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CURRENT STATE OF THE ART OF DILATED CARDIOMYOPATHY

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Abstract:

This article based on disease "Cardiomyopathy which is an acute, subacute or chronic lesion of the heart muscle of unknown or unclear etiology, often combined with damage to the endocardium, and sometimes the pericardium." His basic research has made significant contributions to the study of cardiomyopathy. In subsequent years, heated discussions arose about the nature of this disease and its classification, which served as the reason for the creation in 1980 of a committee for the study of ILC. Depending on the leading pathophysiological mechanism or, if possible, etiological (pathogenetic) factor, they are divided into four forms: dilated, hypertrophic, restrictive CMP and arrhythmogenic right ventricular CMP. Along with four nonspecific cardiomyopathies, including dilated, hypertrophic, restrictive, right ventricular arrhythmogenic dysplasia, as before, specific cardiomyopathies were identified and unclassified cardiomyopathies.

Keywords: Cardiomyopathy, congestive cardiomyopathy, morphofunctional types, pharmacotherapy, Society of Cardiology.

In 1956, the term cardiomyopathy appeared for the first time in English literature. W. Brigden in 1957 proposed using the term cardiomyopathy (CM) for heart diseases of a non-coronarogenic nature. In subsequent works by a number of scientists T. Mattingly (1958-1970), W. Harvey (1964), O. Storstein (1964), N. Fowler and M. Gueron (1965), J. Segal (1965), R. Massumi (1968) the doctrine of the nosological independence of CMP was further developed. Over the years, the very definition of the concept of ILC and their classification have changed several times. The source of considerable confusion was the abundance of different terms and the lack of common views on their definition.

In the early 70s of the twentieth century, a more active study of the ILC began. In 1972 J.F. Goodwin defined cardiomyopathies: "Cardiomyopathy is an acute, subacute or chronic lesion of the heart muscle of unknown or unclear etiology, often associated with damage to the endocardium and sometimes the pericardium." His basic research has made significant contributions to the study of cardiomyopathy. In subsequent years, heated discussions arose about the nature of this disease and its classification, which served as the reason for the creation in 1980 of a committee for the study of ILC at the WHO.

In the domestic literature, the first works on cardiomyopathies were published by M.I. Theodori (1962). A significant contribution to the development of the doctrine of ILC was made by the following studies: N.M. Mukharlyamova (1975, 1982), L.F. Nikolaeva (1982), A.A. Kedrova (1980), N.R. Paleeva (1982), G.P. Kuznetsova (1984-2004), V.P. Polyakova (1997-2003), B.C. Moiseeva, A.V. Sumarokova, V.Yu. Styazhkina (1993), E.N. Amosova (1991, 1999) and V.N. Kovalenko, E.G. Nesukai (2001).

The study of ILC in Samara began in 1980. Head of the Department of Faculty Therapy of the Medical Institute, Professor G.P. Kuznetsov, consulting a 20-year-old patient with severe heart failure and systolic murmur at the apex of the heart, diagnosed rheumatism. The patient died; during postmortem examination, the heart valves were intact. Marked dilatation of the heart chambers and thinning of the walls of the left ventricle were noted. The diagnosis of rheumatism was not confirmed, which forced Professor G.P. Kuznetsov to turn to the literature, where he first encountered the concept of congestive cardiomyopathy. Thus, retrospectively, during a pathological examination in Samara, a diagnosis of cardiomyopathy was made for the first time and work began to study this pathology. In 1985, on the basis of the clinic and the department of faculty therapy of the medical institute and professor G.P. Kuznetsov created the Regional Center for the diagnosis and treatment of patients with cardiomyopathies. The center was the first in the Volga region. Patients from Samara and neighboring regions of Ulyanovsk, Penza and Tatarstan were examined and treated there [1, 2, 3].

In 1980, WHO created a special working group of experts on cardiomyopathies and published the first classification of cardiomyopathies. According to WHO experts, "Cardiomyopathies are diseases caused by primary damage to the myocardium, and not by arterial hypertension, congenital, acquired heart defects, coronary heart disease or diseases of peripheral vessels and pericardium." In accordance with WHO recommendations, (1980) (WHO/ISFC Task force report

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on the definition and classification of cardiomyopathies, 1980), the etiology of cardiomyopathies began to be distinguished:

I. Primary CMP - Idiopathic: dilated (D), restrictive (R), hypertrophic (G), familial (D, G), Loeffler's endocarditis (R), endomyocardial fibrosis (R);

II. Secondary: - Infectious - viral myocarditis, bacterial myocarditis, fungal myocarditis, parasitic myocarditis, spirochetosis, rickettsiosis;

III. Hereditary storage diseases (D, R): glycogenosis, mucopolysaccharidosis, hemochromatosis and secondary hemosiderosis;

IV. Eating and electrolyte disorders (D);

V. Rheumatic diseases: systemic lupus erythematosus, periarteritis nodosa, systemic scleroderma, dermatomyositis;

VI. Infiltrative diseases (R, D): amyloidosis, sarcoidosis, malignant neoplasms;

VII. Neuromuscular diseases (D): myopathies, atrophic myotonia, Friedreich's ataxia (D, D);

VIII. Allergy and intoxication (D): alcohol, radiation, drugs;

IX. Postpartum (periportal) cardiomyopathy (D)

In this regard, CMP began to be divided into primary diseases of the heart muscle - of unknown etiology and secondary, the causes of which are known or associated with other diseases. It is the secondary ones that are called specific CMPs. In many cases, the term CMP is used to describe the characteristics of damage to the morphological structures of the myocardium and therefore the terms are used: ischemic CMP, metabolic CMP, alcoholic CMP. The nature of cardiac remodeling in such cases fits into one of three morphofunctional types characteristic of cardiomyopathies: dilated, hypertrophic or restrictive. However, the abuse of the terminology of specific cardiomyopathies can lead to obfuscation (blurring) of a number of nosological forms. As noted by G.P. Kuznetsov (2004) the use of the terms "inflammatory cardiomyopathy", "valvular cardiomyopathy" is unfounded, since it replaces the nosological form with a syndromic concept. As the author emphasizes, "Diagnoses based only on individual ECG or physical symptoms, such as tonsillogenic CMP, anemic CMP, etc., are completely unacceptable. Sometimes the severity of myocardial dysfunction does not correspond to the severity of the etiological factor, which makes one think about the individual reaction of the patient or the presence of additional, undetected effects on the heart."

Domestic school of cardiologists G.F. Langa has long used the term myocardial dystrophy: alcoholic, dishormonal, metabolic electrolyte, etc. This term is most adequate to understand the ILC. The modern definition and classification of CMP were proposed by a working group of WHO experts and the International Society of Cardiology in 1996. In accordance with these ideas, the very concept of cardiomyopathy was significantly expanded to a vague formulation that "cardiomyopathies are myocardial diseases associated with cardiac dysfunction." This interpretation of the definition of cardiomyopathies allows us to speak not only about the nosological, but also the syndromic affiliation of this term [4,5, 6].

Depending on the leading pathophysiological mechanism or, if possible, etiological (pathogenetic) factor, they are divided into four forms: dilated, hypertrophic, restrictive CMP and arrhythmogenic right ventricular CMP. Along with four nonspecific cardiomyopathies, including dilated, hypertrophic, restrictive, right ventricular arrhythmogenic dysplasia, as before, specific cardiomyopathies were identified and unclassified cardiomyopathies were added. Dilated cardiomyopathy (DCM) is the most common form of cardiomyopathy. The relevance of the problem of studying DCM lies in the fact that mainly people of working age are affected. Asymptomatic onset of the disease, late hyper- and underdiagnosis of manifest forms, refractoriness to pharmacotherapy, steadily progressive course, 100% disability of patients in the first or second years after clinical manifestations and high mortality give this problem not only medical, but also social significance [7, 8,9,10].

Principles of pharmacotherapy of patients with chronic heart failure

The etiology and pathogenesis of DCM have not been fully established. Therefore, the treatment of this disease to date remains nonspecific, syndromic, symptomatic and is mainly determined by the dominant clinical symptoms. In the clinical picture, the severity of the condition of patients with DCM, the course of the disease and the prognosis are determined mainly by the severity of the symptoms of CHF. Over the past decades, approaches to the treatment of CHF in patients with DCM have undergone significant changes [11,12,13].

The goal of treatment of patients with DCM with CHF is: to reduce the risk of complications; stop and/or reduce and control disease symptoms and risk factors; improve quality of life; reduce morbidity (frequency and severity of relapses, hospitalizations and progression of CHF); reduce mortality and increase life expectancy [14,15,16,17,18,19].

Based on this, pharmacotherapy for patients with DCM is based on the correction and prevention of the main clinical manifestations of the disease and its complications: congestive heart failure, arrhythmias and thromboembolism.

In recent years, a number of large studies have been conducted on the treatment of heart failure. Based

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on their results, the main groups of drugs that improve the symptoms of the disease, the condition of patients and the survival of patients have been identified [20,21,22,23,19]. These data are presented in table 1.2.1.

Table 1.2.1.
Drugs that reduce symptoms in CHF and affect patient survival

Drug	Symptoms	Survival
Heart transplant	++++	+++
Diuretics	+++	?
ACE inhibitors	++	++
Digoxin	+	0
Hydralazine/minoxidil	0	0/-
Hydralazine+isosorbide dinitrate	++	+
Nitrates	+	0/-
Beta blockers	+	+
Verapamil/diltiazem	-	--
Nifedipine/felodipine	-/0	-/0
Amlodipine (Norvasc)	+	0/+
Amiodarone	?	0
d-Sotalol	?	--
Encainide/flecainide	?	--
Phosphodiesterase inhibitors	+	-
Dobutamine	+	-
Diamorphine	+	-

Thus, one of the most important criteria in the selection of pharmacotherapy in patients with CHF is not only the effect on the clinical manifestations of the disease, its pathogenetic and pathophysiological validity, but also evidence-based medicine data on reducing the mortality of patients and increasing their life expectancy with various methods of intervention [24, 25,17, 26, 27,10].

In recent years, several international documents have been published on the treatment of chronic heart failure. First of all, this is the "Report of the working group on the problem of heart failure of the European Society of Cardiology (1995). Subsequently, the European Association of Cardiology Guidelines for the treatment of CHF (ECO, 1997), the European Society of Cardiology Guidelines for sudden cardiac death (2001) and the American College of Cardiology and American Heart Association Guidelines for the Diagnosis and Treatment of Chronic Heart Failure in Adults (ACC/AHA, 2001). They are based on evidence-based medicine [28,29,30,31, 10].

Despite the presence of these fundamental documents, in practical work there are problems in diagnosing DCM, verifying the diagnosis, risk stratification of patients and choosing methods of medical interventions. These problems become especially acute and significant during long-term observation of patients under conditions of continuous dynamic observation and treatment [30,41,58,68].

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