

**EARLY DIAGNOSIS AND PREDICTION OF THE RISK OF HEART FAILURE IN
DILATED CARDIOMYOPATHY IN CHILDREN**

Dilfuza Mirzabaevna Ruzmatova

Uzbekistan Republican Specialized Scientific-practical medical center of Pediatrics, Ministry of
Health of the Republic of Uzbekistan

Dilorom Ilhamovna Akhmedova

Tashkent pediatric medical institute

ABSTRACT: Dilated cardiomyopathy (DCMP) is characterized by a continuously progressive course, occupies a leading position in the structure of disability and mortality in children, and is the main cause of the formation of chronic heart failure in childhood. The purpose of our study was to study the clinical and diagnostic difficulties of making a diagnosis of dilated cardiomyopathy. Materials and methods: 85 children under 18 years of age with cardiomyopathy were examined, of which 60 children had dilated cardiomyopathy (DCMP). All children were hospitalized in the cardio-rheumatological department of the Republican Specialized Scientific-Practical Medical Center of Pediatrics of the Ministry of Health of the Republic of Uzbekistan. The control group consisted of 30 healthy children. The results of echocardiographic studies showed that in children with DCMP, the end-diastolic volume of the left ventricle reached from 94 ml to 206 ml, which was associated with an increase in its filling pressure and pronounced dilation of the left ventricle, which was accompanied by varying degrees of relative insufficiency of the mitral in some cases and tricuspid valves. Conclusion. To prevent the development of DCMP in children, it is necessary to carry out preventive methods to prevent late childbirth, ensure safe pregnancy and childbirth, prevent and effectively treat diseases of viral etiology in mothers and children, and early diagnosis and timely initiation of therapy are necessary for a favorable outcome of these diseases.

KEY WORDS: dilated cardiomyopathy, children, etiology, heart failure.

INTRODUCTION: According to modern concepts, dilated cardiomyopathy is understood as a disease of the heart muscle of unknown or unclear etiology, characterized by cardiomegaly due to dilation of the heart cavities, especially the cavity of the left ventricle, a progressive decrease in myocardial contractility, suddenly developing and progressive heart failure, arrhythmic and thromboembolic syndromes, which often end in sudden death [1,2]. DCMP is characterized by a continuously progressive course, occupies a leading position in the structure of disability and mortality in children, and is the main cause of the formation of chronic heart failure in childhood [3,4]. The prevalence of DCMP varies from 40 cases per 100,000 per

year in Europe. It is more common in boys than in girls. The proportion of DCMP among other cardiomyopathies is 60% [5,6].

The frequency of sudden death among children with DCMP ranges from 1.5% to 4%, in most cases the cause of death is arrhythmias. Cardiac arrhythmias are both bradycardic (atrioventricular block) and tachycardic (unstable ventricular tachycardia). Risk factors for sudden death include polymorphic ventricular extrasystoles [7,8].

Diagnostic criteria of DCMP, according to European experts, are [9,10] left ventricular ejection fraction less than 45% (according to echocardiography) or fractional shortening of the anterior-posterior size of the left ventricle less than 25%. Acute myocarditis also plays a role in the development of DCMP, when the myocardium is first affected, and then chronic inflammation develops, which, in turn, leads to remodeling of the heart and its dysfunction (post-inflammatory DCMP) [11,12].

Based on the above, this scientific study was aimed at determining the factors predisposing to the development of heart failure in children with dilated cardiomyopathy.

MATERIALS AND METHODS:

We examined 85 children with cardiomyopathy under the age of 18, of which 60 children had dilated cardiomyopathy (DCMP), hospitalized in the cardio-rheumatological department of the Republican specialized scientific-practical medical center of pediatrics of the Ministry of Health of the Republic of Uzbekistan. The research was conducted from 2018 through 2023.

The diagnosis was made based on complaints, anamnesis data (obstetric anamnesis of the mother, anamnesis of the life and illness of the child, previous diseases, the nature of the course and duration of the disease), functional (electrocardiography (ECG), echocardiography (ECHO CG), Holter monitoring), biochemical (determination of cardio specific markers - creatine kinase, lactate dehydrogenase) and instrumental (chest x-ray, chest multispiral computed tomography) research methods.

Growth and development of children were assessed according to the standards of growth and development of children recommended by WHO (2006; 2009).

Echocardiography was performed on patients in a planned manner during each hospitalization in the Department of Cardiorheumatology, both during the initial examination and during re-hospitalization to the department on the ultrasound machine Aplio-500 (Toshiba, Japan) with sector sensors 3.0-6.5 MHz

Determination of biochemical markers. Determination of lactate dehydrogenase, creatine phosphokinase, creatine phosphokinase MB, alanine aminotransferase, aspartate

aminotransferase activity was carried out in blood serum by immunoenzyme method on biochemical autoanalyzer of "HUMAN" company (Germany).

Determination of cerebral natriuretic peptide concentration. Determination of brain natriuretic peptide (BNP) in blood serum was carried out by solid-phase chemiluminescent immunoenzyme method using test kits IMMULITE 2000 "NT-proBNP".

Determination of blood electrolytes. Blood electrolytes (potassium, sodium, calcium) were determined by ionic gas method on Nova Medical device.

Standard (MS Excel 2000, Statistica 6.0) and specially developed programs were used for statistical calculations. Pearson correlation analysis was used. Differences were evaluated using Student's t-criterion. The following significance levels were accepted to assess the statistical validity of the obtained results: $p < 0.05$; $p < 0.01$; $p < 0.001$.

RESULTS AND DISCUSSION:

To determine the features of the course of the disease, children with cardiomyopathies were distributed by age (Fig.1.)

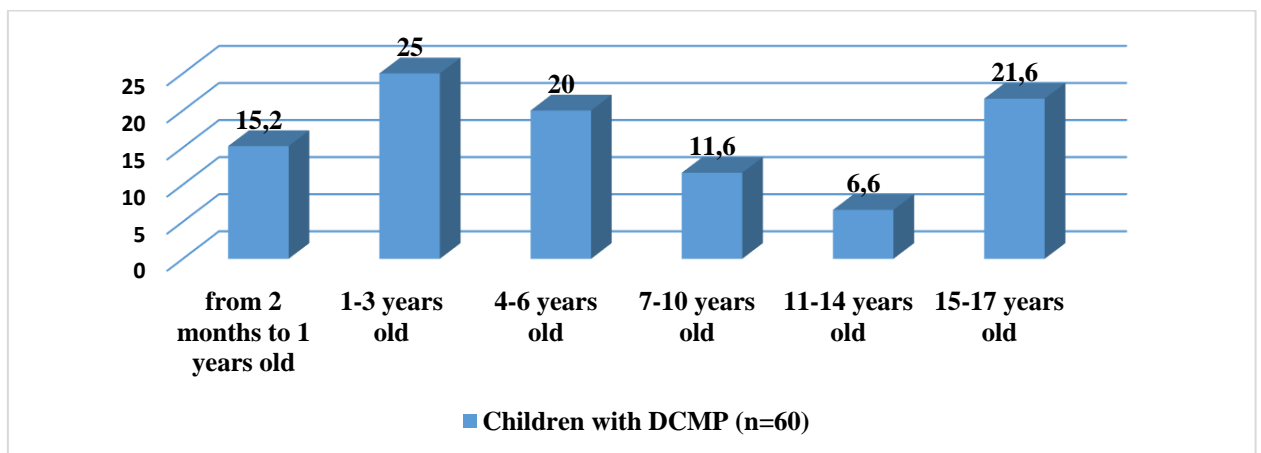


Fig.1. Distribution of children by age (%).

The age distribution of children with dilated cardiomyopathy showed that in children aged 2 months to 1 year, DCMP was (15.2%) of children. The lowest incidence of DCMP was in children aged 11-14 years, with an increase to 21.6% in adolescents aged 15-17 years. In children aged 1 to 4 years, (25%) of balo children were diagnosed with DCMP (Fig.1.)

During the study, we studied the medical and biological factors that contributed to the development of children with cerebral palsy.

The study of biomedical factors showed that DKMP develops more often in boys (63.3%). The development of DCMP was more prevalent in children whose mothers were over 35 years old at the time of birth of this child (38.3%). In 23.3% of children with DCMP, the parents were close relatives (Fig.2.).

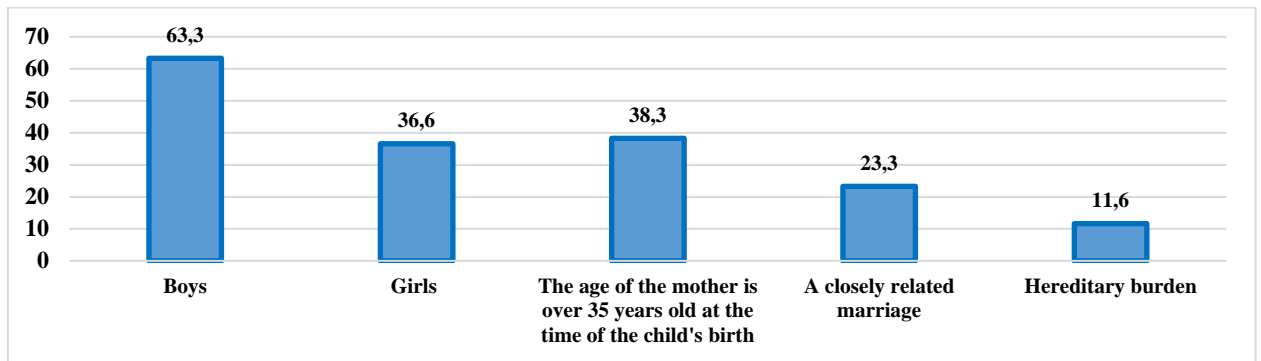


Fig. 2. Biomedical factors (%) in children with DCMP.

As is known, the birth of children with various diseases and congenital anomalies, as well as their subsequent development and health status depend on the course of pregnancy, the nature of childbirth and the health of their mothers.

Predisposition to a number of diseases is genetically determined. Heredity and environment act as etiological factors and play a role in the pathogenesis of any human disease, however, their share of participation in each disease has its own, and the greater the share of one factor, the less the contribution of the other.

The results of the study of hereditary burden in children with DCMP are presented in Table 1.

Table 1

Hereditary burden in children with DCMP

Diseases in relatives	DCMP (n=60)
-----------------------	-------------

	Abs.	%
Acute myocardial infarction	5	8,3
Congenital heart disease	4	6,6
Cardiac arrhythmias	8	13,3
Dilated cardiomyopathy	2	3,3
Myocarditis of various genesis	2	3,3
Death in the family from this disease	3	5,0

As can be seen from Table 1., 39.8% of children with DCMP in the family had congenital and acquired diseases of the cardiovascular system, 5.0% of which ended in death.

The analysis of the transferred diseases showed that children with DKMP at various age stages suffered from both viral and bacterial etiology diseases.

An analysis of anamnestic and objective data showed that the prescription of DCMP in children averaged 16.6 ± 3.4 months. The prescription of the disease also causes a lag in the physical and motor development of children. The more significant the disorders in physical development, the greater the likelihood of diseases. The study of the indicators of physical development of children with DCMP also revealed the presence of deviations in body length/height, body weight and body mass index relative to age norms. In terms of body length/height relative to age, deviations were found in 25.0% of patients with DCMP (range from -3CO to -2CO).

On external examination, perioral/periorbital cyanosis was noted in 19 (31.66%) children, acrocyanosis in 1 (3.3%) child. Chest deformity by the type of cardiac hump was visually determined in 4 (6.7%) children.

A physical examination showed an expansion of the percussion boundaries of relative cardiac dullness in 60 (100%) children with dilated cardiomyopathy. Tachypnea was detected in 17 (28.3%) children with DCMP.

Auscultationally, deafness of heart tones was noted in 50 (83.3%) children, systolic noise - in 52 (86.7%) patients. 1 (1.7%) child had wet wheezing in his lungs. Hepatomegaly was detected in 20 (33.3%) children, splenomegaly in 7 (11.66%) children. In addition, pasty shins and feet were noted in 4 (6.7%) children.

14 children (23.3%) were diagnosed with post-inflammatory DCM. At the same time, an average of 1 year passed from the onset of myocarditis to the diagnosis of DCMP. Complete recovery from post-myocarditis DCMP has been documented in one child.

X-ray examination revealed an increase in the size of the heart mainly due to the left

sections in 76.7% of children, total expansion was noted in 13.3% of children, the cardiothoracic index averaged $63.3 \pm 0.5\%$.

During the examination, the diagnosis of DCMP was found in 3 children admitted to the gastroenterology department with vomiting, swelling in the lower extremities and rapid stools. In these children, a planned echocardiography examination revealed a significant decrease in myocardial contractility with a decrease in the ejection fraction to 25%. Two children had family cases, and these children had symptoms of the disease in the form of serious heart failure from the first year of life. In 14 children (23.3%), the disease made its manifest debut: sudden cardiac arrest - in 6 children; syncopal state during physical activity - in 4 children (6.7%). In 6 deceased children (10%), the first sign of DCMP was sudden death within the first 6 hours after the appearance of signs of heart failure, despite the ongoing therapeutic measures.

An analysis of the research results showed that the following biochemical blood parameters were significantly increased in children with DCMP: LDH – $476,8 \pm 43.8$ IU/l ($p < 0.01$); ALT - de Ritis coefficient AST/ALT – $1,48 \pm 0.09$ ($p < 0.01$).

An analysis of the results of NT-proBNP studies in the blood showed (Fig.3.) that the level of NT-proBNP was significantly higher in children with DCMP with grade III CI (IV FC) and reached up to 38,000 pg/ml ($p < 0.001$), whereas in children with non-rheumatic myocarditis with Grade III CI (IV FC) this indicator reached 1200 pg/ml (Fig. 3.).

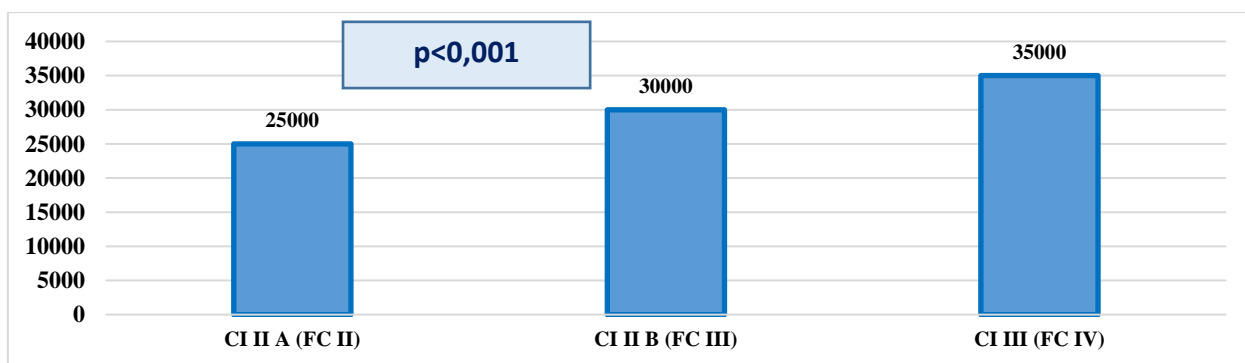


Fig. 3. The content of NT-proBNP in children with DCMP, depending on the stage of HF, pg/ml

Conclusion: As can be seen, the most pronounced increase in biochemical markers is observed in DCMP, which is confirmed by unfavorable outcomes during the progression of this

pathology in children. All this causes, in the presence of risk factors for the development of DCMP, along with functional studies, it is necessary to determine cardiospecific markers – creatine phosphokinase, creatine phosphokinase MV, as well as a cerebral natriuretic peptide. Determining the level of NT-proBNP in blood plasma helps to assess the severity of chronic heart failure, predict the further development of the disease, and evaluate the effectiveness of therapy.

References:

1. Vaykhanskaya T.G. . Sivitskaya L.N. . Kurushko T.B. and co-authors// Dilated cardiomyopathy: a new look at the problem // Russian cardiologic journal. 2019;24(4):35–47.
2. Zaklyazminskaya E.V. and co-authors. Dilated cardiomyopathy: Diversity of genetic causes and DNA diagnostic strategy // Clin. and experimental surg. Journal named after academician B.V. Petrovsky. 2019. T. 7 . № 3. P. 44–53. doi: 10.24411/2308- 1198-2019-13005.
3. Ivkina S.S . Zaryankina A.I. Non-rheumatic carditis in children /Teaching aid. Homel. 2018.23p.
4. Mershina E.A. . Sinicin V.E. . Larina O.M. Magnetic resonance imaging of the heart in the diagnosis of hypertrophic cardiomyopathy and risk stratification of sudden cardiac death // Clin. and experimental. surg. Journal named after academician B.V. Petrovsky. 2019. T. 7 . № 3. P. 70–78. doi: 10.24411/2308-1198-2019-13008.
5. Mikhailov V.S. and co-authors. Clin. and experimental. surg. Journal named after academician B.V. Petrovsky. 2018. № 1. P. 70–76
6. Maron B.J. . Towbin J. A. . Thiene G. . Antzelevich C. et al. Contemporary definitions and classification of the cardiomyopathies: an American Heart Association Scientific Statement from the Council on Clinical Cardiology . Yeart Failure and Transplantation Committee; Quality of Care and Outcomes Reasearch and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiologyand Prevention. Circulation 2018; 113: 1807 – 1816. DOI 10.1161/CIRCULATIONAHA.106.174287.
7. Lipshultz S.E. . Cochran T.R. . Briston D.A. . Brown S.R. . et al. Pediatric cardiomyopathies: causes . epidemiology . clinical course . preventive strategies and therapies. Future Cardiol 2019; 9; 817-848. DOI: 10.2217/fca. 13.66.
8. Wilkinson J. . Landy D. . Colan S. . Towbin J. . Sleeper L.A. . Orav E.J. . et al. Pediatric Cardiomyopathy Registry and Heart Failure: Key Results from the First 15 Years. Heart Fail Clin 2019; 6 (4): 401-413. DOI: 10. 1016/ j. hfc. 2010.05.002.

9. HEARTS: technical package for cardiovascular disease management in primary health care. WHO . 2018
10. Rusconi P. . et al. A Report from the Pediatric Cardiomyopathy Registry Study Group. Circ Heart Fail. 2017.
11. Halliday B.P. . Cleland J.G. . Goldberger J.J. . Prasad S.K. Personalizing risk Stratification for Sudden Death in Dilated Cardiomyopathy: The Past . Present . Future. Circulation 2020; 33(9): 888-909. DOI: 10.1161/ CIRCULA-TIONAHA. 116.027.
12. Mason J. W. Myocarditis and dilated cardiomyopathy: an inflammatory link / J.W. Mason // Cardiovasc. Res. – 2013. – Vol. 60. - P.5-10.

Authors: Dilfuza Mirzabaevna Ruzmatova Doctor of Philosophy Uzbekistan Republican Specialized Scientific-practical medical center of Pediatrics, Ministry of Health of the Republic of Uzbekistan (909050823)

Dilorom Ilhamovna Akhmedova Doctor of Medicine, Professor Tashkent pediatric medical institute