and this is due to cardiac fibrosis. Accordingly, studies have shown that the elevated level of galektin-3 may be the prognostic indicator of the HF. The ratio of albumin to creatinine in the urine (AU / CR) and lipocalin associated with neutrophil gelatinase (NGAL) were used as a kidney damage markers. Recent studies have also shown that AU / CR and NGAL can be markers to assess the HF forecast.

These kidney biomarkers can reflect nephrotoxic damage and systemic endothelial dysfunction. Although the direct mechanism with HF is unclear, these markers can be an early indicator of kidney damage under HF. [37]. In addition, the occurrence of pneumonia in patients with acute HF is a frequently discussed issue, and it is necessary to begin the corresponding antibacterial therapy.

Prokalcitonin is a valuable diagnostic marker of infection during the exacerbation of HF, the authors note. The combination of several biomarkers can have potential benefits for diagnosis and prognostic forecast in patients with HF. However, further validation is required for clinical cohort.

Examine study (study of cardiovascular outcomes using aloglyptin in comparison with the standard of treatment) was a multicenter, no less effective, double disguised, placebo-controlled study, in which 5380 patients with DM 2 diabetes after a recent acute coronary syndrome were randomized to obtain Aloglyptin or placebo. . The initial biomarkers were measured in 5154 patients: NT-PROBNP (N-terminal sodium-type pro-B-type peptide), highly sensitive troponin I, adiponectin, growth differentiation factor-15 and galektin-3. Improving the risk stratification of HF outcomes in patients with type 2 diabetes is crucial, given the appearance of treatment methods that can reduce the risk of the occurrence and recurrence of HF. Although multi-chic acid approaches to the risk stratification of the SN outcomes were demonstrated in populations with HF, data on patients with diabetes mellitus 2 are few. The authors rated the role of combined clinical variables and biomarkers to improve the stratification of the risk of HF outcome in patients with type 2 diabetes after acute coronary syndrome (ACS) in Examine study. These results showed that biomarkers, especially NT-PROBNP, were one of the strongest parameters associated with the future risk of extended exodes of HF [38]. At the same time, in patients with chronic HF of such studies at DM 2 we did not find.

So, as the analysis showed the literature recently, a soluble source of carcinogenicity 2 (SST2), a growth differentiation factor 15 (GDF-15), Galektin-3 and other heart markers, but data from large cohort are still missing.

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