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NEW RECOMMENDATIONS IN THE TREATMENT OF HEART FAILURE

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Relevance. According to the World Health Organization, cardiovascular diseases (CVDs) are the leading cause of death worldwide, killing an estimated 17.9 million people each year. Cardiovascular diseases are a group of diseases of the heart and blood vessels, which include coronary heart disease, cerebrovascular disease, rheumatic heart disease and other pathologies. More than four out of five deaths from CVDs occur because of heart attack and stroke, and a third of these deaths are premature and occur in people under 70 years of age. [1]

For the first time, chronic heart failure (CHF) declared itself as a serious public problem in 1960, when US hospital statistics registered a kind of record: the number of patients with CHF exceeded 1% of all hospitalized patients, and the frequency of the first diagnosis of CHF was two per 1000 of all requests per year [2].

A number of specialists studying heart failure tend to use the term "Global Pandemic" since this problem tends to progress. Most heart failure sufferers are multimorbid. Heart failure can develop because of any disease that leads to impaired heart function. In addition, establishing its specific cause is a priority task. [3]

The mechanisms of the onset and progression of CHF cause AMI are caused by changes in geometry and the presence of myocardial contractility, called "left ventricular remodeling"; with ICMP, there is a decrease in total myocardial contractility, called "myocardial hibernation". In hypertension, regardless of the etiology of hypertension, the structural restructuring of the organ has a specific name - "hypertensive heart". The mechanism of CHF in this case could potentially be LV diastolic dysfunction.

Other etiological factors for the development of CHF are: infective endocarditis; toxic cardiomyopathy (exposure to alcohol, drugs, radiation therapy); SZST (SKV, SSD, DM); systemic vasculitis; cardiomyopathy of pregnancy; endocrine and metabolic disorders (hyperthyroidism, hypothyroidism, acromegaly, uremia, diabetes, obesity); dilated cardiomyopathy - CHF in patients without risk factors or manifestations of IBS (idiopathic, familial); hypertrophic cardiomyopathy (LV hypertrophy according to ECG and EchoCG; identified obstruction of the LV outflow tract). Diabetes mellitus remains a widespread pathology and is present in 40% of patients with preserved and 45% of patients with low LVEF. In 2011, at the 71st Congress of the American Diabetes Association, it was stated that one in three people born after 2000 will develop diabetes during their lifetime. According to WHO estimates, there are about 422 million people with diabetes in the world, while the number of such people increased from 108 million in 1980 to 422 million in 2014 and continues to increase. Numerous data prove that the concomitant course of diabetes and

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CHF largely determines the unfavorable prognosis, creating difficulties for both cardiologists and endocrinologists. At the same time, a clear relationship has been observed between type 2 diabetes and unfavorable outcome of HF [4]. In a recent HF incident study conducted between 1998 and 2017 in the United Kingdom (UK), the age-adjusted rate of first hospitalization increased by 28% for both all-cause and HF hospitalizations, and by 42 % for hospitalizations without cardiovascular disease. This increase was higher in women, possibly due to a higher incidence of comorbidity. The risk of hospitalization for HF is 1.5 times higher in patients with diabetes compared to controls. A meta-analysis of 21 studies performed before 2014, covering 1.1 million patients, showed that the combination of type 2 diabetes with coronary heart disease, arterial hypertension and peripheral vascular diseases is a reliable risk factor for HF [5]. Also, HF can be complicated by diabetes when, as a result of organ hypoperfusion and hyperactivation of neurohumoral systems, pathogenetic changes develop that contribute to an increase in the concentration of glucose in the blood due to a decrease in its consumption by muscle tissue, increased gluconeogenesis in the liver and catecholanaemia. Due to the common combination of diabetes, arterial hypertension and coronary heart disease, the question remains controversial: in which cases is left ventricular diastolic dysfunction directly caused by a glycometabolic disorder, and in which cases is it due to the combined effect of these diseases? A recent meta-analysis of 47 cohort studies involving 12 million people found that the relative risk of developing HF associated with type 2 diabetes was 1.95% in women and 1.74% in men. In women, rapid development of ventricular remodeling, oriented toward concentric hypertrophy, was observed. They had lower quality of life and poorer exercise capacity outcomes than men with type 2 diabetes, even at normal body weight and recommended glycemic levels [6–8]. Based on the above, type 2 diabetes and CHF are acquiring the status of an epidemic of the 21st century and require healthcare costs for the prevention and treatment of these diseases. [9]. The basis for the development of drugs that have an insulin-independent effect and promote the excretion of glucose in the urine was the isolation of the flavonoid phlorizin from apple bark back in 1835, which had the effects of quinine. In the 1980s, it was shown that increased levels of glycosuria are associated with inhibition of sodium-dependent glucose transporters types 1 and 2. Phlorizin proved to be a non-selective blocker of these transporters, but due to its high toxicity it was not suitable for use in humans. In an experiment on rats, it caused glycosuria with a decrease in glycemia and insulin resistance. It was found that inhibition of sodium-dependent glucose transporter type 2 increased the concentration of circulating ketone bodies, which could provide an alternative energy source for cardiomyocytes in conditions of insulin resistance. In addition, other potential mechanisms of action of such drugs are possible: reduction in body weight, blood pressure, sodium levels, oxidative stress, and sympathetic activity. Empagliflozin is considered one of the main drugs in the group of sodium-dependent glucose transporter type 2 inhibitors. Empagliflozin is a representative of a new promising class of drugs that lower blood sugar levels, independent of insulin. In addition to the proven hypoglycemic effect, evidence has accumulated regarding the cardioprotective potential of empagliflozin. Empagliflozin is a once-daily oral, highly selective sodium-glucose cotransporter 2 (SGLT2) inhibitor for the treatment of patients with type 2 diabetes mellitus, included in recommendations to reduce the risk of cardiovascular death in many countries. Registration of empagliflozin for the treatment of chronic heart failure with reduced ejection fraction at the state level will be a breakthrough in healthcare: now cardiologists will be able to independently prescribe it to patients with heart failure with reduced ejection fraction, and the manufacturer will do everything possible to help doctors achieve their professional goal of saving patients. In September 2021, the

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Russian Ministry of Health registered a new indication for the use of empagliflozin for the treatment of heart failure with reduced ejection fraction. For more than five years, the drug has been used in patients with type 2 diabetes to reduce the risk of cardiovascular death. It is now indicated to reduce the risk of cardiovascular death, hospitalization for heart failure, and to slow the decline of kidney function in patients with heart failure with reduced ejection fraction, regardless of the presence of T2DM.[10] Results from the EMPEROR-Reduced® trial showed a 25% relative risk reduction for the composite endpoint of cardiovascular death or heart failure hospitalization compared with placebo in adult patients with heart failure with reduced ejection fraction (HFrEF)2 when adding empagliflozin. (10 mg) to the standard of therapy. The success of empagliflozin is an excellent example of how, through new research, an active substance originally developed for one specific purpose (lowering glucose levels) has come to solve other therapeutic problems aimed at prolonging the lives of patients.

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