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Features of the Course of Chronic Heart Failure Resistant to Antiplant Therapy

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Abstract: Heart failure is a major public health problem and, despite optimal medical therapy, morbidity and mortality remain high. Over the past 20 years, in the problem of chronic heart failure (CHF) there have been many changes that can be characterized as evolutionary. This article about main changes regarding etiology, prevalence of resistance to antiplatelet therapy, as well as mortality in patients with CHF.

Keywords: Heart failure, antiplatelet therapy,thrombosis, acetylsalicylic acid, arterial hypertension, atherothrombosis

Heart failure – clinical syndrome, characterized by typical symptoms (shortness of breath, swelling of the ankles, fatigue), which may be accompanied by signs (increased pressure in the jugular veins, wheezing in the lungs, peripheral edema), caused by structural and functional changes in the heart, leading to: decreased heart function and increase intracardiac pressure at rest or during exercise current determination HF limits itself to stages where symptoms of HF are already present, but before symptoms appear, the patient may have structural or functional changes in the heart (systolic or diastolic LV dysfunction) are “precursors” of HF.

Relationship between CHF and age and gender.

Common to all epidemiological studies is the conclusion about a sharp increase in the incidence of heart failure with increasing age of patients, which leads to the “aging” of the population of patients with CHF as a whole. Thus, if the average number of first-time hospitalizations for CHF from 1968 to 1989 increased from 2.0 to 2.5-2.7 per 1000, then the same figure in the older age group (over 65 years old) increased from 7. 5 to 16.3 per 1000 [3]. The 1990 London Epidemiological Study [2] showed that the number of hospitalized patients with CHF under 65 years of age was 0.06%, while the proportion of patients over 65 years of age was 2.8%. The most obvious connection between the prevalence of CHF and the age of patients was revealed in the Framingham study in 1993: the incidence of CHF in the group of men 50 - 59 years old was 9 times less than in patients 80 - 89 years old[2].

Etiology.

The most common causative or comorbid diseases that contribute to the development of CHF are: arterial hypertension (65%), coronary heart disease (CHD) (50%), chronic kidney disease (43%), atrial fibrillation (41%), post-infarction cardiosclerosis - PICS (27%) and diabetes mellitus - diabetes (27%), obesity (23%), malignant neoplasms (23%), chronic obstructive pulmonary disease (23%), anemia (12%), stroke (12%)[1].

Stages and symptoms of CHF

In medicine, two classifications of chronic heart failure are used: classification of chronic circulatory failure N.D. Strazhesko, V.Kh. Vasilenko and the functional classification of the New York Heart Association. In diagnosing the disease, the indicators of both systems are taken into account.

Classification by N. D. Strazhesko, V. Kh. Vasilenko (1935)

The patient's condition is assessed by the number and severity of clinical manifestations of the disease.

Stage 1 Initial, latent circulatory failure, manifested only during physical activity (shortness of breath, palpitations, excessive fatigue). With rest, these phenomena disappear. Hemodynamics are not impaired.

Stage 2 Severe, prolonged circulatory failure, hemodynamic disturbances (stagnation in the pulmonary and systemic circulation), dysfunction of organs and metabolism are also expressed at rest. Working capacity is sharply limited.

Period 2a Hemodynamic disturbances are moderate, and dysfunction of any part of the heart is noted (right or left ventricular failure).

Period 2b Severe hemodynamic disturbances, involving the entire cardiovascular system, severe hemodynamic disturbances in the small and large circles.

Stage 3 Ultimate, dystrophic. Severe circulatory failure, persistent changes in metabolism and organ functions, irreversible changes in the structure of organs and tissues, pronounced dystrophic changes. Complete loss of ability to work.

Functional classification of the New York Heart Association

Adopted in 1964 by the New York Heart Association (NYHA). This classification is used to describe the severity of symptoms; on its basis, four functional classes of the disease (FC) are distinguished.

First class FC. There are no restrictions on physical activity. Normal physical activity does not cause excessive shortness of breath, fatigue, or palpitations.

Second class FC. Slight limitation in physical activity. Comfortable state at rest. Normal physical activity causes excessive shortness of breath, fatigue, or palpitations.

Third grade FC. Explicit limitation of physical activity. Comfortable state at rest. Less physical activity than usual causes excessive shortness of breath, fatigue, or palpitations.

Fourth grade FC. Inability to perform any physical activity without discomfort. Symptoms may be present at rest. With any physical activity, discomfort increases.

Diagnosis of CHF

Electrocardiography. The most accessible instrumental method that allows you to objectively assess the condition of the heart. When analyzing the ECG, attention should be paid to the presence of signs of hypertrophy of the left and right parts of the heart, ischemic and cicatricial changes in the myocardium, the occurrence of disturbances in the conduction system of the heart and the presence of arrhythmia.

Echocardiography (EchoCG). A visualization technique that plays a primary role in the diagnosis of CHF due to its ease of implementation, safety and widespread use. EchoCG allows you to solve the main diagnostic problem - to clarify the very fact of dysfunction and its nature, as well as to conduct a dynamic assessment of the state of the heart and hemodynamics.

Stress echocardiography. Stress or pharmacological stress echocardiography is a highly informative technique for clarifying the ischemic or non-ischemic etiology of HF, as well as for assessing the effectiveness of treatment measures (revascularization, medication restoration of contractile reserve). However, despite the high sensitivity and specificity of this technique for identifying viable

myocardium in patients with coronary artery disease and systolic heart failure, it cannot be recommended as a routine diagnostic method.

Stress tests. Carrying out stress tests in patients with CHF is justified not for diagnosis, but for the purpose of assessing the patient's functional status and the effectiveness of treatment, as well as to determine the degree of risk. However, the normal result of the stress test in a patient not receiving specific treatment almost completely excludes the diagnosis of CHF. In patients with CHF, long-term exercise is justified (8-12 minutes until stopping criteria are reached) with a minimal increase in load when moving from one stage to another.

Drug treatment of patients with CHF

There is no doubt and are recommended specifically for the treatment of CHF (level of evidence A):

1. ACE inhibitors, which are indicated for all patients with CHF, regardless of the etiology, stage of the process and type of decompensation.
2. AII receptor antagonists, which are used mainly in cases of intolerance to ACE inhibitors as a first-line agent for blockade of the RAAS in patients with clinically significant decompensation, as well as a plus to ACE inhibitors; in patients with CHF in whom the effectiveness of ACE inhibitors alone is insufficient.
3. β-blockers (β-blockers) are neurohormonal modulators applied "on top" (in addition) to ACE inhibitors.
4. Aldosterone receptor antagonists used together with ACE inhibitors and β-AB in patients with severe CHF (III-IV FC) and patients who have undergone past AMI.
5. Diuretics – indicated for all patients with clinical symptoms of CHF, associated with excess sodium and water retention in the body.
6. Cardiac glycosides - in small doses. With atrial fibrillation they remain a "first-line" remedy, and in case of sinus rhythm and ischemic etiology of CHF, the use requires caution and control.
7. Statins, recommended for use in all patients with ischemic etiologies of CHF; in addition, they have the ability to prevent the development of CHF in patients with various forms of coronary artery disease.
8. Indirect anticoagulants, indicated for use in the majority of patients with CHF occurring against the background of atrial fibrillation, as well as in patients with CHF and sinus rhythm.

Anticoagulants should be prescribed to all patients with CHF with concomitant atrial fibrillation, a history of thromboembolism, or a mobile thrombus in the LV (class of recommendation I, level of evidence A). Oral anticoagulants (warfarin) are mandatory for the treatment of patients with atrial fibrillation and an increased risk of thromboembolism. An increased risk is observed in patients with atrial fibrillation in combination with one of the following factors (evidence level A):

- ✓ old age;
- ✓ history of thromboembolism;
- ✓ information about strokes and transient cerebrovascular accidents

To reduce the risk of hemorrhagic complications, anticoagulants should be used with careful monitoring (once a month) of the international normalized ratio (INR). It is known that the risk thromboembolism and survival of patients with CHF and atrial fibrillation directly depend on the duration and correctness (maintaining the INR within 2.0-3.0) of anticoagulant treatment.

For decompensated CHF in patients on bed rest, it is recommended to prescribe low molecular weight heparin (enoxaparin) at a dose of 40 mg per day for 6-14 days to prevent thrombotic complications. Also, low molecular weight heparin should be used in patients with severe CHF who are on bed rest (level of evidence B).

Resistance to ASA is a fairly common phenomenon and, according to various sources, occurs with a frequency of 5% to 48%. Currently, clinical and biochemical aspirin resistance are distinguished. Clinical resistance refers to the inability of a drug to prevent a thrombotic episode in a particular patient. Biochemical resistance is defined as insufficient suppression of platelet function while taking ASA, as determined by the results of various laboratory tests.

Factors that increase the risk of aspirin intolerance.

- Low adherence to therapy, failure to achieve the optimal dose of the drug, poor absorption from the gastrointestinal tract, competitive interaction with other drugs taken.
- Genetic polymorphism of GP IIb/IIIa and collagen receptors, enzymes COX-1, COX-2, thromboxane synthetase.
- And other factors, such as: smoking, obesity, hypercholesterolemia, hyperglycemia, physical activity, stress and others.

According to the literature, the ineffectiveness of aspirin therapy in patients with coronary heart disease complicated by heart failure is statistically significantly correlated with smoking, arterial hypertension, diabetes mellitus, and BMI more than 25 kg/m2.

Conclusions: Despite the fact that the platelet is one of the main participants in thrombosis, the mechanism of thrombus formation is complex, and assessment of platelet function does not provide a clear prognosis for a particular patient due to the influence of many other factors. So far, in patients with coronary artery disease, the effectiveness of using tests to determine platelet activity to adjust antiplatelet therapy has not been clearly proven. The main difficulties in personalizing antiplatelet therapy remain the choice of method for assessing platelet activity during therapy, the lack of standardization of these methods, and the influence of many factors on resistance to antiplatelet therapy.

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