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Heart Failure in the Background of Ischemic Heart Disease: Some Issues of Epidemiology, Pathogenesis and Treatment

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Annotation: One of the main postulates of medicine is that optimal treatment of a disease is impossible without knowing its cause. However, this principle is difficult to apply when it comes to chronic heart failure (CHF). This is due to the fact that, although the diseases that lead to the development of CHF are numerous and varied, the final outcome for all is the same - decompensation of cardiac activity. There is an illusion that the cause of CHF is no longer important and the treatment is the same in all cases: be it ischemic heart disease (IHD), dilated cardiomyopathy (DCM) or hypertensive heart. However, this is not true. Any disease underlying CHF has its own characteristics that leave an imprint on the course of heart failure, its therapy and prognosis. This is especially true for such a common and serious disease as ischemic heart disease.

Keywords: ischemic etiology, dilated cardiomyopathy, ischemic heart disease.

Introduction

It is currently considered proven that coronary heart disease is the main etiologic cause of heart failure. This conclusion can be made based on the analysis of the results of special epidemiological studies in populations, as well as on the assessment of the contingent of patients included in multicenter studies on the survival of patients with CHF. Thus, in 10 such studies conducted in recent years; ischemic etiology of decompensation was noted in an average of 64% of patients. The spread of the results of these studies is explained by the difference in the diagnostic criteria of coronary heart disease used in different centers, population differences, and may also be associated with the drug under study. Thus, in studies related to b-blockers (in the table, these are the CIBISI and USCT studies), a significant place among the causes of CHF along with coronary heart disease is occupied by DCM (36 and 52%, respectively); where ACE inhibitors are studied, the main cause of decompensation is, as a rule, coronary heart disease, and the incidence of DCM does not exceed 22%.

Epidemiological studies in populations also indicate a significant prevalence of coronary heart disease among the main causes of CHF development. In a study of the prevalence of CHF in Glasgow, coronary heart disease as a cause of CHF was observed in 95% of patients (!). The results of these same studies show that arterial hypertension also occupies a significant place in the structure of causes of CHF development, although in clinical practice hypertension is most often combined with coronary heart disease. But the role of DCM as a cause of CHF in such epidemiological studies is small and is estimated at 0-11%.

A retrospective study conducted at the A.L. Myasnikov Research Institute of Cardiology also revealed that coronary heart disease has become the main cause of heart failure in recent years [2], and the “contribution” of coronary heart disease to the overall incidence of CHF is constantly increasing.

Pathophysiology of heart failure in coronary artery disease mechanisms of development and progression of heart failure in IHD. The most important of them is, of course, *myocardial infarction*

(MI). Sudden “loss” of a more or less extensive area of the heart muscle leads to the development of ventricular dysfunction (s), and if the patient does not die (which happens in 50% of cases), the dysfunction, as a rule, manifests itself over time with symptoms of heart failure. In the *TRACE study* [3], 40% of patients who had an MI had severe LV dysfunction in the first days, and in 65% of them it was their first infarction in their life; 74% of them soon developed a clinical picture of heart failure. However, even those 60% of patients who manage to avoid the development of dysfunction at an early stage of the disease cannot consider themselves “safe”. An *extensive infarction is followed by changes in the heart muscle, called “remodeling” of the heart.* This phenomenon includes processes affecting the affected area and healthy areas of the myocardium, when the infarction zone "stretches", unable to withstand the increased intraventricular pressure, and the unaffected areas hypertrophy and dilate, adapting to new operating conditions. *Remodeling is a process that includes a change in the shape and function of the ventricles over time, expansion of scar tissue,* which is closely associated with a change in the neurohumoral background of the body.

However, the peculiarities of the development of CHF in patients with coronary heart disease are caused not only by ventricular dysfunction, but also by the constant participation of coronary insufficiency in this process.

Frequent episodes of local ischemia led to *periods of increased local systolic dysfunction,* manifested by dyspnea (angina equivalent). For example, in the *SOLVD study,* 37% of patients with CHF also complained of angina pain [4]. Frequent episodes of the appearance and disappearance of ischemia contribute to the prolongation of systolic dysfunction in these areas, which is equivalent to the state of “stunned” myocardium described in the occlusion and subsequent “opening” of the corresponding coronary artery. Persistent coronary insufficiency makes an important contribution to the development of both systolic and diastolic dysfunction of the LV.

Another mechanism of systolic dysfunction in patients with coronary heart disease is associated with such a phenomenon as *myocardial “hibernation”*, which is an adaptive reaction under conditions of constantly reduced coronary blood flow. Under these conditions, tissue perfusion is sufficient to maintain the existence of cardiomyocytes (including ion currents), but is insufficient for their normal contractility. This process leads to *gradual hypocontractility of the entire myocardium and progression of LV dysfunction.* Recent data indicate that myocardial hibernation inevitably ends in necrosis if coronary blood flow does not increase.

Thus, in addition to such irreversible changes as post-infarction scar, persistent myocardial ischemia, stunned and hibernated myocardium are also added - all together they contribute their own specificity to the development of CHF in patients with coronary heart disease.

Another important factor in the development of myocardial dysfunction in patients with coronary heart disease is the dysfunction of the coronary endothelium, which is characteristic of this pathology. Endothelial dysfunction is usually defined as a disorder in the ability of these cells to produce specific (vascular) relaxation factors (NO, prostacyclin, hyperpolarization factor). It has been proven that endothelial dysfunction *activates the activity of neurohormones* responsible for the development and progression of CHF: endothelin-1, renin- angiotensin - aldosterone and sympathoadrenal systems, tumor necrosis factor. In addition, endothelial dysfunction in patients with coronary heart disease blocks the migration of smooth muscle cells and their proliferation in the vessel wall, increases the permeability of the wall for lipids, which contributes to the further development of atherosclerosis and coronary thrombosis, which, in turn, causes persistent myocardial ischemia and LV dysfunction.

RESULT AND METHODOLOGY

Efficiency of drug therapy for CHF in coronary heart disease

Digoxin

The DIG study [5] showed that in patients with CHF, digoxin increases the incidence of MI by 26%, which may indirectly indicate a potentially adverse effect of glycosides on the course of coronary heart

disease. This may be due to an increase in $O_{2\ consumption}$ *against the background of increased myocardial contractility. Despite this, such an important indicator as the risk of death and/or forced hospitalization due to the progression of CHF when using digoxin still tended to decrease, although the degree of this decrease in patients with CHD was less significant (by 21%) than in patients with CHF of non-ischemic etiology (a decrease of 33%).*

ACE inhibitors

Almost all clinical studies indicate a pronounced positive effect of ACE inhibitors not only on mortality rates, but also on the development of coronary heart disease, including in patients with CHF or systolic myocardial dysfunction. Thus, according to the data of the main multicenter studies, the reduction in the risk of developing MI when using ACE inhibitors in such patients reaches 12-25% (Fig. 1).

The success of using ACE inhibitors in patients with CHF of ischemic genesis may be associated with the unique property of this class of drugs to improve not only the hemodynamic and neurohumoral status, but also to normalize the endothelial function of the coronary arteries, the role of which in the pathogenesis of heart failure is currently no longer in doubt. In addition, ACE inhibitors contribute to the improvement of plasma fibrinolytic activity, which has a preventive effect on the development of coronary thrombosis.

Clinical confirmation of the actual "anti-ischemic" properties of ACE inhibitors can be found in the results of the *QUIET study,* in which the addition of ACE inhibitors *quinapril* in patients with coronary heart disease with preserved ventricular function reduced the incidence of coronary complications by 10% [6]. In the case of a combination of coronary heart disease and CHF, the average reduction in the risk of death associated with the use of drugs of this class reaches 23%, as evidenced by the results of a meta-analysis *Garg&Yusuf*, conducted on the basis of the results of 32 placebo-controlled studies [7]. Another important conclusion of this meta-analysis indicates that in the subgroup of patients with heart failure of non-ischemic etiology (not coronary heart disease), the effect of ACE was also positive, but still somewhat less than in patients with myocardial ischemia.

Despite such favorable conditions for patients with coronary heart disease, in the 2 largest studies with enalapril (V- HeFT II and SOLVD prevention & treatment), the effectiveness of treating heart failure of ischemic etiology was significantly lower than in decompensation of non-ischemic genesis.

The fact that the effect of ACE inhibitors depends on the etiology of decompensation and may be less effective in CHF of ischemic genesis was confirmed in a retrospective study conducted at the A.L. Myasnikov Research Institute of Cardiology. *Six-year use of ACE inhibitors* in patients with CHF of ischemic etiology *reduced the risk* of their *death by an average of 26%,* and in similar *patients with DCM - by 60%* [2].

b-Blockers

Myocardial ischemia is one of the main indications for the use of b-blockers, but the addition of IHD of circulatory failure until recently was considered dangerous for the use of drugs with negative inotropic properties and even served as a contraindication for such therapy. Nevertheless, back in the 80s it was known that the effectiveness of b-blockers in patients with MI complicated by CHF is even higher than in patients without heart failure (Fig. 2).

After a long period of search and hesitation, the advisability of using b-blockers in the treatment of patients with CHF ceased to be a subject of discussion and became obvious only in the last 1-2 years, after the completion of three large multicenter studies with carvedilol (*USCT*), bisoprolol (CIBIS *II)* and metoprolol (MERIT-*HF)*.

In all these studies, *the use of b-blockers in addition to the main therapy* (ACE inhibitors + diuretics / glycosides) *reduced the risk of death by an average of 34-65%.* Moreover, the positive effect of therapy was observed in subgroups of patients with both ischemic and non-ischemic genesis of cardiac decompensation.

The anti-ischemic and anti-anginal properties of b-blockers theoretically suggest their advantage when used in patients with CHF of ischemic etiology. However, in real clinical practice this is only partially confirmed. As can be seen from Table 5, with IHD beta-blockers had an insignificant advantage in only 2 of 4 studies. Moreover, in one of the studies (*CIBIS I*), bisoprolol had virtually no effect on mortality in patients with myocardial ischemia, with a pronounced positive effect in patients with DCM.

Amiodarone

The efficacy of amiodarone in patients with coronary artery disease is associated primarily with a reduction in sudden, arrhythmic death.

Thus, in the Canadian (CAMIAT*)* and European (EMIAT*)* studies in patients in the post-infarction period, amiodarone did not affect the overall mortality of patients, but significantly reduced the risk of sudden (arrhythmic) death (Table 6).

According to a meta-analysis *ATMA* , which included 13 major studies in patients with LV dysfunction and/or heart failure, along with a reduction in arrhythmic death (by 29%), the use of amiodarone was still accompanied by a reduction in the risk of overall mortality by an average of 13% [11].

However, the relationship between the etiology of CHF and the effectiveness of this drug is ambiguous. Thus, in the *CHF-STAT study,* a tendency toward a decrease in mortality during amiodarone therapy was noted only in patients with non- ischemic decompensation (20%, p=0.07); at the same time, the effectiveness of therapy in patients with coronary heart disease approached 0. In contrast to this work, in the Argentine *GESICA study,* the reduction in the risk of death and/or hospitalization when using amiodarone in patients with CHF averaged 31%, but patients with coronary heart disease had a relative “success”: their risk reduction was 38% versus 23% in patients with cardiomyopathy.

Calcium antagonists

The effectiveness of calcium antagonists in patients with CHF has not been sufficiently studied. However, even the data from isolated studies provide contradictory results. The PRAISE I study with amlodipine showed that the use of this drug (against the background of ACE inhibitors, diuretics, glycosides) is accompanied by an unreliable decrease in fatal and non-fatal complications, as well as the frequency of fatal outcomes by an average of 9%. Moreover, this “success” was achieved exclusively “thanks” to patients with CHF of non-ischemic etiology (DCM, hypertensive heart, etc.), in whom the reduction in the risk of death associated with amlodipine was 46%; at the same time, in patients with coronary heart disease, this indicator remained virtually unchanged (Table 7).

Directly opposite results were obtained in the *V- HeFT -III study* with the third-generation calcium antagonist felodipine: in patients with CHF, there was an average 18% reduction in mortality; at the same time, in the subgroup of patients without the number of fatal outcomes in the context of this therapy even slightly exceeded the figures in the control group.

Discussion

The immediate and long-term trends in the development of cardiovascular diseases indicate that the growth in the number of patients with coronary heart disease will continue and CHF, as a complication of coronary heart disease, will become one of the main causes of hospitalization and mortality in the next century, especially in the older age group. In this regard, the question arisesare there reliable methods for preventing and treating this syndrome?

Analysis of the main multicenter studies does not provide a clear answer: *the effectiveness of treatment of patients with CHF at IHD is often worse than in patients with decompensation of non-ischemic etiology,* for example, with DCM.

The reasons for this phenomenon may be associated with the “dual” pathogenesis of CHF in occlusive coronary artery disease, which requires not only the impact on cardiac remodeling processes, but also

effective restoration of myocardial perfusion. Hence, it can be assumed that *without adequate revascularization of the heart muscle, it is difficult to achieve success in the prevention and treatment of circulatory failure in patients with coronary atherosclerosis.* Unfortunately, there is no serious confirmation of this concept yet, since patients with symptoms of heart failure are usually excluded from myocardial revascularization studies. Some studies conducted with patients without severe myocardial dysfunction show that with 3-11% surgical mortality, the 5-year survival of operated patients is relatively satisfactory, but no better (and possibly worse) than those who received adequate drug therapy [1]. Of course, these conclusions cannot be final, since the solution to the question of the effectiveness of surgical methods of treatment of such patients requires conducting specially organized studies.

Conclusion

The necessity of "vascular coronary" impact on patients with CHF of ischemic etiology received unexpected confirmation in the study of the effects of hypolipidemic drugs (statins) in patients with dyslipoproteinemia and CHD. Thus, in the *CARE study,* the use of pravastatin reduced the frequency of repeated infarctions and mortality in patients with asymptomatic LV dysfunction, and simvastatin in the 4S study generally prevented the development of heart failure [1]. The results of these studies suggest that if ACE inhibitors can become an important means of treating patients with CHD, then statins are a necessary component of therapy for patients with CHF of ischemic etiology.

Thus, heart failure in patients with coronary heart disease currently remains a serious problem, the solution of which is possible only by combining the efforts of epidemiologists and therapists, cardiologists and cardiac surgeons, aimed at both the prevention and treatment of coronary disease and the elimination of its complications - ventricular dysfunction and heart failure.

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