Procedia of Theoretical and Applied Sciences
Volume 7 | May 2023
ISSN: 2795-5621 Available: http://procedia.online/index.php/applied/index

MODERN ASPECTS OF THE TREATMENT OF CHRONIC HEART FAILURE

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Abstract: The article is devoted to new trends in the treatment of chronic heart failure (CHF). In the etiological structure of CHF, unlike in previous years, arterial hypertension occupied a significant place. Among the methods for diagnosing CHF, the determination of the levels of natriuretic peptides and data from an ultrasound examination of the heart play a leading role. Basic therapy for CHF is based on the use of three groups of drugs: angiotensin-converting enzyme inhibitors, β-blockers, and mineralocorticoid receptor antagonists. New prospects in the treatment of CHF associated with the use of ivabradine and the blocker neprilysin together with sartan are considered.

Key words: eplerenone, ivabradine, natriuretic peptides, chronic heart failure.

Introduction: Chronic heart failure (CHF) is a syndrome characterized by certain symptoms (shortness of breath, edema, fatigue) and clinical signs (swelling of the neck veins, fine bubbling rales in the lungs, etc.) resulting from violations of the structure and function of the heart [1].

CHANGES IN THE ETIOLOGICAL STRUCTURE OF CHRONIC HEART FAILURE

Until recently, the leading role among the causes of CHF was assigned to coronary heart disease (CHD), then in descending order followed by heart defects, myocarditis, pericarditis, and cardiomyopathy. These diseases in most cases are accompanied by a decrease in systolic function of the heart. The situation changed significantly, when, because of the studies, it became clear that more than half of the cases of CHF are associated mainly with a violation of the diastolic function of the heart while maintaining its contractility. Moreover, it turned out that diastolic dysfunction of the heart, as a rule, precedes a decrease in the ability of the myocardium to fully contract, i.e. occurs in the early stages of CHF [2]. A classic example of diastolic dysfunction of the heart is arterial hypertension (AH). Thus, at present, hypertension (AH) without the presence of coronary artery disease in 40-50% of cases is the cause of CHF.

NEW APPROACHES TO THE TREATMENT OF CHRONIC HEART FAILURE

As with the treatment of any disease, therapeutic interventions for CHF should begin with non-pharmacological measures. Limiting salt intake is essential. Patients should know that the greatest amount of salt is found in products from the store: in bread, hard cheeses, sausages, canned food, etc. Therefore, homemade, easily digestible low-salt food should be preferred. At the same time, the amount of liquid drunk per day is not so strictly limited, although its volume should not exceed 1.5–2 liters. A routine procedure for assessing the degree of fluid retention in the body and the effectiveness of diuretic therapy should be the weighing of patients with CHF, which must be carried out both in hospital and at

Procedia of Theoretical and Applied Sciences
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ISSN: 2795-5621 Available: http://procedia.online/index.php/applied/index

home at least 3 times / week. An increase in weight of more than 2 kg in 1–3 days indicates fluid retention in the body and the need to correct treatment with diuretics. Measuring the amount of fluid drunk per day and the amount of urine excreted is an inaccurate and difficult procedure for most patients with CHF, because of which it is no longer recommended for this disease. With all FC CHF, a feasible dynamic load is shown: with FC I–II - brisk walking, an exercise bike, swimming, with FC III–IV - exercises for small and large muscle groups, walking, breathing training (inflation of the ball). Static loads are strictly prohibited, especially at high FC CHF.

Significant changes have also been made in the medical management of patients with CHF. Currently, the basic therapy for CHF consists of only three groups of drugs (class of recommendations IA): angiotensin-converting enzyme inhibitors (ACE inhibitors), β-blockers (BAB) and mineralocorticoid receptor antagonists (MCR). Failure to prescribe medication to each of these three groups cannot be justified, and the reason for not taking any medication should be reflected in the patient's medical history or outpatient record. These groups of drugs are prescribed for any form of CHF (systolic, diastolic or mixed dysfunction).

A large number of publications have been devoted to the use of ACE inhibitors in CHF. All ACE inhibitors have CHF in their testimony. The methodology for their application has been developed over the course of three decades and has not undergone significant changes in recent years. It should only be remembered that the minimum systolic pressure at which an ACE inhibitor is possible is 85 mm Hg. Art. In these cases, treatment should begin with ¼ of the therapeutic dose with an increase of no more than 2 times and no more than one time / week under the control of blood pressure. Plasma potassium levels >5.5–6.0 mmol/l and/or creatinine levels >265 μmol/l are grounds for not using ACE inhibitors.

If it is not possible to use ACE inhibitors in CHF, an alternative is the use of angiotensin receptor blockers (ARBs) - evidence class IIA. However, of the 8 existing ARBs, only three can be prescribed: losartan, valsartan, and candesartan. The method of their use and contraindications to the appointment are similar to those for ACE inhibitors. In 2014, the results of the PARADIGM-HF study were published, which for the first time showed that a combination drug containing the neprilysin inhibitor sacubitril and valsartan was more effective than enalapril in the treatment of congestive CHF with a decrease in EF <35% [4].

Of the BBs for CHF, only 4 drugs can be prescribed: carvedilol, metoprolol succinate (but not tartrate), bisoprolol and nebivolol. When using BAB, the following rules should be strictly observed:

1. BAB can be prescribed for CHF only after the convergence of edema, the disappearance of attacks of cardiac asthma.
2. Before prescribing BAB, adequate doses of ACE inhibitors and diuretics should be selected.
3. The use of BAB should be started carefully, with 1/8 of the therapeutic dose.
4. Doses of BAB should be titrated very slowly, doubling them once every 2 weeks or even a month.
5. Doses of BAB should be titrated to reduce the number of heartbeats (HR) ≤ 70 bpm.

Unfortunately, the optimal use of β-blockers in CHF can be carried out only in 35–40% of cases [5]. Hypotension, the development of atrioventricular blockades, bronchial obstruction, and even worsening of symptoms of CHF due to the fact that during the first 4-6 weeks of using BAB, EF may decrease.

New prospects in this direction are associated with the introduction of sinus node blocker ivabradine into the practice of treating patients with CHF (level of evidence IIA). Ivabradine can be prescribed in the presence of sinus rhythm and heart rate > 70 bpm. A distinctive feature of this drug is a decrease in heart rate without affecting the level of blood pressure, rhythm and conduction disturbances. Ivabradine

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ISSN: 2795-5621 Available: http://procedia.online/index.php/applied/index

reduces the risk of death or hospitalization of patients by 29% in the absence of serious adverse reactions [6]. Ivabradine, unlike BAB, increases the stroke volume of the heart and EF from the very beginning of treatment - the effect on systolic dysfunction, and also improves LV filling by prolonging the diastole - the effect on diastolic dysfunction [7]. Ivabradine, the only one of all drugs to reduce heart rate, improves collateral blood flow in IHD, which makes its use very promising in patients with ischemic myocardial dysfunction [8, 9]. This drug reduces end-systolic and end-diastolic LV volumes. Ivabradine, unlike BAB, improves myocardial contractility not only at rest, but also during exercise [10]. With equal control of heart rate, ivabradine has a more effective effect on coronary blood flow and stroke volume than metoprolol. It also enhances the important effects of BBs without exacerbating the negative ones. The combination of ivabradine and beta-blockers more effectively improves CHF FC and exercise tolerance than beta-blockers alone. Reduction of heart rate in CHF to target values returns normal contractility to the myocardium. For every 5 beats of heart rate reduction, there is an 18% reduction in the risk of death [1].

In patients with CHF, the concentration of aldosterone in the blood can be many times higher than that in healthy individuals. The production of aldosterone limits the effect of long-term use of blockers of the renin-angiotensin-aldosterone system. The AMCR class includes 2 drugs: spironolactone and eplerenone. The first of them is non-selective AMPR, and the second is selective. Spirolactone blocks receptors not only for mineralcorticoid, but for sex hormones, which causes the appearance of such undesirable phenomena as gynecomastia, decreased potency in men, hirsutism and menstrual irregularities in women in 10–15% of cases. As a result, eplerenone should now be preferred in all cases. AMPKs are also called potassium-sparing diuretics, although their diuretic effect is extremely weak [11]. The results of studying the effect of AMKR on the survival of patients with CHF showed a significant reduction in the risk of death in the group of patients taking these drugs at an average dose of 26 mg for 2 months [12]. In the large EPHESUS study, patients who had a myocardial infarction with an EF <40% received eplerenone at a mean dose of 43 mg for 16 months. At the same time, cardiovascular mortality and the need for hospitalization decreased by 21%, and the frequency of sudden death by 33% [13]. In 2011, the results of the EMPHASIS-HF study [14] were published, which demonstrated a significant reduction in the frequency of hospitalizations due to worsening CHF with the use of MCR. Subsequently, evidence was obtained that the use of AMCR prevents the formation of fibrosis in the myocardium and thus prevents the increase in its stiffness, which is especially important in diastolic myocardial dysfunction in patients with CHF with preserved EF [14]. In addition, in the EPHESUS-HF study, new cases of atrial fibrillation in patients receiving eplerenone were significantly less common (2.7 vs. 4.5% in the placebo group).

In recent years, the methodology for prescribing diuretics in CHF has also undergone a significant change. If earlier they were prescribed only for cardiac asthma, liver enlargement, the presence of edema, ascites, hydrothorax, then at present their use is recognized as appropriate even in the absence of obvious congestion, i.e. with II FC CHF. In practice, this means that the appearance of shortness of breath in a patient in combination with radiographic signs of stagnation in the pulmonary circulation is the basis for prescribing small doses of diuretics. Another difference in the modern dehydration therapy of patients with CHF is the predominant use of the loop diuretic torasemide instead of the previously widely used furosemide. Its dose should be such that during the period of CHF decompensation and convergence of edema, the patient loses 0.8-1 kg / day in weight, and when the disease is compensated, the patient's weight should remain stable with daily diuretic intake. Daily doses of torasemide 10–20 mg are small, 20–40 mg are medium, ≥40 mg are large. If necessary, the dose of the drug can be increased to 100-200

Procedia of Theoretical and Applied Sciences
Volume 7 | May 2023
ISSN: 2795-5621 Available: http://procedia.online/index.php/applied/index

mg / day in 1-2 doses.

Furosemide retained its value in urgent situations (pulmonary edema) and in terminal renal failure, accompanied by hyperkalemia. With III-IV FC CHF, it is necessary to prescribe a carbonic anhydrase inhibitor acetazolamide (Diacarb) at 0.25 2-3 times / day for 3-4 days 1 time in 2 weeks to correct the acid-base balance and enhance the diuretic effect.

The use of digoxin in CHF is currently limited by the presence of tachysystolic atrial fibrillation or a decrease in EF <40% while maintaining sinus rhythm. Digoxin is prescribed when CHF compensation is achieved for a long period. Its standard dose for glomerular filtration rate >60 ml/min is 1 tablet (25 mcg) per day, and for GFR <60 125="" p="">

Thus, according to the words of Petr Panikowski, chairman of the working group on the development of European guidelines governing the diagnosis, treatment and prevention of CHF, “heart failure is becoming a disease that can be prevented and treated” [3].

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Procedia of Theoretical and Applied Sciences
Volume 7 | May 2023
ISSN: 2795-5621 Available: http://procedia.online/index.php/applied/index

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